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FORENSIC MEDICINE AND TOXICOLOGY

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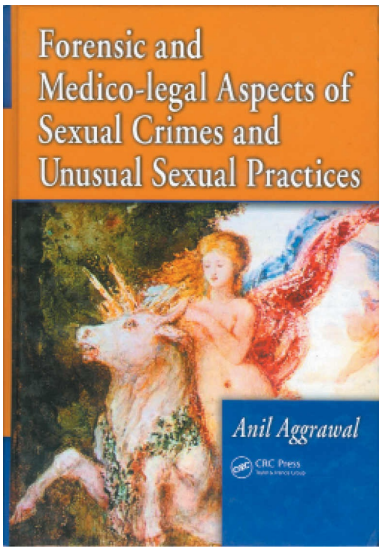

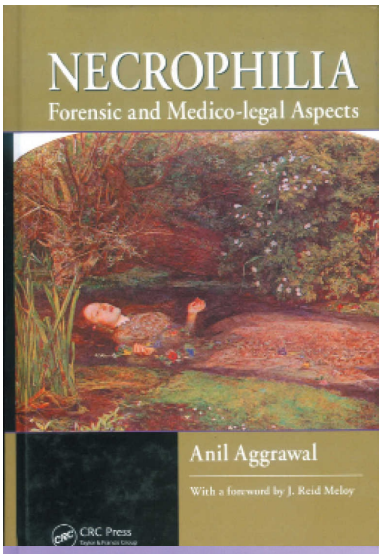
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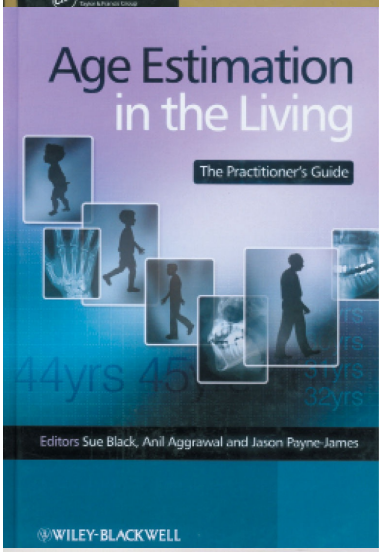
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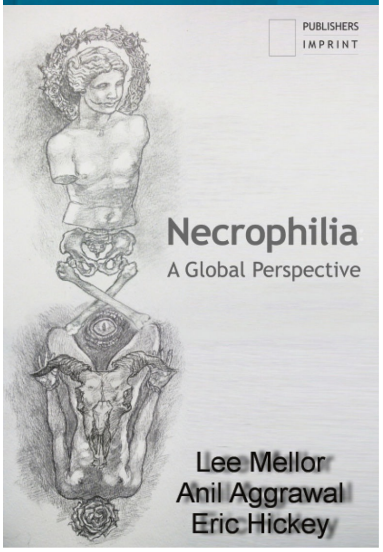
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Forensic Medicine AND Toxicology FOR MBBS

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AVICHAL PUBLISHING COMPANY
Industrial Area, Trilokpur Road, Kala Amb 173 030, Distt. Sirmour (HP)
Delhi Office: 1002 Faiz Road (opp. Hanuman Murti), Karol Bagh, New Delhi 110 005

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Published by:

AVICHAL PUBLISHING COMPANY

8, Industrial Area, Trilokpur Road
Kala Amb 173 030, Distt. Sirmour (HP)
Ph. : 01702-238688, 238832

Delhi Office:
1002 Faiz Road (opp. Hanuman Murti)
Karol Bagh, New Delhi 110 005 (India)
Phone: 011-28752745, 28752604, 28755383
Fax: 011-28756921
Email: medical@apcbooks.co.in
Website: www.apcbooks.co.in

ISBN–978-81–7739–491–7

© Author

First Edition: 2016

Typeset at
Laser Tech Prints (D)

Printed at
Prince Print Process, Delhi

Preface

The widespread acceptance of my "**Textbook of Forensic Medicine and Toxicology**" and "**Essentials of Forensic Medicine and Toxicology**" has led to requests for a shorter textbook that would give just bare essentials needed for understanding the subject and passing in exams.

In preparing the **Forensic Medicine and Toxicology for MBBS**, I have rewritten content, eliminated nonessential information, and modified illustrations in an effort to produce a comprehensive text that is shorter but equally accurate, up-to-date, and appealing to the student. A number of new diagrams, figures and illustrations have been included. Many more memory aids have been added, as they were found very helpful by the students.

I am grateful to Dr. Mohit Chauhan, Senior Resident, Department of Forensic Medicine, MAMC, who spared his valuable time to go through the entire manuscript word by word and suggest changes. Dr. Jatin Bodwal, Specialist, DDU hospital suggested necessary changes based on his vast experience with UG teaching. The contributions by Dr. Bodwal on firearm injuries and asphyxia, and by Dr. Chauhan on Identification chapters are highly appreciated.

I am thankful to the entire team of 'Laser Tech Prints' led by Shri Rajiv Manchanda, who provided me with a family like atmosphere during my month long visit to them. Special mention must be made of Shri V.K. Manchanda, whose magnificent presence and inspiring talks always filled me up with enthusiasm. Also worthy of praise are Ms Sangeeta Verma and Nilesh, who truly produced what I call "Medical Art", where a picture speaks a thousand words.

I am grateful to my publisher Dr. Vipin Gupta for reposing faith in me and going ahead with my idea. Without his encouragement and support such a project would never have seen the light of the day. I will always remain short of words to pay my sincere gratitude to him.

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Abbreviations used in this Book

1. Ac – Acute
2. BAC – Blood alcohol concentration
3. BV – Blood Vessels
4. cf – confer, compare
5. Ch – Chronic
6. COD – Cause of Death
7. D/b – Differentiate between
8. d/t – due to
9. E/m – Electron microscopy

10. Ex – Example

11. FD – Fatal Dose

12. FP – Fatal Period

13. H/P – Histopathology

14. HR – Heart rate

15. ICP – Intracranial Pressure

16. k/a – Known as

17. Lit – Literally

18. MAC – Maximum allowable concentration

19. MC – Most Common

20. MLI – Medicolegal Importance

21. MOA – Mechanism of Action

22. PChE – Plasma cholinesterase

23. PMI – Postmortem Interval

24. ppb – Parts per billion

25. ppm – Parts per million

26. PO – Per Orally

27. Pt – Patient

28. SI – Simple Imprisonment
[no work allotted to prisoner]

29. RI – Rigorous Imprisonment
[work allotted to prisoner]

30. RR – Respiratory rate

31. S & S – Signs and symptoms

32. Symb – Symbol [of chemical]

33. Syn – Synonym

34. TLV – Threshold Limit Value

35. TSD – Time since Death

36. w.e.f. – With effect from

37. w.r.t. – With respect to

Memory Aids and Mnemonics Index

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1. Introduction and Legal Procedure

I. INTRODUCTION

A. Forensic Medicine

Forensic medicine, Legal medicine or **State medicine** is the application of medical knowledge for the purposes of law and administration of justice. Briefly: **Medicine (as applied to) ⇒ Law**.

B. Medical Jurisprudence

Medical jurisprudence [Latin juris, law; prudentia knowledge] is the study of legal principles that guide medical personnel. Briefly:

Law (as applied to) ⇒ Medical personnel

C. Medical Ethics

Medical ethics is the study of moral principles guiding medical men in their dealings with their patients.

Salient features:

Graphically: **Doctor Patient**

D. Medical Etiquette

Medical etiquette deals with the conventional laws of courtesy observed between members of medical profession. Or graphically:

Doctor Doctor

Violation of medical etiquette is **NOT punishable under law**.

II. LEGAL PROCEDURE

A. Civil Law

Deals with disputes between two private individuals or parties. **Ex**. Doctor D negligently places tight plaster over patient P's leg. Patient's leg is paralyzed. He brings a suit against the doctor for compensation of `10 lakhs. Patient is plaintiff; doctor is defendant. Case is cited as **P vs. D** [please compare with example given under criminal law].

B. Criminal Law

Deals with offences, which are against public interest (e.g. offences against the

person, property, public safety, security of state etc).

(1) **Fundamental purpose of criminal law** – is to punish those individuals who commit crimes [as contrasted to the fundamental purpose of civil law, which is to grant compensation to the affected individual].
(2) **Civil case vs. criminal case** – Civil case is a private fight between two individuals [e.g. patient and doctor]; criminal case is a fight between the State and the individual [e.g. State trying to prosecute a doctor].

C. Public Prosecutor

Public prosecutor [PP] is the lawyer of the state, and is the person in-charge of prosecutions [**S.2(u), CrPC**].

Memory Aid 1: Public prosecutor

S.2(**u**)-p**u**blic prosecutor

(1) **Duties** - He represents the State in all prosecutions.

D. Statutory Law

Law duly passed by legislature. **Ex**. According to S.375 IPC, sexual intercourse with a girl under 18 years of age – even with her consent - is rape. This is called statutory rape.

III. INQUEST

An ***inquest*** is an enquiry or investigation into the cause of death.

A. Types

There are **six** types of inquests (**Table 1**).

Table 1: Various types of inquests

S. No.	Type of inquest	Conducted by (u/s)	Prevalent in	Level of excellence
1.	Police inquest	Police officer u/s174, CrPC	India	Not considered very good
2.	Executive Magistrate inquest	Executive Magistrate u/S.176(1), CrPC	India	Very good
3.	Judicial Magistrate inquest	Judicial Magistrate u/S.176(1A), CrPC	India	Very good. Superior to even Executive Magistrate inquest
4.	Coroner's inquest	Coroner	UK, Canada, Australia	Very good
5.	Procurator fiscal's inquest	Procurator fiscal, who is similar to a coroner in UK	Scotland	Very good
6.	Medical examiner system	Official who is both legally and medically qualified	USA	Best. Most superior system of inquest.

1. Police inquest

Police inquest refers to inquest held by a police officer.

Salient features:

(1) held **u/s174, CrPC.**

(2) **IO**- Generally the officer-in-charge of a **Police Station** (SHO or Station House Officer) or **Police Post** conducts inquest. He is usually of the rank of sub-inspector (SI), but no such rank has been specified in S.174, CrPC. Police officer conducting the inquest is known as the **Investigating Officer** (IO).

(3) **Most common** type of inquest in India.

(4) **Inferior** to Magistrate's inquest, because police officer is less qualified than magistrate.

Memory Aid 2: Police Inquest

Police inquest is **Po**or

a. Procedure

(1) Whenever officer in-charge of a police station receives information that a suspicious death has occurred somewhere, he immediately **informs to the nearest Executive Magistrate**.

(2) Then he (IO) **proceeds to the place** where the body of dead person is lying.

(3) In the presence of two or more respectable persons (panchas, panch witnesses or panchayatdars; so called because originally there used to be 5 people) makes an investigation and draws up a report of the apparent cause of death. He describes such wounds, fractures, bruises and other marks of injury as may be found on the body. He states how and with what weapon such marks could have been produced.

(4) **Power to summon people** - Police officer may summon any person to answer questions related to crime u/s 175, CrPC. Refusal to answer punishable u/s 179, IPC. (6 months and/or `1000).

(5) **Inquest report** – The report thus prepared is called the inquest report (panchanama), and is signed by IO and panchas.

(6) **Disposal** - If no foul play is suspected, body is handed over to the relatives for disposal; if foul play is suspected, body is sent for postmortem.

(7) In both cases, the inquest report is forwarded to the District Magistrate (DM) or Sub divisional Magistrate (SDM).

2. Magistrate's inquest

a. Executive Magistrate's inquest

Executive Magistrate's [EM] inquest refers to inquest held by an executive magistrate.

Salient features:

(1) **Executive Magistrates empowered to hold inquests** are: (i) District Magistrate [DM] (ii) Additional District Magistrate [ADM] (iii) Sub-divisional Magistrate [SDM] (iv) Special Executive Magistrate (v) Collector (vi) Sub Collector (vii) Deputy Collector (viii) Deputy Commissioner (ix) Revenue Divisional Officer [RDO] (x) Tahsildar and (xi) any other executive magistrate empowered by the State Govt or DM [**S.20, CrPC** to **S.23, CrPC**; **S.174(4), CrPC**].

(2) It is held **u/s176(1), CrPC**.

(3) **Held in cases of** (i) suicide of a woman within 7 years of marriage (ii) death of a woman within 7 years of marriage in any circumstances raising a suspicion that some other person has committed an offence in relation to such woman [homicide etc] (iii) Exhumation.

(4) **Presence of relatives** - Wherever practicable, the magistrate shall inform the relatives of the deceased whose names and addresses are known, and shall allow them to remain present at the inquiry **[S.176(4), CrPC]**.

(5) **Imp points** – It is erroneous to say that magistrate's inquest is held in **dowry deaths** [**S.304B, IPC**] cases. If a woman commits suicide within 7 y of marriage, and there is no allegation of dowry, legally it is not dowry death [**ch 11**], yet inquest would be done by an executive magistrate.

b. Judicial Magistrate's inquest

Judicial Magistrate's [JM] inquest refers to inquest held by a judicial magistrate.

Salient features:

(1) ***Judicial magistrate's inquest*** is held **u/s176(1A) of the CrPC** in cases of (i) death or disappearance of any person (a) in police custody or (b) in any other custody authorized by magistrate or court [**S.176(1A), (a)**] [jail, children's remand home, psychiatric hospital, psychiatric nursing home etc]. Officers empowered to hold inquests under this section are judicial magistrates and metropolitan magistrates [both are judicial officers and trained in law]. (ii) rape is alleged to have been committed on any woman, while in police custody or any other custody [as above]. Death may occur even outside custody [**S.176(1A), (b)**].

Table 2: Differences between police, executive magistrate and Judicial magistrate's inquests

S. No	Feature	Police inquest	Executive magistrate's inquest	Judicial Magistrate's inquest
1.	Conducted u/s	174, CrPC	176(1), CrPC	176(1A), CrPC
2.	Conducted by	Police	Executive magistrate	Judicial magistrate
3.	Information given to	Executive magistrate of the area	None	None
4.	Cases in which done	All except those handled by magistrates	(1) Death of a woman within 7 y of marriage [for details please see text] (2) Exhumation	Death in custody [for details please see text]
5.	Examination of witnesses	Done u/s 175, CrPC	176(1), CrPC	176(1A), CrPC
6.	Value of statements made by witnesses	No value **u/s 25, IEA**. Not admissible in court	Valuable and admissible in court	Valuable and admissible in court
7.	Exhumation	Cannot order	Can order	Can order
8.	Examination of viscera	Cannot order, but can make a request	Can order	Can order
9.	Quality of investigation	Inferior	Superior	Best

3. Coroner's inquest

Currently held in - Coroner's inquest is currently held in Australia, Canada, UK, some states in USA and in some other countries, but not in India. During British rule, coroner's inquest was held in India under the Coroner's Act, 1871.

After gaining independence, India gradually did away with the British system. By 1977, only the cities of Kolkata and Mumbai were having Coroner's inquest. Since April 1978, coroner's system has been abolished in Kolkata and since 29 July 1999 in Mumbai.

Table 3: Differences in Coroner's and Magistrate's court

S. No.	Feature	Coroner's court	Magistrate's court
1.	Type of court	Court of enquiry	Court of trial
2.	Accused	Need not be present	Should be present
3.	Powers	No power to award punishment or fine	Can impose
4.	Contempt	Can punish a person, if contempt is done within court	Can punish if contempt is done anywhere

4. Procurator fiscal system

Same as the Coroner system. The coroner is called Procurator Fiscal in Scotland.

5. Medical examiner system

In ***medical examiner system***, an official who is both legally and medically qualified (known as the ***medical examiner***) conducts an inquest as well as the postmortem examination.

Salient features:

(1) **Best system** - Considered the best system, as the same person visits scene of death [to conduct inquest] and conducts autopsy. Thus there is no loss of

information.

(2) **Followed in** - USA (first introduced in 1877, in Massachusetts).

IV. INVESTIGATION OF THE SCENE OF DEATH

Duties of Forensic Pathologist at the Scene of Death

Record -

(1) Date, time [of arrival and departure from scene], address, location

(2) Sketching - (a) Useful, especially when pathologist wants to record own notes, e.g. Distance of weapon from body, depth of water level at the site of drowning etc [**Fig 1.1**] (b) Make to uniform scale. Be careful to accurately note relative distances and measurements. (c) At a corner of sketch record directions [N-S].

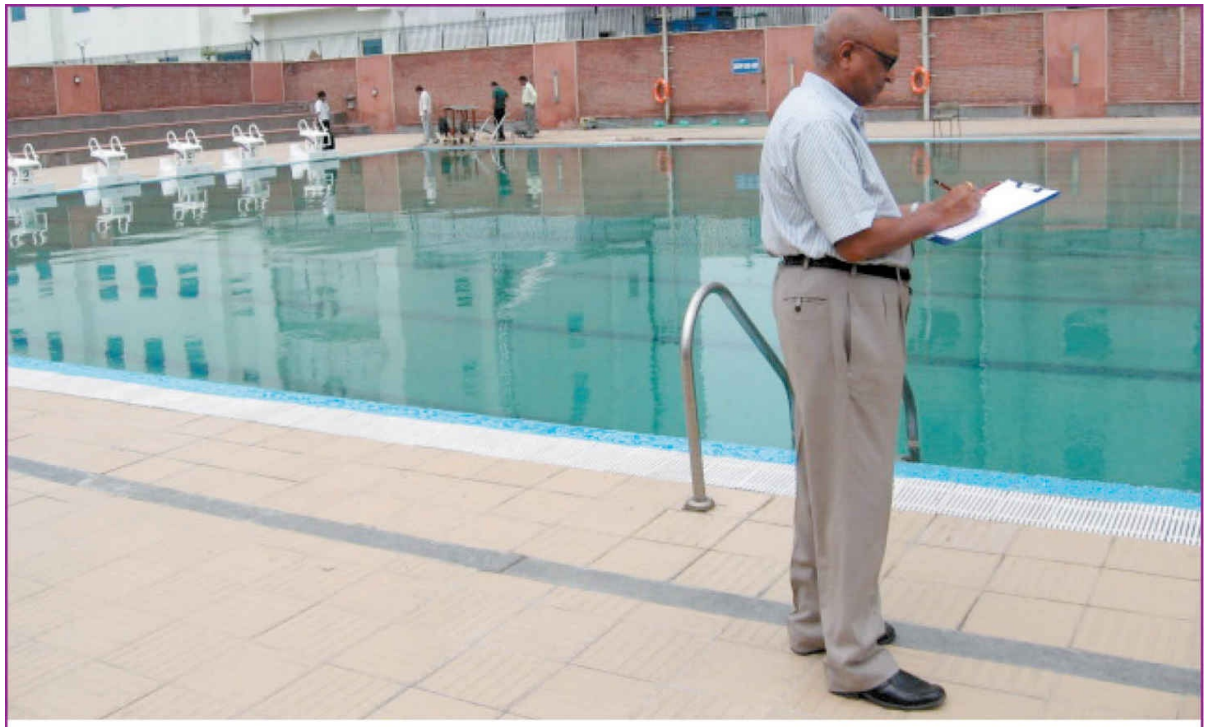


Fig. 1.1: Author preparing a scene of death sketch at the site

V. JURY

A ***jury*** is a sworn group of responsible, educated people of good social position, who assist the judge in deciding a case.

VI. COURTS OF LAW

Courts of law are of two types:

(1) Civil and

(2) Criminal. There are **4 tiers** of courts – **Supreme court, high court, sessions court** and **judicial magistrates court**.

A. Supreme Court

It can try all kinds of criminal offences, and it can pass any sentence authorized by law, including the death sentence **[Article 134 Constitution of India]**.

B. High Court

Same as that of SC. [**S.28(1) of the CrPC]**.

C. Sessions Court

6 levels of officers sit here [3 kinds of Judges and magistrates each). **Powers** – **Table 4**.

D. Judicial Magistrates Court

Pl. see **Table 4**.

Table 4: Powers of criminal courts

S.No	Judge/Court	Imprisonment	Fine	Law
1.	Supreme Court Judge **[Supreme Court]**	Any period. Capital punishment also	Any amount	Article **134 Constitution of India**
2.	High court Judge **[High Court]**	Any period. Capital punishment also	Any amount	S.28(1), CrPC
3.	District and Sessions Judge (DSJ) **[Sessions court]**	Any period. Capital punishment also. But the Capital punishment has to be confirmed by the High Court	Any amount	S.28(2), CrPC
4.	Additional District and Sessions Judge (Add DSJ) **[Sessions court]**	Same as above	Any amount	S.28(2), CrPC
5.	Assistant Sessions Judge **[Sessions court]**	10 years	Any amount	S.28(3), CrPC
6.	Chief Judicial Magistrate [CMM in metropolitan cities] **[Judicial magistrate's court]**	7 years	Any amount	S.29(1), CrPC
7.	Additional Chief Judicial Magistrate [Additional CMM in metropolitan cities] **[Judicial magistrate's court]**	7 years	Any amount	S.12(2), CrPC [ACJM]; S.17(2), CrPC [ACMM]
8.	1st Class Judicial Magistrate [MM in metropolitan cities] **[Judicial magistrate's court]**	3 years	10,000 Rs	S.29(2), CrPC
9.	2nd Class Judicial Magistrate **[Judicial magistrate's court]**	1 year	5,000 Rs	S.29(3), CrPC

VII. OFFENCE

The word **offence** is defined slightly differently in IPC and CrPC. (A) The word "Offence" denotes a thing made punishable by the ***Indian Penal Code*** [***S.40, IPC***].(B) "Offence" means any act or omission made punishable by any law for the time being in force [***S.2(n), CrPC***].

Classification

Offence may be classified as following.

1. Cognizable or non-cognizable

This classification takes into account whether police can arrest without warrant or not.

a. Cognizable offence

Cognizable offence means an offence for which, a police officer may arrest without warrant. [**S.2(c), CrPC**]. **Examples** (IPC sections are mentioned within

brackets): (i) Murder [S.302] (ii) Causing death by rash or negligent act [S.304A] (iii) Dowry death [S.304B] (iv) Abetment of suicide [S.306].

b. Non-cognizable offence

Non-cognizable offence means an offence for which, a police officer has no authority to arrest without warrant.[**S.2(l), CrPC**]. **Examples**– (i) Causing miscarriage with woman's consent [S.312, IPC], (ii) Voluntarily causing hurt [S.323, IPC], (iii) Assault or use of criminal force on grave and sudden provocation [S.358, IPC]. Differences between the two are given in **Table 5**.

Table 5: Differences between a cognizable and non-cognizable offence

S.No	Criteria	Cognizable offence	Non Cognizable offence
1.	Definition	Defined in **S.2(c), CrPC**	Defined in **S.2(*l*), CrPC**
2.	Recording of information	Police has to record information in writing [**S.154, CrPC**].	Police has to enter information in register prescribed for it and refer the informant to a magistrate [**S.155, CrPC**].
3.	Arrest	Police can arrest without warrant	Police cannot arrest without warrant from a magistrate
4.	Starting of investigation	Police can start investigation without the order of a magistrate.	Police officer cannot investigate the case without the order of a magistrate.
5.	Seriousness of offences	Generally more serious	Generally less serious

VIII. PUNISHMENTS

(1) **Sentences authorized by law** [**S.53, IPC**] are:

(i) Death

(ii) Imprisonment for life

(iii) Imprisonment (a) Rigorous, i.e. with hard labor, including solitary confinement [**S.73**, **S.74, IPC**] (b) Simple

(iv) Forfeiture of property

(v) Fine

(2) **Double jeopardy** - No person can be prosecuted and punished for the same offence more than once [**Article 20(2), Constitution of India**].

IX. SUBPOENA [SUMMONS]

Subpoena [L. sub, under; poena, penalty] is a document compelling the attendance of a witness in a court of law under penalty on a particular day, time

and place, for the purpose of giving evidence.

Salient features:

(1) **Types** – Subpoena can be classified according to (i) the nature of court which issues it [civil court or criminal court] (ii) whether a document is required to be produced or not [Subpoena ad testificandum and Subpoena duces tecum].

(2) **Failure to produce document** – Imprisonment of 6 months or `1000 fine, or both [**S.175, IPC**].

(3) **Failure to produce thing** – Simple imprisonment of 7 days [**S.349, CrPC**].

(4) **Procedure of issue** – Subpoena is issued by the court in writing, in duplicate. It is signed by the presiding officer of the court, and bears the seal of the court [**S.61, CrPC**].

(5) **Things mentioned in summons:** (i) Name of accused (ii) Case details, crime number (iii) Date and time to arrive to court.

(6) **Served by:** (i) Court official or (ii) Police officer or (iii) some other public servant [**S.62(1), CrPC**].

(7) **Procedure of delivery:** (i) Summon is delivered personally if practicable [**S.62(2), CrPC**] (ii) Person receives one copy of summons and signs at the back of the duplicate [**S.62(3), CrPC**] and returns it to the delivering official.

(8) **Procedure of delivery to a Govt servant:** (i) Summon delivered to head of office in which govt servant works (ii) Head of office signs on the back of duplicate and delivers the original copy to the concerned Govt servant [**S.66, CrPC**].

(9) **If person summoned resides in a different city** – Court will send summon in duplicate to the area magistrate and he will deliver the summon to the person concerned [**S.67, CrPC**].

X. CONDUCT MONEY

Conduct money is money paid to a witness to meet his expenses for attending the court.

Salient features:

(1) **Civil case:** (i) Conduct money is usually paid at the time of service of summons. (ii) If money is not paid, the doctor must still attend the court and demand his expenses at the end of his evidence. (iii) The party which calls the doctor pays. (iv) If doctor feels that the amount is less, he must bring it to the notice of the Judge. The judge will decide the amount to be paid.

(2) **Criminal case:** (i) No money is paid at the time of service of summons (ii) If he is a doctor in private practice, he may request the court for conduct money at the end of his evidence. Generally conduct money is paid by the court. (iii) If he is a Govt doctor, paid by his office.

XI. EVIDENCE

"Evidence" according to **S.3, IEA** means and includes.

(1) all statements which the Court permits or requires to be made before it by witnesses, in relation to matters of fact under inquiry [**oral evidence**];

(2) all documents including electronic records produced for the inspection of the Court [**documentary evidence**]. An evidence tendered by a medical personnel is known as ***medical evidence***.

Types of Evidence

Evidence may be classified in two ways–

(1) How the evidence was acquired by witness.

(2) How the evidence was delivered in court [**Fig 1.2**].

1. According to acquisition of evidence

a. Direct evidence

Witness directly saw the crime or felt it by any of his senses. **Ex** (1) Doctor conducted surgery or postmortem. Saw injuries himself (2) A passerby saw a murder on the street.

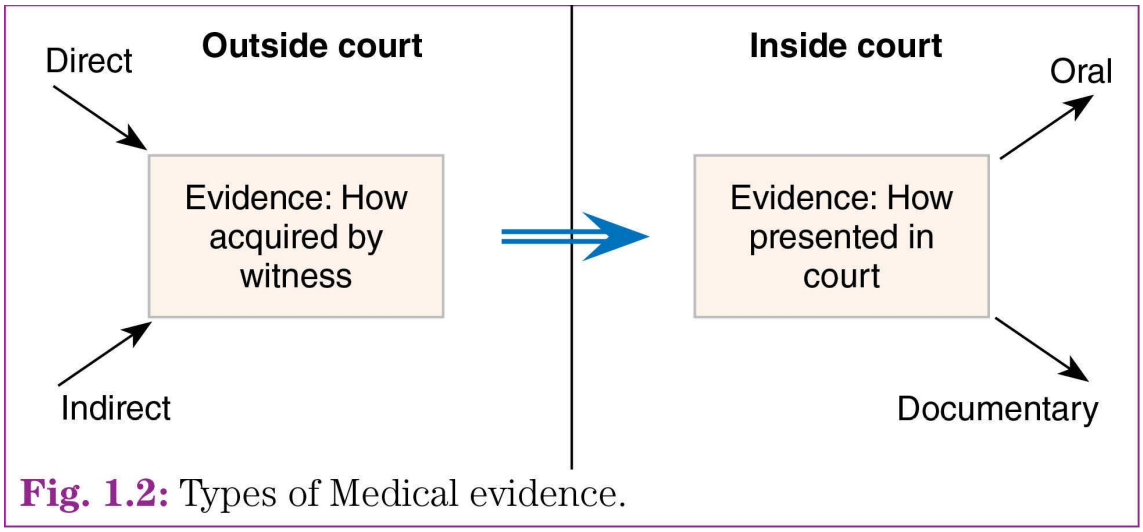


Fig. 1.2: Types of Medical evidence.

b. Indirect evidence

Witness did not directly see the crime. It can be of 2 types.

i. Circumstantial

Witness did not see actual crime, but several related things exist, which point strongly towards the commission of crime.

Salient features:

Admissibility - Circumstantial evidence is generally admissible in court unless the connection between the fact and the inference is too weak to be of help in deciding the case. Many convictions for various crimes have rested largely on circumstantial evidence.

ii. Hearsay

Witness only heard about the crime from someone.

Salient features:

Admissibility - Generally hearsay evidence is not admitted. **Two notable exceptions** (i) Dying declarations [**S.32(1), IEA**] and (ii) Res gestae [**S.6, IEA**]. Res gestae are involuntary exclamations or acts made at the time the offense was committed. These utterances or acts are not planned, but are forced from the individual by the excitement of the moment.

2. According to presentation in court

a. Documentary evidence

It may be of following types.

i. Medical certificates

Medical certificates are documents prepared by the doctor at the request of the patient, and handed over to him. **Ex –** certificate of age, death, disability, pregnancy, sickness, unsoundness of mind, vaccination etc.

Salient features:

(1) Accepted by a court of law only when issued by a qualified registered medical practitioner.

(2) **Certificate of ill health:** (i) To be given on a recommended format - According to S.1.3.3 of The Indian Medical Council (Professional Conduct, Etiquette and Ethics) Regulations, 2002 [IMC Regulations 2002], should be given in recommended format given in appendix 2. It should contain the following (a) identification marks of the patient (b) exact nature of illness (c) period of expected absence (ii) Confidentiality - Patient's medical records should be kept confidential [S.2.2, IMC Regulations 2002] (iii) Maintenance of register – Doctor should keep one copy of certificate with him. Patient's signature/thumb impression, and his full address should be recorded on both copies.

(3) **Death Certificate:** (i) Doctor is legally bound - If doctor has attended a patient during his last illness, and if he has expired, the doctor must issue the certificate without charging any fee [S10(3) Registration of Births and Deaths Act, 1969] (ii) If doctor refuses to issue a death certificate under above condition, fine is `50 [S.23(3) of same Act]. (iii) Death certificate must never be issued without inspecting the dead body, and satisfying himself that the patient is really dead [criteria to apply are Minnesota criteria – please see ch 8]. (iv) If doctor is suspicious about the cause of death, the matter must be informed to the police.

(4) **Issuing false certificates** – Issuing false, fictitious or backdate certificates is an offence **u/s197,** IPC. Punishment 7 y + fine. Patient using such a certificate is punishable **u/s198, IPC**. Punishment same.

(5) **Issuing backdate certificates** – Must never be done. There are instances of

criminals getting certificates in back date and then producing them as alibi in a court of law. This can land doctor in serious trouble. Punishment same as for issuing false certificate.

ii. Medicolegal reports

Medicolegal reports are documents prepared by the doctor at the request of some investigating authority, e.g. police, magistrate etc, and handed over to him. They may be in relation to living [e.g. injury reports], dead [Postmortem reports] or inanimate objects [e.g. vaginal swab reports].

Salient features:

Structure of the report – It consists of 3 parts - (i) Preliminaries – (a) Serial number [e.g. 273/2012 means that it is the 273rd MLC recorded in the year 2012] (b) name (c) father's/guardian's name; husband's name in cases of married women (d) age, (e) sex, (f) address, (g) occupation (h) name and address of accompanying/identifying person if any (i) two identification marks, (j) date, time and place of examination, (k) consent (l) name and number of the police official to whom information was sent and MLC was handed over. In case the patient first reported to the police, and police brought the patient to doctor for examination, the name, number and police station (PS) of accompanying police officer. The preliminaries are to be filled up in all MLCs whether related to physical injury, age estimation, examination for mental soundness, poisoning [e.g. alcohol] etc. (ii) Body – Main part. Consists of (a) history as alleged by the patient/accompanying person, (b) observations, (c) findings of physical examination [type of each injury, its size, shape and direction, on what part of the body inflicted, its age and remarks if any], (d) special investigations requested etc (iii) Opinion and subsidiaries – Conclusions derived from the observations (a) nature of injury [e.g. simple or grievous] (b) likely weapon with which caused, and whether it was dangerous or not (c) if police wants to take the statement of the patient, the doctor must certify if patient is fit for statement or not.

iii. Dying declarations

Dying declarations are documents prepared on the statements of a person dying as a result of some unlawful act.

Salient features:

(1) It is an example of **hearsay evidence**. It is admitted on the principle of necessity [for a similar situation, please see **ch 28** " Wills in extremis].

(2) **Manner of death** – May be made in all manners of death – suicide, homicide, accident.

(3) **Preliminaries** - Before recording the statement, the doctor should certify that the declarant is fit to make a statement [compos mentis] [ch 28].

(4) **Who can record** – Anyone. (i) Executive magistrate [EM]– Ideally he should be called if there is time. If not possible, other persons who can record are (ii) Doctor (iii) Police (iv) village headman (v) Any other person [Recording by anyone other than the EM, should preferably in the presence of 2 independent witnesses, who should sign the dying declaration] (vi) The statement can be oral, tape-recorded, or may even be made on phone, by SMS or e-mail to someone. Evidentiary value is maximum when recorded by magistrate, by SMS or e-mail followed by doctor.

(5) **Attestation** - Dying declaration recorded by the police in the presence of medical officer. Medical officer need not attest the statement.

(6) **Recording**: (i) Oath – Not administered. The general belief is that a dying person will not speak a lie (ii) Language - Dying declaration narrated in one language (e.g. Punjabi), but recorded in another (Urdu) " Admissible. (iii) Format – (a) If possible, it should be written by the person himself, and then signed by him. If person is not in a condition to write, but can speak, it should preferably be (b) video recorded. If that is not possible, then person recording the d/dec should write the statement (c) in person's own words. **Precautions** - (I) No alteration of terms and phrases. (II) No prompting or assistance (III) Statement must be on facts, not on opinions and judgments [my sister-in-law might have helped my husband in burning me]. If declaration is made on judgments, questions should be asked by the recorder to clarify facts [did you see your sister-in-law helping your husband?]. Alternatively, it can be (d) in the form of questions and answers; but leading questions should not be asked [It is not proper to ask "Did your mother-in-law put fire on your body?"; instead it must be asked "who burnt you?"] (e) if unable to speak - questions put and signs/gestures made in reply are recorded (iv) Reading out – After recording the statement, it should be read out to the declarant, and asked if he agrees. If yes, his signatures or thumb impressions are taken. If signatures or thumb impressions cannot be taken for some reason, this fact should be

recorded. The value of d/dec would be same.

(7) **Onward transmission to Magistrate** – In case d/dec is taken by any person other than magistrate, it should be forwarded to him in a sealed cover.

b. Oral Evidence

Oral evidence includes all verbal statements under oath made before the court which it permits.

i. Dying deposition

Dying deposition is a statement of a dying person on oath, recorded by the magistrate in the presence of the accused or his lawyer, who is allowed to cross examine the witness.

Salient features:

(1) This is court at the bedside. Its evidentiary value is more [**Table 6**].

(2) **Dictum** - It works on the dictum "if the person cannot go to the court, the court must go to the person".

(3) Held in many countries like UK, Australia and many states of USA, but not in India.

Table 6: Differences between dying declaration and dying deposition

S.No.	Criteria	Dying declaration	Dying deposition
1.	Recorded by	Anyone - a police officer, a magistrate, or a doctor	Always recorded by a judicial magistrate or judge
2.	Oath	not administered	Must be administered
3.	Presence of accused and lawyer	Not necessary	necessary
4.	Cross-examination	No	yes
5.	Nature	Merely the recording of a statement	Full-fledged court procedure ["the court by the bedside"]
6.	Type of evidence	Documentary	Oral
7.	Victim survives	Loses value	Retains value
8.	Legal Value	Less	More
9.	Prevalent in India	Yes	No

ii. Exceptions to oral Evidence

Documentary evidence is accepted by the court only on oral testimony [oral evidence]. Imp exceptions are

(1) **S.32, IEA** - Statements, written or verbal, of relevant facts made by a person: (i) who is dead [dying declaration], or (ii) who cannot be found, or (iii) who

has become incapable of giving evidence, or (iv) whose attendance cannot be procured without an amount of delay or expense which, under the circumstances of the case, appears to the Court unreasonable

(2) **S.33, IEA** -Evidence given by a witness in a previous judicial proceeding is admissible in a subsequent judicial proceeding, or in a later stage of the same judicial proceeding when (i) the witness is dead or (ii) cannot be found, or (iii) is incapable of giving evidence, or (iv) is kept out of the way by the adverse party, or (v) if his presence cannot be obtained without an amount of delay or expense which, under the circumstances of the case, the Court considers unreasonable. Following additional provisos apply (vi) the proceeding must be between the same parties (vii) the adverse party in the first proceeding had the right and opportunity to cross-examine (viii) questions in issue are substantially the same in the first as in the second proceeding.

(3) **S.60, IEA** - Opinions of experts expressed in any treatise commonly offered for sale, and the grounds on which such opinions are held, may be proved by the production of such treatises if (i) the author is dead or (ii) cannot be found, or (iii) has become incapable giving evidence, or (iv) cannot be called as a witness without an amount of delay or expense which the Court regards as unreasonable.

(4) **S.291, CrPC -** Evidence of a doctor recorded in court, if taken and attested by a Magistrate in the presence of the accused, may be given in evidence as such in any inquiry, trial or other proceeding. But he is liable to be called if court thinks fit [e.g. additional cross examination required, evidence is deficient etc].

(5) **S.292, CrPC -** Written reports of mint officers, Security Printing Press, Controller of Stamps and Stationery, any Central Government or State Examiner of Questioned Documents are accepted normally without calling them to court. However, the Court may, if it thinks fit, summon and examine any such officer.

(6) **S.293, CrPC -** Reports of certain Government scientific experts: (a) Chemical Examiner or Assistant Chemical Examiner to Government; (b) Chief Controller of Explosives; (c) Director of the Finger Print Bureau; (d) Director, Haffkine Institute, Mumbai (e) Director, Deputy Director or Assistant Director of a Central Forensic Science Laboratory [CSFL] or a State Forensic Science Laboratory [SFSL] (f) Serologist to the Government; (g) any

other Government Scientific Expert specified, by notification, by the Central Government for this purpose [**S.293(4), CrPC**]. The Court may however summon and examine any such expert if it thinks fit [**S.293(2), CrPC**].

(7) **Hospital Records** – Routine entries made during ordinary course of work, e.g. Dates of admission and discharge, Experts called, Pulse, BP, Signs noted, Special investigations ordered, Temperature, Treatment given. But their interpretations e.g. cause of disease, basis of diagnosis etc are not accepted without oral testimony.

(8) **Public records** – Kept in public office, e.g. birth, marriage, death etc [S35, 74, 76, 78 IEA].

XII. WITNESS

A ***witness*** is one who has firsthand knowledge about a crime or significant event through his senses [e.g. seeing, hearing, smelling, touching] and can help clarify important points about the crime or event.

Salient features:

(1) **Sections 118-134** [ch IX] of **IEA** deal with witnesses.

(2) **Persons competent to be witnesses** [**S.118, IEA**]– All persons are competent to testify, unless they cannot understand questions put to them because of (i) Disease of body or mind (ii) Extreme young age (iii) Extreme old age. No age is specified. Even a mentally ill person can be a witness if he can understand questions and give rational answers.

(3) **Dumb witness** [**S.119, IEA**]– can testify by writing or by making signs. Such writing or signs must be made in open court. It is deemed as oral evidence.

Types of Witnesses

Witnesses are of two types:

(1) Common (ordinary) witness

(2) Expert witness

Any of these may become a hostile witness.

1. Common witness

Common witness [lay witness, occurrence witness, witness of fact] is a person, who tenders evidence about the facts observed or perceived by him. No specialized knowledge is required in tendering evidence.

Salient features:

First hand knowledge principle or rule - The common witness must demonstrate that

(i) he was capable of perceiving the fact by one of his senses [e.g. he was not drunk at the time] and (ii) he actually observed this fact [i.e. he was present at the site at the material time].

2. Expert witness

Expert witness is a person who is skilled or trained in some scientific or technical subject and is capable of drawing conclusions and opinions from the facts observed by himself, or noticed by others. **E.g.** Forensic pathologist, an expert in DNA analysis, fingerprinting, firearms, handwriting or voice analysis etc [**S.45, IEA**]. The value of expert medical testimony was first recognized in the Bamberger Code (1507) and Caroline Code (1553) [please see above].

Salient features:

(1) **Areas in which expert witness gives evidence:** (i) upon facts which are admitted or proved by (a) himself (b) other witnesses at the trial (ii) on hypothetical questions. Doctor may or may not have first-hand knowledge of the actual case. These are questions based on stated assumptions (iii) on matters of common knowledge.

(2) **Rules to follow:** please see “conduct and duties of doctor in the witness box” below.

Table 7: Differences between a common witness and expert witness

S.No	Criteria	Common witness	Expert witness
1.	Description	Gives evidence about the facts observed or perceived by him	Gives evidence not only about facts observed but conclusions and inferences drawn from them, eg autopsy surgeon, fingerprint expert
2.	Type of person	Anyone. Even a completely uneducated person, or even a young child	Only a highly educated and skilled person
3.	Legal definition	Legally defined u/s 118, IEA	Legally defined u/s 45 IEA
4.	Drawing inferences from observations	Not allowed	Allowed
5.	Expressing opinion on observations made by others	Not allowed	Can express

3. Hostile witness

A ***hostile witness*** (adverse witness, unfavorable witness, unwilling witness) is one who conceals whole or part of the truth and offers adverse testimony against his own party.

Salient features:

(1) **Reasons for turning hostile** – bribed, threatened or won over by opposite party.

(2) **Who can declare a witness hostile** – The party who has called the witness will request the court to declare him hostile, whereupon court would do so.

(3) **Earlier testimony** - of hostile witness is rejected.

(4) **Leading questions** - can be asked during examination in chief from hostile witness [**S.154, IEA**].

(5) Both common and expert witness may become hostile.

(6) **Punishment** - Hostile witness may be prosecuted for **perjury u/s.193, IPC** [7 y+fine]. The Indian Evidence Act, 1872 does not expressly mention the term “hostile witness” anywhere. It is just a convenient term to address a witness who is concealing the truth.

XIII. PERJURY

Perjury means giving willful false evidence. The word “perjury” as such appears nowhere in Indian law. Indian law only mentions “false evidence”, which is synonymous with perjury.

Salient features:

(1) **Definition** — Perjury has 5 major components. Whoever, (i) being legally bound by an oath or (ii) by an express provision of law to state the truth, or (iii) being bound by law to make a declaration upon any subject, (iv) makes any statement which is false, and (v) which he either knows or believes to be false or does not believe to be true, is said to give **false evidence** [**S.191, IPC**].

(2) **Punishment for perjury:** (i) Normal trial - 7y+fine [**S.193, IPC**] (ii) Summary trial - 3 months, or ` 500 fine, or both [**S.344, CrPC**].

XIV. RECORDING OF EVIDENCE

(1) **Sequence** - The recording of evidence always in this order (i) Oath " (ii) Examination in chief is " (iii) Cross examination " (iv) Re-examination. Judge can ask questions at any stage. This order is defined in **S.138, IEA**. **S.137 IEA** defines examination-in-chief, cross-examination and re-examination.

(2) **Presence of accused** – Recording of evidence must be in the presence of accused, or his pleader [**S.273, CrPC]**.

(3) Evidence may be recorded by **audio-video electronic means** [**S.275, CrPC]**.

(4) **Recording in summons-cases** - the Magistrate makes a memorandum of the substance of witness' evidence or may dictate it in open Court. The memorandum is then signed by the Magistrate and forms part of the record [**S.274, CrPC]**.

(5) **Recording in warrant-cases** — All procedures as above, but here it can be in the form of question and answer [**S.275, CrPC]**.

(6) **Recording in Sessions court trials** – procedure same as in warrant cases [**S.276, CrPC]**.

A. Oath

An ***oath*** is a promise made in the name of God, that the witness will speak truth and nothing but the truth. In India, it is governed by The Oaths Act, 1969.

Salient features:

(1) **To whom administered:** All witnesses, jurors and interpreters [S.4(1), The Oaths Act, 1969]. **Exceptions** – (a) Child <12 y, [S.4(1), The Oaths Act, 1969]. (b) Atheist - If any person does not want to swear in the name of God [e.g. an **atheist**], he may solemnly affirm [S.5, The Oaths Act, 1969][solemn

affirmation]. It will have the same value as an oath [**S.51, IPC**, **S.3(37) The General Clauses Act**, **1897**]. (c) When accused is examined as a prosecution witness in a criminal trial [**S.313(2), CrPC;** S.4(2), The Oaths Act, 1969 [please see below " “trial procedure in Warrant and Summons cases].

(2) **Form** - Witness - I do swear in the name of God/solemnly affirm that what I shall state shall be truth, the whole truth and nothing but the truth.

(3) **Duty to speak the truth** – After taking oath, all persons are bound to state the truth [S.8, The Oaths Act, 1969].

(4) **Refusal to take oath** - 6 months simple imprisonment or ` 1000 fine or both [**S.178, IPC].**

B. Examination-in-Chief

Examination-in-chief [syn, direct examination, EIC] is the first examination of witness.

Salient features:

(1) **Done by** - Lawyer of the party which calls him (i) In a criminal case [e.g. grievous hurt, rape, murder trial]- since State is the prosecution party, the burden of proof lies on it. Since medical witness often proves guilt, he is usually summoned by the State. Public prosecutor [PP; **S.24, 25, CrPC**; lawyer representing the State] first examines the witness (ii) In a civil case – Party who calls the witness first examines.

(2) **Purpose:** (i) to elicit all relevant facts before the court, and the conclusions which the doctor has drawn from the facts. (ii) interpretation by the doctor of the findings of ancillary investigations, provided by scientific labs, analysts, serologists, toxicologists etc.

(3) **Before giving evidence:** (i) it is advisable that the doctor meets the PP and discuss the report, MLC, death certificate, photographs, preserved articles etc with him. Usually in important cases, as the witness reports in the court, PP would himself come to the witness, show him the case file, marked with several flags and discuss important points with him. There is nothing illegal in doing so. (ii) If witness wants to modify any of this conclusions, it is better to point it out to PP at this stage. (iii) Doctor may help the PP in framing proper questions in proper sequence, so that all essential facts may be elicited. (iv) Conversely PP is often able to tell the expected questions in cross-examination, which doctor may prepare beforehand.

(4) **Leading questions:** (i) A leading question [LQ] is any question which suggests its own answer, or which can be conclusively answered as "yes" or "no" [**S.141, IEA**]. (ii) Normally leading questions are not allowed during EIC and re-examination [**S.142, IEA**] (iii) Situations when LQ are allowed in EIC - (a) If the questions are introductory in nature or undisputed (b) If not objected to by the other party. (c) If objected to by the other party but allowed by the judge [**S.154, IEA**] (d) When the witness has turned **hostile**.

(5) **EIC is exempted** - In certain cases of offences of **rape,** sexual harassment (**S.354** and **354A, IPC**), attempt to disrobe(**S.354B, IPC**), voyeurism(**s354C IPC**), stalking(**S.354D, IPC**) and insulting modesty of woman (**S.509, IPC**)] [**S.164 (5-A) CrPC** (**ch 25)**].

C. Cross Examination

Cross examination of a witness is done by the lawyer of the opposite party opposite to the one which summoned him [**S.137, IEA**].

Salient features:

(1) **Objectives:** (i) To elicit facts favorable to the cross-examining party (ii) To test the accuracy of statements made by the witness [**S.146, IEA**] (iii) to discover who he is and what is his position in life [**S.146, IEA**] (iv) to discredit him, by injuring his character, including questions the answer to which might criminate him or expose him to a penalty [**S.146, IEA**] (v) to develop new or old facts (vi) To modify or explain what has been said (vii) To remove any undue or excessive emphasis which may have been given to a particular statement (viii) To try to weaken the evidence by showing that the evidence was biased, contradictory, ill-founded, inaccurate, inconsistent, influenced, untrustworthy and not impartial.

(2) **In a criminal case** - defense witness is cross-examined by the PP.

(3) **Leading questions** - may be asked in cross-examination [**S.143, IEA**].

(4) **Other questions** – Need not be confined to the facts elicited in EIC [**S.138, IEA**].

(5) **Competence and credibility of witness** – is often tested by questioning his qualifications, experience and number of autopsies, MLCs etc he has made. If the witness finds the questions objectionable, he must point it out to judge [please see "conduct and duties of doctors in the witness box" below].

(6) **Person called to produce a document** —cannot be cross-examined unless

and until he is called as a witness [**S.139, IEA**].

(7) Questions may be asked regarding the witness's previous convictions or questions tending to impeach his impartiality in order to shake his credit by injuring his character [**S.153, IEA**].

(8) **Impeaching credit of witness** — The credit of a witness may be impeached in cross examination [and with the consent of the Court during examination-in-chief and re-examination] (i) by the evidence of persons who testify that they, from their knowledge of the witness, believe him to be unworthy of credit (ii) by proof that the witness has been bribed (iii) by getting him to deliver contradictory statements [**S.155, IEA**].

(9) **Indecent and scandalous questions** — The Court may forbid any questions or inquiries which it regards as indecent or scandalous, unless they relate to facts in issue, or to matters necessary to be known [**S.151, IEA**].

(10) **Questions intended to insult or annoy** - The Court shall forbid any question which appears to it to be intended to insult or annoy, or which, though proper in itself, appears to the Court needlessly offensive in form [**S.152, IEA**].

(11) **Protection from self-incrimination** - If a witness is forced to give an answer admitting any guilt or crime on his part, such answer can not be used against him in any criminal proceeding [**S.132, IEA**].

(12) **Medical witness** – is neither a witness for prosecution, nor for defense. Thus even if an answer is favorable to defense, witness should immediately and unhesitatingly answer it.

(13) **Cross-examination as to previous statements in writing** – Sometimes witness's current statements may be contradictory to his own previously given statements in writing. His earlier statements may be used to contradict him [**S.145, IEA**]. If such contradictions are because of intervening medical advances, the witness must say so.

(14) **Hypothetical questions** – Witness may raise objection, but if court insists, he must answer.

(15) **Time limit** – There is no time limit for cross-examination and the witness may be cross-examined for days together.

(16) **Cross examination is a double edge sword** – i.e. it may injure the very case of the party cross-examining him. It happens if defense lawyer asks questions the answer to which he himself does not know. In such cases, the

answer may harm his own interest.

D. Re-examination

(1) **Conducted by** - the lawyer of the party who summoned witness.

(2) **Objectives:**

(i) To add details to statements, the witness made during CE

(ii) To clarify ambiguities

(iii) To correct any mistake.

(3) **Objections to testimony:**

(i) When an improper question is asked from witness during any stage, the opposing lawyer may immediately object by saying "I object", and must give reasons for his objections.

(ii) Witness must wait till the Court gives its ruling on objection. If Judge says "objection sustained" it means the object is right, and witness must not answer; if he says "objection overruled" it means the objection is wrong and the witness must answer.

E. Questions by Judge

(1) Judge may ask any question in any form, at any time [i.e. during EIC, CE, RE], about any fact relevant or irrelevant to clear up doubts **[S.165, IEA**].

(2) Can recall and re-examine any person at any stage [even if already examined] if his evidence is essential to the decision of the case [**S.311, CrPC**].

XV. TRIAL PROCEDURE IN WARRANT AND SUMMONS CASES

All offences punishable with death, imprisonment for life, or for a term exceeding 2 y are tried as **warrant cases**. All other cases are tried as **summons cases**.

Memory Aid 3: Summons Cases

Summons cases may be viewed as **s**imple cases

A. Warrant Case

Warrant-case refers to a case relating to an offence punishable with more than 2 years of imprisonment, or with death [***S.2(x), CrPC***]. **Ex** - Murder is a warrant case, because maximum punishment for this offence is death.

B. Summons Case

Summons-case refers to a case relating to an offence punishable with less than 2 years of imprisonment [**S.2(w), CrPC**]. **Ex:** Voluntarily causing hurt is a summons case, because maximum punishment for this offence is 1 year.

2. Medical Law and Ethics

I. INTRODUCTION

(1) The medical profession is guided by various legal statutes [medical law, eg MTP Act].
(2) Additionally, the Medical Council of India (MCI) has formulated The Indian Medical Council (Professional Conduct, Etiquette and Ethics Regulations) in 2002, which prescribes ethical guidelines to the doctors.
(3) World Medical Association also prescribes Codes of conduct from time to time.

II. ACTS

A. The Indian Medical Council Act, 1956

In 1933, the Indian Legislative Assembly passed the **Indian Medical Councils Act, 1933**. **Dr. B.C. Roy** was the first Indian President of Medical Council of India [MCI] in **1939**. In 1956, it was repealed, and a new Act known as **The Indian Medical Councils Act, 1956** [**IMC Act, 1956**] was passed. In **1964**, the Act was amended. This Act regulates medical practitioners trained in western system of medicine.

B. The Medical Council of India Regulations, 2000

MCI Regulations, 2000 have been made by the MCI [with previous sanction of the Central Govt] in exercise of the powers conferred upon it by **S.33 of IMC Act, 1956**. These are made to carry out the purposes of this Act, and provide for (a) the management of the property of the Council and the maintenance and audit of its accounts; (b) the summoning and holding of meetings of the Council, the times and places where such meetings are to be held, the conduct of business thereat and the number of members necessary to constitute a quorum; (c) the resignation of members of the Council etc.

1. Indian Medical Council

Indian Medical Council or the **Medical Council of India** (MCI) is constituted under The **Indian Medical Council Act, 1956**.

a. Constitution

(1) **Constitution** - According to **S.3 of the IMC Act, 1956**, MCI consists of

(i) **One member from each State** other than a union territory to be **nominated** by the Central Government in consultation with the concerned State Government

(ii) **One member from each University** to be elected from amongst the members of the medical faculty of that University

(iii) **One member from each State** in which a State Medical Register is maintained, to be **elected** from amongst themselves by persons enrolled on such register

(iv) S**even members to be elected** from amongst themselves by persons enrolled on any of the State Medical Registers, and

(v) **Eight members to be nominated** by the Central Government.

(2) A **president** and a **vice-president** - are elected from among these members.

(3) **Registrar** - The Council appoints a Registrar, who acts as secretary and may also act as treasurer, who looks after the day-to-day work.

(4) **Tenure** - The Council holds office for a term of five years.

(5) **Executive Committee** - The Indian Medical Council carries out its main purposes through an Executive Committee, which consists of **president**, **vice-president** and **7-10 other members**. In 2010, following some controversies, the President of India temporarily dissolved the Medical Council, and replaced

it with a seven-member Governing Council.

b. Functions

Functions of the Indian Medical Council are summarized in **Table 1**.

Table 1: Functions of the Indian Medical Council

1. Maintenance of medical register
2. Maintenance of standards of medical education
3. Undergraduate medical education
4. Postgraduate medical education
5. Recognition of foreign medical qualification
6. Appeal against disciplinary action
7. Warning notice

i. Medical register

The MCI maintains a register of medical practitioners, known as the ***Indian Medical Register*** [S.2(d) IMC Act, 1956].

Salient features:

(1) It contains the names of all doctors who are enrolled directly with MCI or with any State Medical Council.

(2) It is maintained by the registrar of MCI [**S.21, IMC Act 1956**; **S.61(1),** MCI Regulations 2000]. It is kept up-to-date by adding or erasing the names of medical practitioners, as the case may be.

(3) **Registered Medical Practitioner:**

(i) IMC Act 1956 - does not define this term anywhere, but some other Acts eg The Drugs and Magic Remedies Act 1954 and PCPNDT Act 1994 define it as a person who holds a degree specified in S.2(h)**,** IMC Act 1956.

ii. Maintenance of the standards of medical education

(1) The Council maintains standards of both the **Undergraduate** [S.19A of IMC Act, 1956] as well as the **Postgraduate** [S.20 of IMC Act, 1956] medical education.

(2) Any medical college or institution, imparting undergraduate medical education, should have a proper space, a properly trained staff, laboratories, equipment, library and other facilities.

(3) MCI **inspects** these facilities from time to time. If any college or institution does not have these facilities, the MCI makes a representation to the Central Government to withdraw recognition of medical degree awarded by that college.

(4) Every five years inspection is held by the MCI to see whether the standards are being maintained or not.

(5) **Schedules**: There are three important appendices to The Indian Medical Councils Act, 1956, known as Schedules. ***According to S.2(h), IMC Act*** 1956, a "recognized medical qualification" include only the medical qualifications mentioned in the three schedules.

(i) The **First Schedule** contains recognized medical qualifications granted by universities in India.

(ii) The **Second Schedule** contains medical qualifications granted outside India (recognized by the MCI)

(iii) **Third Schedule** has two parts - **Part I -** additional recognized qualifications granted by Indian medical institutions. **Part II** - additional recognized qualifications granted by foreign medical institutions.

iii. Recognition of foreign medical qualifications

If an Indian national has acquired a degree from a foreign medical college whose qualification is neither included in schedule 2 nor in part II of Schedule 3, he makes a representation to the Government of India, along with the relevant information (Course of study, syllabus, duration of the course etc). This representation is then forwarded to the MCI, which has an authority to enter into negotiations with any of the Medical Councils of the foreign countries, and can recognize such foreign qualifications on reciprocal basis. The Central Government may, by notification in the Official Gazette, amend part II of the Third Schedule so as to include such qualification therein.

iv. Appeal against disciplinary action

If name of any doctor is removed from the State Medical Register, he may appeal to the **Central Government** after exhausting all remedies under the State

Medical Council Act (within 30 days). The application should state the **grounds of the appeal** and be accompanied by all **relevant documents**. The Central Government confers with the MCI, and gives its decision which is binding on the State Government and the State Medical Council.

v. Warning notice

The Council prescribes standards of professional conduct. From time to time the MCI issues warning notices, describing acts amounting to **serious professional misconduct**.

2. State Medical Councils

a. Constitution

The State Medical Council consists of (i) **members elected by the registered medical practitioners** and those (ii) **nominated by the State government**. The members of the Council then elect the **president** and the **vice-president** amongst themselves.

b. Functions

The functions and working of the State Medical Council is similar to those of the MCI. The main functions are enumerated in **Table 2**.

Table 2: Functions of State Medical Councils

Maintenance of the medical register
Disciplinary control
Erasure of name
Penal erasure

i. Maintenance of the medical register

The Council appoints a **Registrar** who keeps a register of medical practitioners. This register contains the name, address, qualification and other particulars of all medical practitioners registered with that Council. Any new or an existing medical graduate can have his name entered on payment of prescribed fee. The

Registrar of the State Medical Council informs the Indian Medical Council of all the additions and other amendments in the State Medical Register made from time to time.

ii. Disciplinary control

If a doctor is found guilty of **professional misconduct** (see below), then the Council may exert a disciplinary action on that doctor.

iii. Erasure of name

The causes of erasure of the name of a registered medical practitioner are:

(1) Death of doctor (2) Doctor not traceable (3) Name entered by fault or fraud (4) Penal erasure

Penal erasure

(1) ***Penal erasure*** refers to removal of a medical practitioner's name from the Medical Register temporarily or permanently, as a ***penalty***.

(2) Done when doctor is guilty of **serious professional misconduct** (**infamous conduct**).

(3) Doctor can not practice medicine till his name remains removed from register.

(4) If name removed permanently, doctor ***can not practice for life***. This is known as the **Professional Death Sentence**.

(5) **Case studies:** Temporary erasure - British doctor **John Bodkin Adams** [1899 –1983] was found guilty of 13 offences of prescription fraud, lying on cremation forms, obstructing a police search and failing to keep a dangerous drugs register. His name was removed from the Medical Register in **1957**. He was reinstated in 1961 after two failed applications.

III. PROFESSIONAL MISCONDUCT (INFAMOUS CONDUCT)

Professional misconduct (syn. Infamous conduct, ethical misconduct, ethical negligence, ethical malpraxis) is that act of a medical man which would be reasonably regarded as disgraceful or dishonorable by his professional brethren of good repute and competence.

Salient features:

Strictly speaking, professional misconduct should include only those actions for which there are no punishments in IPC or in any other existing civil or criminal law, yet MCI or SMC can take action by removing the name of the doctor, eg dichotomy, putting up an extra large signboard, employment of touts etc. However the list of professional misconducts issued by The Indian Medical Council (Professional Conduct, Etiquette and Ethics) Regulations, 2002 [please see below] includes many acts for which there are punishments both in IPC [criminal misconduct] and MCI [ethical misconduct].

A. List of Professional Misconducts

The Indian Medical Council (Professional Conduct, Etiquette and Ethics) Regulations, 2002 enumerates some instances of **professional misconduct** in chapters **6** and **7. It is important to remember that a list of this nature can never be complete and exhaustive**. Depending on circumstances, even acts not mentioned in this list may be taken as examples of **infamous conduct**.

(1) **Adultery** or improper conduct or association with a patient.

(2) Conviction by court of law of offences involving **moral turpitude.**

(3) Issuance of **false certificates**, reports and other documents.

(4) Association with unqualified persons.

(5) Contraventions of drug acts and regulations.

(6) Selling a scheduled **poison** to the public.

(7) Performing an **illegal abortion** or enabling an unqualified person to perform an abortion.

(8) Issuance of certificate of efficiency in modern medicine to unqualified or non-medical persons.

(9) Contributing articles to the lay press and giving interviews regarding diseases and treatments **which may have the effect of advertising himself or soliciting practice**. But it is open to him to contribute articles which educate the public about hygienic and healthy living and also to give public lectures on health.

(10) Using an unusually large signboard. It is also improper to affix a signboard on a chemist’s shop or in places where he does not reside or work.

(11) **Disclosing the secrets of his patients** (obtained in the exercise of his profession) to others, except when bound by law (this subject is dealt with

later under the headings “Professional secrecy” and “Privileged communication”).

(12) **Refusing** treatment on **religious grounds.**

(13) **Publishing photographs** of his **patient** in any medical or other journal (by which his identity could be made out) without first obtaining the consent of that patient. However if the identity of such a patient is concealed, the consent of that patient is not required (to be sure, no patient under these conditions could sue the doctor either, because he wouldn’t have a way of knowing it was his photograph).

(14) **Publicly displaying his fees**, except in his consulting room.

(15) Using **touts** or agents to procure patients.

(16) **Receiving or giving commission** or other benefits from fellow doctors, traders, chemists, manufacturers etc. Giving of commission by one doctor to another for favor of recommending or referring the patient to him is known as **dichotomy** or **fee-sharing** or **fee-splitting**.

(17) **Talking disparagingly** about other colleagues or doing anything that means **unfair competition**.

(18) Lack of eagerness to respond in .

(19) Not attending a patient when he is already under his treatment.

(20) Addiction to narcotic drugs.

(21) Alcoholism.

(22) Using unsterile instruments, or not furnishing his patients with proper information about drugs or diet.

(23) **Covering** i.e. assisting someone who has no medical qualification to attend, treat or perform an operation on some person in cases requiring profession skill.

(24) Running an **open shop** for the sale of medicines.

(25) Writing prescriptions in a **secret formula** known to some particular pharmacy only.

(26) Commercialization of a **secret remedy**.

(27) Accepting any **gift** or **travel facilities** etc from **pharmaceutical companies**.

B. The 6 A’s of Serious Professional Misconduct

The above list is quite big. Of these, the **six most important offences** may be

described as the **6 A**'s. These are:

(1) **A**ssociation with unqualified persons – e.g. employment of touts to procure patients.

(2) **A**dvertising – It is improper to use an unusually large sign board to advertise oneself.

(3) **A**dultery–arising out of professional relationship.

(4) **A**bortion (unlawful) – Unlawful abortion includes prenatal determination of sex and then inducing abortion.

(5) **A**ddiction – Addiction to drugs or doctor supplying drugs of addiction (e.g. pethidine) to addicts. **Case study** - In 2011, Britain's General Medical Council cancelled the license of **Peter Brackenridge** for 6 months, as he prescribed Ibogaine [a hallucinogen] to a patient. Although it is used as a drug for drug addiction [**ch 46**] in some countries, it is unlicensed in the UK.

(6) **A**lcohol – Seeing patients under the influence of alcohol.

C. Punishment and Disciplinary Action

S.82 of Professional Conduct, Etiquette and Ethics) Regulations, 2002 " If a medical practitioner is found guilty of any of the above offences, his name may be erased from the medical register either temporarily or permanently (professional death sentence).

D. Oaths, Codes and Declarations Relating to Professional Misconduct

Historically, the doctors have been voluntarily taking oaths against committing an act of professional misconduct. Most important are:

Declarations of the World Medical Association (WMA)

World Medical Association is an association of the National Medical Associations of all countries. It held its 1st assembly in 1947 at Paris, France. Since then it has been holding annual assemblies at different cities. In several of these assemblies, important ethical declarations have been adopted, which are important for medical practitioners. Most important of these declarations are:

i. The Declaration of Geneva (1948)

The **Declaration of Geneva** (**Physician's Oath; modernized version of Hippocratic Oath**) was adopted by the General Assembly of the **World Medical Association** (an association of national medical associations) at **Geneva** in **1948**. It is a declaration of physicians' dedication to the humanitarian goals of medicine. This declaration became important in view of the medical crimes which had just been committed in ***Nazi Germany***.

ii. International Code of Medical Ethics (1949)

Adopted by the General Assembly of the **World Medical Association** at London in **October 1949**. Prescribes (i) Duties of Doctors in General (ii) Duties of Doctors to the Sick and (iii) Duties of Doctors to Each Other. Like the Declaration of Geneva, it also is a modern version of the Hippocratic Oath.

iii. Declaration of Helsinki (1964)

Please see "**Human Experimentation**" below.

iv. Declaration of Sydney (1968)

Adopted by the 22th World Medical Assembly at Sydney, Australia in August 1968. It is a statement on **when to pronounce death**

Memory Aid 1: Declaration of Sydney

Sy**d**ney"**D**eath

v. Declaration of Oslo (1970)

Adopted by the 24th World Medical Assembly held at Oslo, Norway in 1970. It is a statement on **therapeutic abortion -** Abortion should be performed only as a therapeutic measure.

Memory Aid 2: Declaration of Oslo

Oslo"Ab**o**rtion.

vi. Declaration of Tokyo (1975)

Adopted by the 29th World Medical Assembly at Tokyo, Japan in October 1975. Doctors will not take part in **infliction of torture**.

Memory Aid 3: Declaration of Tokyo

Tokyo"**T**orture.

IV. RIGHTS AND PRIVILEGES OF A REGISTERED

MEDICAL PRACTITIONER

A. Rights of a Registered Medical Practitioner

The rights are

(1) Right to choose his patient (EXCEPT IN EMERGENCY).

(i) Doctor need not accept all patients who come to him for treatment.

(ii) He may arbitrarily refuse to see a patient, even if there is no other physician around.

(iii) If patient is unable to pay doctor's fees, he may refuse to see patient. (a) in cases of **military necessity.**

(2) to add titles, qualifications etc after his name.

(3) Right to practice and dispense medicines.

(4) Right to possess and supply dangerous drugs to his patients.

(5) Right to recovery of fees.

(6) Right to give evidence as an expert.

(7) Right to issue medical certificates.

(8) Right for appointment to public and local hospitals.

B. Red Cross and Allied Emblems

Red cross is one of the three "Emblems of the International Red Cross and Red Crescent Movement". These are - the **Red Cross**, the **Red Crescent**, and the **Red Crystal** [**Fig 2.1**]. The Red cross emblem was first used in **1864**. [I] **Who can put up:** (1) Under **S.12** of the ***Geneva Conventions Act 1960*** (to which **India** is a **signatory**), no person can, without the approval of the Central Government, use the Red Cross or other allied emblems [Red Crescent, Red Crystal] for any purpose. (2) They can only be used by the armed forces medical services, their personnel, units, installations and means of transport. [II] **Punishment for unauthorized use** – `500 + forfeiture of property upon which the emblem was used [**S.13]**.

Fig. 2.1: From L to R—Red Cross, Red Crescent, Red Crystal

C. Duties of a Medical Practitioner

Medical practitioners have rights and privileges, but at the same time they have certain duties towards their patients and society in general. These have been outlined in various chapters of The Indian Medical Council (Professional Conduct, Etiquette and Ethics Regulations), 2002.

1. General duties and responsibilities

These are mentioned in **chapter 1** of The Indian Medical Council (Professional Conduct, Etiquette and Ethics Regulations), 2002.

(1) **Dignity** - Doctor should uphold the dignity and honor of his profession.

(2) **Modesty** - Should be modest, sober, pleasing in personality, should have patience. Should be eager to help patients.

(3) **Knowledge** - Should keep abreast of advances of medical science, and improve his knowledge and skills by referring to latest books, journals, attending conferences. **Case studies** - (i) **Vigneault v. Dr. Hewson Dental Co**. – Defendant dentist in preparation for the extraction of teeth used multiple injections of a local anesthetic " osteomyelitis of jaw developed " At that time, block method of injection was **commonly** being used " possibility of osteomyelitis was much less by this “advanced method” " defendant was held to be negligent in not keeping abreast of the developments in his profession. [Vigneault v. Dr. Hewson Dental Co., 15 N.E. (2d) 185] (ii) **Ferrell v. Ellis** - Defendant physician attempted to disclaim liability for negligence on the

ground that all country physicians know no better. The court held that the standard of care is based on present conditions of medical knowledge and not on past conditions acquiesced in by country folk [Ferrell v. Ellis, 129 Iowa 614].

2. Duties of medical practitioners to their patients in particular

These are mentioned in **chapter 2** of The Indian Medical Council (Professional Conduct, Etiquette and Ethics Regulations), 2002.

a. Duty to exercise a reasonable degree of skill and knowledge

As soon as a doctor agrees to treat a patient, a ***doctor-patient relationship*** is established, and he owes a duty to the patient to exercise a ***reasonable degree of skill and knowledge*** in treating his patient.

b. Legal duty to render service

(1) When a doctor agrees to attend a patient, the physician-patient relation is said to have been established.

(2) After this, the doctor is under obligation to attend to the case as long as it requires attention. He cannot leave the patient at his own will.

(3) **Doctor can withdraw treatment under following conditions:**

(i) treatment is no longer required;

(ii) patient asks him to withdraw treatment;

(iii) mutual assent of both parties;

(iv) after giving notice with a reasonable time for patient to find another doctor;

(v) physician himself becomes sick;

(vi) patient is found to be malingering;

(vii) patient is using remedies other than those prescribed by him;

(viii) patient ignoring his instructions;

(ix) patient seeing another doctor without informing him;

(x) patient is using contraband drugs, intoxicants or poisons;

(xi) patient not fulfilling his earlier financial obligations [not paying fees]**.**

(4) **Case studies:** (i) **Tadlock v. Lloyd -** Plaintiff employed physician to attend his sick child " physician diagnosed illness scarlet fever " The following day he was requested to come again " He did not come " said that disease must run its course " Several other requests were made " Finally, in response to an

urgent appeal, he did visit the patient " Child died shortly after his arrival " Doctor had legal duty to render service, but did not do so " found negligent [Tadlock v. Lloyd, 65 Colo. 40].

c. Prognosis

Doctor should neither exaggerate, nor minimize the gravity of his patient's condition.

d. Patient must not be neglected

e. Engagement for an obstetric case

(1) If a doctor **A** who was engaged to attend to an obstetric case is absent, and another doctor **B** is sent to conduct delivery, **B** is entitled for his professional fees.

(2) When **A** returns, **B** should leave the patient only after securing the patient's consent.

f. Duty in emergency

No doctor can refuse a patient in emergency.

g. Duty to give instructions

Doctor must give proper instructions to the patient or his relatives regarding administration of medicine and diet.

h. Duty to inform and warn

The doctor has the duty to inform and warn the patient regarding the possible side effects and harmful effects of any drug or device that he is prescribing.

i. Fees and other charges

Doctor should clearly display his fees and other charges on the board of his chamber and the hospitals he is visiting.

j. Professional secrecy (confidentiality)

Professional secrecy refers to the doctor's obligation to keep secret all information regarding patient that he comes to know in the course of his professional work.

Salient features:

(1) **Contract** - It is an implied term of contract between doctor and patient.

(2) **Information can be divulged** - with patient's consent.

(3) **Situations when doctor *should not reveal* condition of patient:** (i) **Press conferences** - If patient happens to be a celebrity, do not hold **press conferences** regarding his medical condition (ii) **Information to parents** - If patient is major, doctor should not inform about his illness to his parents, even if they are paying his professional fees. Doctor can communicate illness of a minor or mentally ill person to the legal guardians. (iii) **Information to third parties** – no information should be revealed, even if the third party happens to be near relative. There are some important exceptions to this rule [please see privileged communication below] (iv) **Husband and wife** – Condition of one can not be told to the other. (v) **When free treatment is provided** – Doctor still has to maintain professional secrecy (vi) **Information regarding dead person** – consent must be taken from nearest relative for imparting information to anyone (vii) **Reporting in a medical journal** – condition may be revealed to readers, but identity of patient must be hidden (viii) **Postmortem findings** – should be revealed only to the police. Not even to the closest relatives. However if a patient files an application under RTI Act, 2005 (Right to Information Act, 2005), the condition may have to be revealed and even entire PM report may have to be given to the relatives according to CIC (Central Information Commission) judgments.

(4) **Situations when doctor *may reveal* condition of patient** (please also see privileged communication below) (i) When one party sends another to doctor for examination with specific intent to discover illness – When one party A sends another party B to a doctor to discover illness, B knows that his illness may be reported to A, and with this knowledge he submits for examination; in such cases doctor can reveal illness to A (a) **Master sends servant** - to doctor to discover if he is having an illness (eg typhoid carrier status, so he may not be employed as a cook). Doctor can inform master (b) **Govt sends an employee or would be employee** - to doctor to discover any disability or illness to know if he is fit for govt service. Doctor can inform govt. (c) **Insurance company sends a prospective client** to know if he is having a life threatening disease. Doctor can inform insurance company. (ii) When person in police custody, undertrial prisoner, convicted prisoner is examined.

(5) **Punishment for not maintaining professional secrecy -** The patient can bring both a (i) criminal action (**u/s 499, IPC** – defamation) and (ii) civil action for damages (until and unless the case is one of privileged communication – please see below). Punishment for defamation is 2 years and/or fine [**u/s 500, IPC**] (iii) He can complain to MCI or State medical council, which can take disciplinary action.

k. Privileged communication

There occur some situations where the doctor has to divulge the patient's details to certain authorities. This divulgence of the details is known as ***privileged communication*** and is an ***exception to professional secrecy***. The situations where privileged communication holds true are as follows.

(1) **In Society's interest: (a)** Patient suffering from venereal disease " tries to enter a swimming pool " doctor advises him against doing so, as it can cause infection to others " patient does not agree " doctor can communicate about his illness to swimming pool manager **(b)** Patient suffering from color blindness " doctor sees him driving a truck " advises him against this, as it can cause accidents (inability to see red lights etc) " patient does not agree " doctor can approach road transport authority and communicate about his illness.

(2) **In patient's interest:** Patient has suicidal tendencies " Doctor informs the parents to keep watch over him, as he has tried to commit suicide " This is privileged communication (patient can not sue doctor).

(3) **In a third party's interest:** Doctor treating HIV patient "H" " He gets information that "H" is marrying a girl "G" " Doctor warns G that H is suffering from HIV " G refuses to marry H " H brings action against doctor for breach of confidentiality " Can not succeed, as the information was given in a third party's (G's) interest. This third party can be a legally married spouse too.

(4) **In doctor's own interest:** Patient brings negligence suit against the doctor " Court asks what illness did he treat " Doctor informs court " privileged communication.

(5) **Court of law**: A doctor has to divulge all the details about the patient if they are asked in the court of law. If he feels that he should disclose them openly, he should write the same and then hand the envelope over to the judge, but he can't refuse to divulge the facts.

(6) **Notifiable diseases**: if, during his practice, a doctor comes across a patient who is suffering from a Notifiable disease, it his duty to inform the same to the concerned authorities.

(7) **Suspected crime**: if the doctor comes across a patient who has committed or has intention to commit a crime which falls under any of the sections of IPC, he has to inform the same to the nearest police station or the magistrate [please see **ch 31** for detailed reasoning]. This is a **medicolegal case** [for definition of a medicolegal case, please see **ch 11**].

(8) **Employers**: if the person being examined is found to be suffering from a disease which can put the life of the people he is serving in danger, the doctor must try to persuade the person to not join his duty. If he does not agree, then the employer has to be informed. (a) **waiter or cook** is found to be suffering from an infectious disease " Condition must be reported to employer. (b) **driver** is found to be suffering from a disease like epilepsy, alcoholism, drug addiction, high blood pressure, color blindness " his condition must be reported to state transport authorities (c) **Teacher** suffering from infectious diseases (eg tuberculosis) " Condition must be reported to employers.

(9) **Medical for insurance**: if a doctor examines a person for his insurance policy, the person knows fully well that any adverse medical findings would be communicated to the insurance company. He has thus given his tacit consent for communication of medical findings to the insurance company. In such a situation, the doctor can give all the findings to the insurance company.

(10) **Waiver of professional secrecy** – When a patient gives his express permission to the doctor to disclose professional secrets.

3. Duties of medical practitioners in consultation

These are mentioned in **chapter 3** of The Indian Medical Council (Professional Conduct, Etiquette and Ethics Regulations), 2002. They concern both the **doctor who sends his patient** to the consultant, as well as the **consultant**.

Unnecessary consultations should be avoided - The doctor should ask for consultation from another doctor only when it is required. Patient’s consent - Whenever doctor asks for consultation he should talk about the same with the patient or his relatives and inform them about the reasons for the same. If they object, he should mention the same in the case records.

4. Duties of medical practitioners to one another

These are mentioned in **chapter 4** of The Indian Medical Council (Professional Conduct, Etiquette and Ethics Regulations), 2002.

Dependence of physicians on each other - A doctor should consider it a pleasure and privilege to render gratuitous service to all fellow doctors and their family dependents.

5. Duties of medical practitioners to the public in general and to the paramedical profession

These are mentioned in **chapter 5** of The Indian Medical Council (Professional Conduct, Etiquette and Ethics Regulations), 2002.

Physicians as citizens -

(i) Should disseminate advice on public health issues.

(ii) Should co-operate with the authorities in the administration of sanitary and public health laws and regulations.

6. Duties of medical practitioner in criminal matters

These are described by **s201, IPC** which states - Whoever, knowing or having reason to believe that an offence has been committed, causes any evidence of the commission of that offence to disappear, with the intention of screening the offender from legal punishment, or with that intention gives false information shall be punished [the quantum of imprisonment differs according to the seriousness of crime whose evidence was made to disappear]. **The doctor must report all cases of homicide, suicide and accident to the police**, because all these constitute offences, and fall within the meaning of **S.201, IPC**. Failure to do so would make him liable to prosecution **u/s 176, IPC** [Omission to give notice or information to public servant by person legally bound to give it]. Punishment is imprisonment of 6 months, or `1000 fine or both.

V. RIGHTS AND DUTIES OF PATIENTS

A. Rights of Patients

Right to treatment – All patients have a right to treatment regardless of their age, sex, religion, economic and social status. **Instances when doctor can refuse treatment - Communication made in good faith** — No communication

made in good faith is an offence by reason of any harm to the person to whom it is made, if it is made for the benefit of that person [**S.93, IPC]**.

B. Duties of Patients

Patient should **co-operate with the doctor** by giving information about the disease process, family and personal history.

1. Infringement by doctors of patient's rights

a. Paternalism

Paternalism [Gk. pater, father] is acting upon one's own idea of what is best for another person without consulting that other person.

Salient features:

(1) **Paternalism** and **patient autonomy** are two extremes of a continuum. At one end, the doctor's judgment and decision are supreme, and on the other, the patient's [Therapeutic privilege (please see below) lies somewhere in between]. Up to 19th century, paternalism was prevalent and legal. It is no more legal.

(2) **MLI** - Paternalism (in a conscious, mentally sound, adult patient), although done in the patient's best interests, is illegal. The doctor may be sued **u/s 350, IPC** [using criminal force].

b. Therapeutic privilege

Therapeutic privilege [TP] is the doctor's privilege not to reveal to the patient that part of the information about his illness, which he genuinely thinks would lower patient's morale and his will to live.

Salient features:

(1) **Precautions** – In order to save himself from later litigation doctor should (i) obtain expert consultation that patient is really emotionally disturbed and divulging information may cause more harm. Report of that consultation must be attached to the case sheet (ii) record in patient's case sheet (a) what he not disclosing and (b) why (iii) As far as possible, information should be told to patient's spouse or next of kin.

(2) The **prudent patient rule** or **test** – Used in two ways (i) Imparting information - This implies that the patient must be given such information about the procedure, side effects, and complications as might be required by a

prudent [reasonable] patient. TP extends only as far as prudent patient rule allows. (ii) Giving treatment – If doctor exercises TP, he must ensure that he imparts so much treatment as would be accepted by a prudent patient, if he genuinely had all information about the disease.

(3) **Examples of TP** – not telling about malignancy, unavoidable fatal lesion. HIV does not come under TP. Patient must know about it, so he can take precautions about not spreading disease.

2. Abuse by patients of their rights

Malingering

Malingering or ***shamming*** is conscious planning, feigning and pretending a disease for the sake of some advantage.

Salient features:

(1) **Advantages** - Malingering is done by people to have following advantages: (i) by **workmen** to claim compensation (ii) by **policemen** or **military** people to prevent hard postings (iii) by **prisoners** to avoid hard labor, or capital punishment (iv) by **govt. servants** to prevent transfers (v) by **businessmen** to avoid contracts (vi) by **beggars** to attract public sympathy.

(2) **Diseases feigned** - Virtually any disease may be feigned, but those most easy to feign are amnesia, aphasia, bleeding (may injure anus or vagina to produce anal and vaginal bleeding respectively. May take coumarin to induce bleeding diathesis), bruises (made by juices of irritant plants, eg Dhobi's marking nut), burns, chest pain (may ingest digitalis to alter his ECG pattern), diabetes, diarrhea, dyspepsia, epilepsy, hematemesis (may injure his nasopharynx with a sharp instrument, swallow the blood and regurgitate it in front of doctor), hemoptysis, intestinal colic, jaundice (eating large amounts of carrot produces carotinemia, which simulates jaundice), lumbago, mental illness, neurasthenia, ophthalmia (pour juices of irritant plants in eyes, eg ratti), paralysis, rheumatism, sciatica, ulcers and vertigo. Sometimes patients may exaggerate a disease they are already suffering from.

(3) **M/L Importance:** (i) If the patient gets a medical certificate by narrating false symptoms, and uses it for any purpose, he may be sued **u/s 198, IPC** [punishment 7 y+fine] (ii) Cheating – May be sued **u/s 415, IPC** [cheating]. Punishment 1 y or fine or both [**S.417, IPC**] (iii) Must be differentiated from **perjury** [please see ch 1]. Malingering is not perjury, as patient does not

speak a lie under oath.

VI. TORT

A ***tort*** [Latin tortus, twisted] is a civil wrong that results when a defendant engages in conduct that harms another person. **Ex of tort** -

(1) A drives car recklessly and hits another vehicle " A has committed the tort of negligence.

(2) A negligently ligates ureter of B during an abdominal operation " A has committed the tort of medical negligence. B claims compensation " A is tortfeasor; B is plaintiff.

(3) A writes false things on the internet that harm another person's reputation " A has committed the tort of defamation.

VII. PROFESSIONAL NEGLIGENCE

A. Definition

Professional negligence, medical negligence or ***malpraxis*** is absence of reasonable care and skill, or willful negligence of a medical practitioner in the treatment of a patient, which causes injury or death of the patient.

Salient features:

(1) **Alternative definition** - doing something that one is not supposed to do [act of commission] and failing to do something that one is supposed to do [act of omission].

(2) **Reasonable care:** (i) Elements - Sometimes also called "due care". (a) includes such reasonable care and attention that the patient may require. (b) Must anticipate and appropriately manage known, expected or foreseeable events and complications of patient's disease and treatment (c) It should be proportionate with (i) qualification of doctor - [eg an MCh is expected of a higher level of skill than an MS, who in turn has a higher level of skill than MBBS]. For the same level of negligence, an MCh may be held responsible, but not MBBS (II) known inability of the patient to take care of himself – eg unconscious patient requires higher level of care and attention than a

conscious patient. (ii) When breached - Reasonable care is breached in 2 situations (a) when doctor unjustifiably deviates from accepted methods, practices, procedures and treatments. (b) when he used accepted methods but does so unskillfully.

(3) **Free service** - Doctor provided free medical service " injury or death of patient occurred " doctor still liable to be prosecuted under criminal law, but cannot be made to pay compensation under COPRA (please see below)].

(4) **Components of medical negligence** - For a case of medical negligence to be established, the following components must be present (**4Ds**).

1. Duty

The doctor begins to owe a duty towards a patient

(1) as soon as he agrees to treat him.

(2) when he is in emergency. A **doctor-patient relationship** between the doctor and the patient is established at that point in time. Doctor-patient relationship is **not formed** when patient is not in emergency and the doctor did not agree to treat the patient.

2. Dereliction of duty

Once the presence of duty has been established, there has to be a dereliction of duty on the part of the doctor, i.e. the doctor should have been negligent in performing his duties towards the patient.

3. Damage

(1) The damage must occur as a result of dereliction, and it must be foreseeable.

(2) Even if doctor is negligent, patient cannot sue him for compensation, if no damage has occurred. He can however be sued criminally **u/s 336, IPC** [please see below].

(3) Some examples of possible damages are:

(i) **Aggravation** - of a preexisting condition

(ii) Diminishing patient’s chances of recovery

(iii) **Expenses incurred** – eg hospital and medicine expenses, special diet etc.

(iv) **Pain and suffering** – causing either physical or mental [embarrassment, fright, humiliation] pain or increasing it

(v) **Loss of earning** – due to absence from work

(vi) **Loss of potency**
(vii) Prolonging his illness
(viii) **Reduced enjoyment of life** – eg loss of a limb or sense
(ix) **Reduction** in expectation of life
(x) Death.

4. Direct causation

Damage must result directly from dereliction [**proximate cause**], and not from any other cause. ***Proximate cause*** refers to a cause, which in natural and continuous sequence, unbroken by any efficient intervening cause, produces the injury, and without which the injury would not have occurred. It may also be conceived of as a series of "**falling dominoes**". If the **final domino [damage]** can logically fall by "pushing" the first domino **[dereliction]**, the "**push**" is the **proximate cause [direct causation]** of fall of **final domino** [**Fig 2.2**]. **Table 3** lists some differences between professional negligence and professional misconduct.

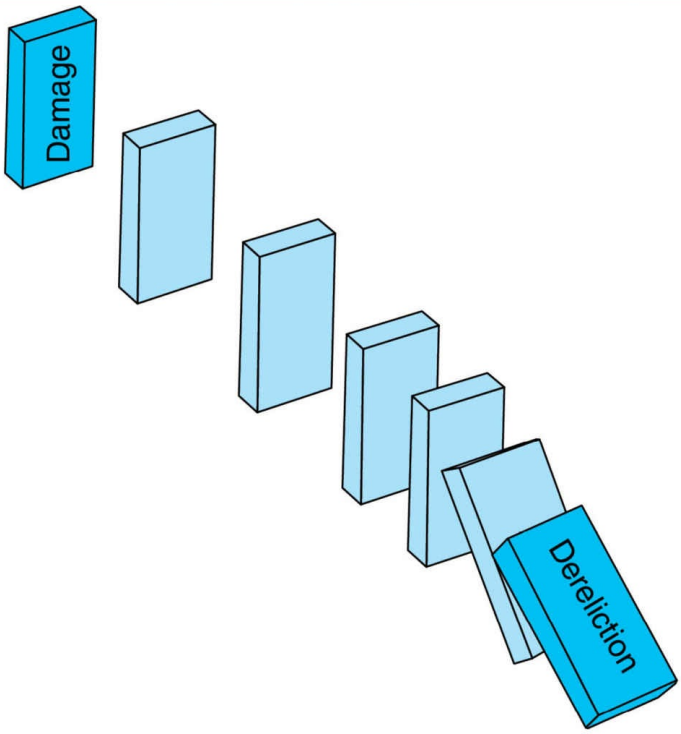


Fig. 2.2: Direct causation as understood by falling dominoes. As derelication occurs (falling of the first domino), damage will finally occur (falling of the last domino) as a direct cause of dereliction. Please also compare with **Fig. 2.3**

Table 3: Differences between Professional Negligence and Professional Misconduct

S.No	Trait	Professional negligence	Professional misconduct (syn. Infamous conduct, ethical negligence, ethical malpraxis)
1.	Offence	Absence of reasonable care and skill in the treatment of patient	No absence of care and skill, but the doctor did not adhere to the ethical standards befitting a doctor
2.	Duty of care towards the patient	The doctor must have a duty towards his patient, which he neglected	No such duty is necessary. Doctor may be accused of professional misconduct even in the absence of such duty, e.g. dichotomy, or when he puts up an unusually large sign board
3.	Damage to the patient	Must be present	Need not be present
4.	Trial by	Civil or Criminal Court	Medical Council of India or State Medical Councils
5.	Punishment	Imprisonment or fine	Warning or erasure of name from the medical register.
6.	Appeal	In higher court	To State or Central Governments

B. Types of Negligence

Table 4 enumerates various types of negligence.

Table 4: Types of negligence

1. Medical Negligence [doctor's negligence]
   (a) Civil negligence (b) Criminal negligence
2. Patient negligence
3. Contributory negligence
4. Composite negligence
5. Corporate negligence

1. Medical negligence (MALPRAXIS)

a. Civil negligence (Civil Malpraxis)

Civil negligence is said to occur when patient demands ***monetary compensation*** for the damage that he has suffered due to the doctor's negligence. Civil negligence comes under **tort law** [please see above].

Salient features:

(1) **Question of civil negligence also arises** - When patient refuses to pay doctor's fees alleging negligence, and doctor brings a civil suit against patient to recover his fees.

(2) **Court** - The case goes either to civil court or to consumer forum.

(3) **Punishment** - The doctor only has to pay whatever compensation the court decides. There is **no criminal liability** for him and he cannot be sent to jail for his action.

(4) **Additional punishments** - However if the patient complains to medical council also, it can take action against the doctor and can punish him accordingly (e.g. removal of name from medical register temporarily or even permanently).

(5) **Standard of proof** – Rests on **preponderance of evidence** [**syn** "balance of probabilities"] i.e. there should be >50% probability of the doctor being negligent in his conduct [please correlate with "Standard of proof" in **ch 1**]. It is the duty of the patient to prove negligence.

(6) **Defenses**

(i) Informed consent

(ii) Contributory negligence [please see below].

b. Criminal negligence (Criminal Malpraxis)

Criminal negligence is that when patient complains to the police regarding the misconduct of the doctor, and the police registers a case against the doctor.

Salient features:

(1) **Who fights the case** - State i.e. the government [not the suffering patient] fights the case against the erring doctor.

(2) **Punishment** - The doctor is prosecuted under the various sections of the Indian Penal Code (IPC) and if found guilty the doctor is liable to pay **damages as well as** can be **sentenced to a term in the prison**.

(3) **Standard of proof** - In criminal negligence - in contrast to civil negligence - the burden of proof that is required to prove the guilt is "**beyond reasonable doubt**". There should be >99% probability of the doctor being guilty. The test is therefore more severe than the one applied for criminal negligence.

Classification

Cases of **criminal negligence** can be classified as follows:

(1) **When no injury to the patient occurs** (**S.336, IPC**) – Negligent action but no injury " Patient cannot claim compensation, but he can still lodge a criminal complaint against him under S.336**,** IPC if the doctor's conduct was so rash or negligent as to endanger patient's life or his personal safety. Punishment is imprisonment of 3 **months**, or `250 fine, or both.

(2) When **injury occurs to the patient –**Further subdivided into following: (i) **When hurt is caused to the patient** (**S.337, IPC**) – Negligent action " Patient suffers **hurt** [for definition of hurt, please see chapter 11] " S.337, IPC. Punishment is imprisonment of 6 **months**, or **`500** fine, or both. (ii) **When *grievous hurt* is caused to the patient** (**S.338, IPC**) - Negligent action " Patient suffers **grievous hurt** " **338, IPC**. Punishment is imprisonment of **2 years**, or **`1000 fine**, or both. **When *death* is caused** (**S.304A, IPC**) – Negligent action " Death of patient " S.304A, IPC. The punishment is imprisonment of **2 years**, or fine (**any amount**) or both.

2. Patient negligence

Patient negligence is outright negligence by patient only. Doctor is not

negligent.

Salient features:

(1) Doctor is not liable in such cases.

(2) **Examples:** (i) Patient does not give correct history (ii) starts an alternative form of treatment without informing doctor (iii) does not follow doctor's instructions. (iv) discontinues treatment (v) refuses to take treatment (vi) LAMA – leaving [hospital] against Medical advice.

3. Contributory negligence

Contributory negligence is the name given to a situation, where ***both*** doctor and the patient are negligent.

Salient features:

(1) **Examples:** (i) Doctor applies tight plaster cast on patient's leg [doctor negligent], but instructs him to report numbness " Patient develops numbness in his toes " does not inform doctor [patient negligent] " suffers permanent injury " Doctor entitled to the defense of contributory negligence. (ii) Doctor prescribes drug " fails to inform the patient of its side effects [doctor negligent] " patient starts getting side effects " fails to inform the doctor [patient negligent] " suffers injury " contributory negligence.

(2) **Burden of proof** – lies with the doctor.

(3) **Doctrine of comparative** [or proportionate] **negligence - Quantum of injury** caused by patient's and doctor's negligence is decided by the court and the compensation awarded accordingly. If, say, patient's negligence is responsible for 80% of his injury, and doctor's negligence for 20%, patient would be entitled only to 20% of the compensation.

(4) Contributory negligence is a good defense in civil cases, but cannot be taken as a defense in criminal cases.

(5) The doctrine of contributory negligence is subject to following exceptions (i) ***Last clear chance doctrine*** - Both doctor and patient are negligent. But if it is shown that the defendant (doctor) had a "Last Clear Chance" to avert the damage caused to the patient, and he did not avail of this chance, he loses his defense of contributory negligence. **Example**: Doctor prescribes drug to patient " fails to inform of side-effects [doctor negligent] " Patient develops rashes, but fails to inform doctor [patient also negligent, so concept of contributory negligence comes into play] " Patient visits doctor on the next

date " doctor sees rashes " fails to take remedial action " Patient suffers injury " sues doctor " Doctor takes plea of contributory negligence. **Analysis** - Patient can allege that the doctor had the “last clear chance” to avoid injury, because on the patient’s next visit, the doctor did see his rashes and yet did not do anything. (ii) The ***Avoidable Consequences Rule*** - Both parties are negligent, but patient could have avoided his own injuries by being more careful [It is in fact a variation of the Last clear chance doctrine” in as much as he himself had the “last chance” to avoid injuries]. He could have avoided the consequences of doctor’s negligence, but he did not carefully act upon it. So he himself is responsible for his injuries and doctor need not pay. **Example**: Doctor does not apply bandage and antibiotics to a gaping lacerated wound (doctor negligent) " He however advises the patient not to allow the wound to get contaminated, and visit him next week to get his wound examined again " patient comes home and thinks that applying cow dung would heal his wound faster " applies cow dung on his wounds " develops pus, but he does not contact the doctor. Nor does he visit the doctor next week, as was advised by the doctor (patient negligent) " Patient suffers injury. **Analysis** - Here both doctor and patient are negligent and normally the compensation should have been shared between the doctor and the patient. But since the patient aggravated his own injury by doing something which was avoidable, the doctor would be covered under the “avoidable consequences rule” and he does not have to pay anything. (iii) **Good Samaritan doctrine** - One who assists another who is in serious danger cannot be charged with contributory negligence, unless the assistance is rash or reckless.

4. Composite negligence

Composite negligence is said to occur, when a patient suffers injury as a result of the combined negligence of two or more doctors.

5. Corporate negligence

Corporate negligence is the negligence of a corporation [hospital]. The individual doctor may or may not necessarily be liable in this case.

C. Important Concepts Related to Medical Negligence

1. Vicarious liability

Vicarious liability [syn ***vicarious responsibility, VL]*** means that a person is liable for the wrongful acts or omissions of another.

Salient features:

(1) **Respondeat superior [RS]-** [lit, let the superior reply] Vicarious liability arises under the principle of respondeat superior. The principle of RS holds that the employer is responsible not only for his own negligence but also for the negligence of his employees, if such acts occur in the course of the employment and within its scope. (i) Also known as **"captain of the ship" doctrine**. (ii) RS becomes applicable when the superior had the "right, ability or duty to control" the activities of a violator. (iii) Difference between the two terms - ***RS*** is the principle, while ***VL*** is the liability it puts on a superior. (iv) RS principle ensures (a) that employers engage the best possible talent in their hospital (b) that patient gets enough compensation due to him. Individual negligent doctor may not have enough funds to pay the patient, but hospital has. Also known as "**doctrine of deep pockets**". Hospital has more money or "deep pockets" so to say to compensate the patient.

(2) **Preconditions:** (i) There must be an employer – employee relationship (ii) Employee's conduct must occur within the scope of his employment [i.e. employee did what he was legally supposed to do] and (iii) It should occur while on the job.

(3) **Compensation:** (i) Both the negligent doctor as well as his employer may be asked to compensate patient by the court. (ii) Employer may engage in a separate "third party proceeding" to recover money from the negligent doctor.

(4) **Relationships when one is vicariously responsible for the negligence of other:** (i) Senior doctor - responsible for negligence of junior doctor, resident, intern or an assistant working under his supervision (ii) Hospital - responsible for negligence of its doctor (iii) Two or more doctors working in partnership – each is responsible for negligence of other (iv) Two or more doctors attending on a patient independently – each may be help liable for the negligence of other, if negligence produced symptoms and signs which an ordinary doctor should have noticed and objected to. (v) Doctor referring his patient to an incompetent doctor knowingly – Referring doctor is vicariously responsible for negligence of incompetent doctor. (vi) Doctor writing illegible prescription and pharmacist dispensing wrong drug – doctor is vicariously liable. (vii)

Doctor instructing prescription over telephone and pharmacist dispensing wrong drug – doctor vicariously responsible (viii) When an employee has been temporarily borrowed from another – please see "Borrowed servant doctrine" below.

(5) **Relationships when one is *not* vicariously responsible for the negligence of other:** (i) Surgeon and anesthetist (ii) Qualified nurse acting independently, not under the direct supervision and control of doctor – doctor not vicariously responsible (iii) Doctor referring his patient to a competent doctor – referring doctor not responsible [cf referral to an incompetent doctor]. (iv) Doctor referring an already injured patient due to his negligence to a new doctor or hospital – the latter is not vicariously responsible (v) Doctor writing clear, legible prescription and pharmacist dispensing wrong drug – doctor not liable.

2. Borrowed servant doctrine

Borrowed servant doctrine states that if an employee is temporarily borrowed by an employer from the principal employer, then the temporary new employer becomes vicariously liable for any negligent act done by the employee.

Salient features:

(1) **Principle** – is that the lending employer temporarily surrenders control over the employee. The new master (i) gains control over the employee (ii) directly supervises the employee (iii) can give orders to him.

(2) The new master is responsible for the actions of hired employee [borrowed servant] only as long as he is under his direct supervision.

(3) After the employee is returned, the original employer becomes responsible for his negligent actions again.

(4) **Ex** – Resident [R] works on a regular basis in a big private hospital [H]. An independent private specialist [S] hires an OT in H and also services of R " R commits an act of negligence when he had been hired by S " Although R is a regular employee of H, the latter is not vicariously responsible; S is vicariously responsible, because negligence occurred when R was in direct supervision of S.

3. Res ipsa loquitur

Ordinarily the professional negligence of a doctor must be proved by the patient. ***Res ipsa loquitur*** [the thing or fact speaks for itself] is an important exception to

this rule, because here the professional negligence is so glaring and obvious that it speaks for itself.

Salient features:

(1) **Conditions to be satisfied before this rule can be legally applied:** (i) Injury to the patient would not have occurred in the absence of negligence (ii) Doctor had exclusive control over the injury producing instrument or treatment (iii) the patient was not guilty of contributory negligence.

(2) **How proved** - Generally without expert medical evidence. Common knowledge or experience of a layman should be enough to understand and appreciate that the negligence must have caused the injury ["Doctrine of common knowledge" or "Common knowledge doctrine" – please see below].

(3) **Applicable** - to both civil and criminal negligence.

(4) **Generally used** - when there is no direct evidence of the doctor's negligence.

(5) **Examples:** (i) Antiseptic – Failure to apply to an open cut (ii) Anti tetanus serum – failure to give in cases of gross injuries. Patient developing tetanus. (iii) Artery forceps, swabs etc - left in the abdomen. (iv) Blood transfusions – mismatched (v) Breaking of needles (vi) Burns – (a) from cauterizing tool at unintended places (b) from application of hot water bottles for excessively long time (c) from negligent application of X-ray therapy (vii) Fluids – failure to give in obvious dehydration (viii) Giving poisonous medicines recklessly (ix) Prescribing overdose of medicines (x) Prolonged splinting; too tight a bandage or plaster – loss of use of a limb (xi) Wrong amputation – eg right leg [eye, kidney etc] has been amputated instead of the left (xii) Wrong teeth removed (xiii) Wrong person -operated upon.

Doctrine of common knowledge

Doctrine of common knowledge" states that when negligence is of such obvious nature that even a layman with ordinary common knowledge having no technical expertise can appreciate it, then evidence of a medical expert to prove negligence is not required.

D. Defenses Against Negligence

1. Actual denial

The doctor denies his responsibility straightaway and that he discharged his

duties according to the prevailing standards. His records should be proper and complete to prove this assertion.

2. Calculated risk doctrine

Calculated risk doctrine [syn, ***doctrine of professional medical judgment***] asserts that doctor is not liable if he took a reasonable and calculated risk to save life of patient and in the process patient suffered injury.

Salient features:

(1) **Principle** (i) Medical profession is a highly risk prone area and treatment often involves a calculated risk and possible disagreement among experts as to whether the risk was warranted or not. (ii) If a physician chooses for a patient a course of treatment within a range of medically accepted choices, after a proper examination and evaluation, the calculated risk doctrine will insulate him from liability. (iii) To impose liability, a plaintiff must show that the doctor's decision was something that fell below the certified medical determination.

(2) It is an important exception to the principle of res ipsa loquitur, since it covers inherent risks of a medical procedure or operation.

(3) **What doctor must prove** – He must produce expert evidence or statistics that the line of treatment he chose was a medically acceptable procedure under the conditions, and that it had unavoidable risks.

(4) **Examples** - adverse reactions to penicillin, streptomycin, IV antibiotics, immunizations of all sorts, blood transfusions, novocaine, X-ray dye studies, injections into the spinal canal, injections of gold for rheumatoid arthritis, and injections of cortisone directly into the joint.

(5) **Case study:** Siverson v. Weber (1962) - Dr. Charles Weber, a gynecologist, performed hysterectomy on Lillian Siverson. 10 days later a vesicovaginal fistula developed. Siverson brought a case of negligence against Weber. Citing the doctrine of res ipsa loquitur, she asserted that she did not have to prove anything. Merely the fact that hysterectomy was performed and that a fistula developed after that meant that "the facts themselves spoke of negligence". The court however said that fistulas could occur even though the surgeon had exercised the care and skill generally possessed and exercised by reputable gynecologists and the development of a fistula, although rare, is considered an inherent risk of the operation. It was a calculated risk taken by Dr. Weber

[Siverson v. Weber 57 Adv. Cal. 881, 372 P.2d 97 (1962)].

3. Contributory negligence

Please see above.

4. Emergency

In emergency doctor may take some drastic measures to save life, which may cause injury eg fracture of ribs during cardiac massage. Falls under calculated risk doctrine also.

5. Law of limitation

Law of limitation specifies that a claim for compensation can be brought against a doctor within a specified period. If the claim is brought after that period, the doctor can take the defense of limitation. For cases related to consumer protection act, the aggrieved patient must file a complaint within a period of 2 years of discovery of alleged negligence.

6. Medical misadventure

Medical misadventure [syn, Medical maloccurrence] is defined as a bad or undesirable outcome that is unrelated to the quality of care provided.

Salient features:

It is an unintentional and unforeseen accident, disaster or mischief, which occurs due to individual biological variations.

Types: (i) **Therapeutic misadventure** [TM]– when treatment is being given [eg serious drug reaction to a common drug like aspirin; breaking of a needle during IM inj due to sudden muscular contraction; patient dying of shock during pleural or peritoneal tap]. (a) History of sensitivity - (i) should be obtained before injecting any drug, especially antibiotics, biological products and sera. (ii) Negative history and even negative tests do not however rule out possibility of anaphylactoid reactions and death. Patient should be specifically informed of this orally and in writing (b) In the event of a reaction - (i) an alternate drug should be advised. (ii) If doctor fails to stop offending drug, he cannot take this defense. (c) If a drug causes drowsiness or other accident prone side-effects, doctor should clearly inform the patient, and advise to avoid driving, working near dangerous machines etc. For legal immunity, it is advisable to even write

this information on the prescription sheet. (ii) **Diagnostic misadventure** - When diagnostic procedures are being carried out [eg fatal allergic reaction to an ionic contrast media before taking CT]. (iii) **Experimental misadventure** – Where a patient was a participant in an experimental study.

7. No duty owed towards the patient

If the doctor had not agreed to treat patient, no duty exists.

8. Novus actus interveniens

Novus actus interveniens [Syn - Nova Causa Interveniens, an unrelated act intervening] refers to a situation, where the doctor has been negligent, but a completely unexpected and unforeseen act happened which further worsened the patient's condition.

Salient features:

(1) The new act intervening should be completely unexpected and unforeseen. Sometimes referred to as an "Act of God".

(2) **Breaks the chain of causation** between the act of negligence and the resulting damage [**Fig 2.3**]. Thus injury no more remains a proximate cause of doctor's negligence.

(3) **Analogy** – Student does not prepare for exams thinking that he can **cram up** on the last night"On the last night there is suddenly a failure of electricity [an act of God]"Student cannot cram up"fails. **Analysis** – Student has to face **consequences at 2 places** – in the examination results [a **stricter option**, just like "**criminal negligence**"] and before his parents [a **softer option**, just like "**civil negligence**"] His plea of NII [electricity failure on last night] will not help him pass the examination [loss of case in a stricter option], but he may perhaps convince his parents about his stand [a softer option].

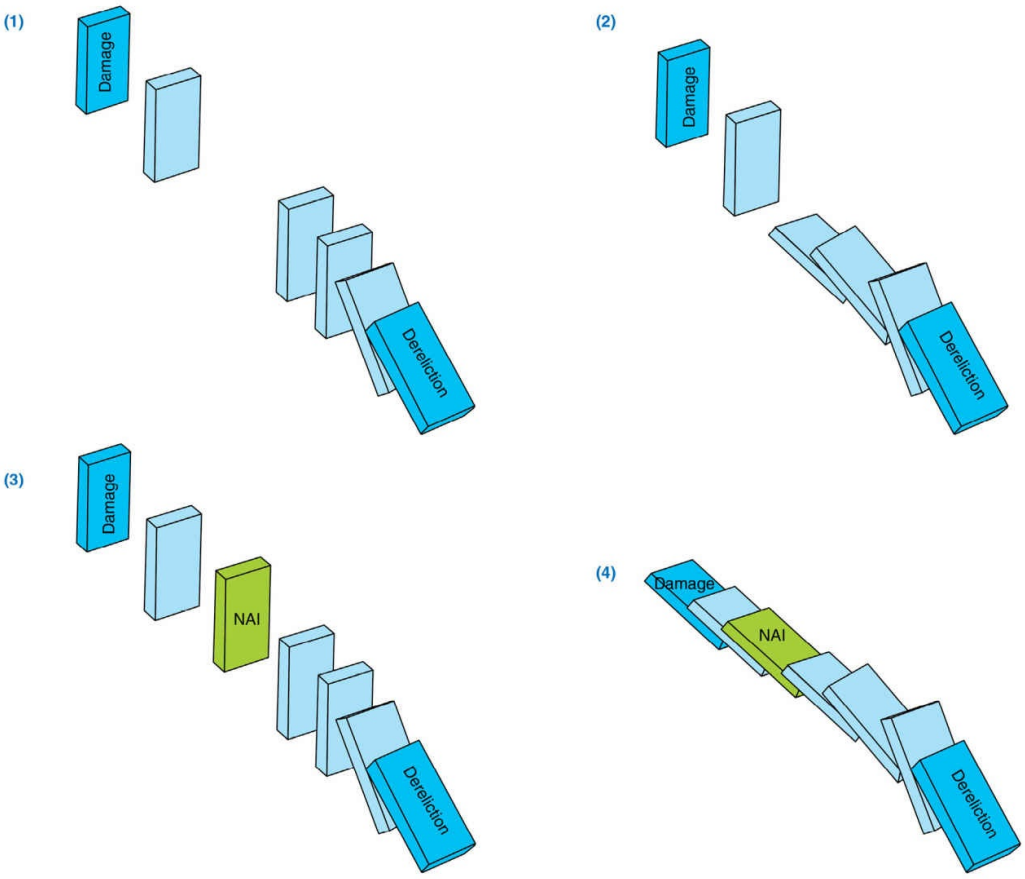


Fig. 2.3: Concept of proximate cause and novus actus interveniens as understood by falling dominoes. (1) & (2) Dereliction may not necessarily result in damage as shown here. (3) & (4) However sometimes an unexpected event [Novus Actus Interveniens (NAI)] intervenes, causing the damage to occur. Pl. see text for details. Pl also compare with **Fig. 2.2**

9. Patient negligence

Please see above.

10. *Res judicata*

(1) Res judicata is a Latin term meaning "the thing has already been decided" [**S.300, CrPC**]. Also known as the "doctrine of double jeopardy".

(2) If a question of negligence against a doctor has been decided in one court [eg a consumer court], the patient cannot start another proceeding in a different court. Appeal to the higher court however is allowed. **Ex -** A patient who has paid for his treatment would prefer to lodge a complaint with District forum, since decisions are quicker. On loosing the case, he can appeal to higher courts, i.e. State Commission, National Commission and Supreme Court. But after getting decision from the highest court, he cannot now lodge a fresh complaint with normal civil court. If he does, the doctor can take plea of res

judicata.

11. *Volenti non fit injuria*

Volenti non fit injuria [Latin, to a willing person, injury is not done] is a legal doctrine which means that if someone willingly places himself in a position where harm might result, he will not be able to bring a claim against the other party.

Salient features:

For this claim to succeed the doctor must show that an informed consent had been taken and all side-effects had been explained, eg if patient sustains radiation burns during therapy, and he had earlier been explained about the risks, he cannot claim damages.

12. Countersuits by doctors

Doctor files a **countersuit** against the patient. Should only be resorted to when the doctor is absolutely sure there was no fault and the litigation is frivolous and vexatious. It is not exactly a defense, but may deter patients to go in litigation in future, and more importantly, settle a compromise deal with the patient and withdraw the case.

Memory Aid 4: Defenses against negligence

C^3D^2E – LMN^2 – **P R** i **V** ate

C-Calculated risk, Contributory negligence, Countersuits
D-Denial, Delegation
E-Emergency
L-Limitation
M-Misadventure
N-No Duty, Novus
P-Patient negligence
R-Res judicata
V-Volenti non fit injuria

VIII. MEDICAL RECORDS

Medical record [***MR***] [syn, health record, medical chart] is a document which records, documents and chronicles patient's history, medical examination, laboratory and ancillary investigations, treatment advised, operation notes,

discharge summary, details of subsequent visits and any other data regarding patient treatment.

IX. PRODUCTS LIABILITY

Products liability occurs, when a manufacturer supplies a defective medical instrument or gadget (product) to the doctor or hospital; doctor inadvertently uses the defective product causing injury to the patient.

Salient features:

(1) **Instances when product liability occurs:** (i) Instruments - (a) Faulty, defective or negligently designed medical or surgical instruments [eg a defective defibrillator] (b) wrong or inadequate operating instructions (ii) Medicines - (a) Components incorrect (b) potency too low or too high (c) instructions of taking wrong, eg oral medicine instructed to be instilled in eye (iii) Defective prosthetic devices - (a) artificial heart valves, limbs etc (b) pacemakers.

(2) **Legal duties of manufacturer:** (i) To use due care and research in development of all medical products, including medicines, medical and surgical instruments etc (ii) To perform studies of its product, when adverse reactions are reported in articles in scientific journals, and to report the results of these studies to physicians.

(3) **When is manufacturer liable:** (i) Almost always, unless doctor used the instrument incorrectly (ii) With respect to medicines if harm was caused due to adulteration, contamination, incorrect dosage or wrong labeling of drug.

(4) **When is doctor liable:** (i) if the instrument functioned satisfactorily during previous operations (ii) Incorrect use – by doctor (iii) Wear and tear - occurred in the instrument either due to normal wear and tear, or due to mishandling by doctor or paramedical personnel (iv) If the instrument was out of warranty, and required periodic inspection, testing and repairing and no request was sent to manufacturer for this (v) If instrument required periodic servicing and it was not sent for servicing (vi) If doctor knew [or should have known] the dangerous side effects, and could have used a substitute drug, yet decided to use the dangerous drug for no plausible reason (vii) If manufacturer has provided information in “package insert”, but doctor failed to convey it to patient. Once the manufacturer has made a warning in this manner, it is the

duty of doctor to convey this warning to the patient. Manufacturer has no duty to ensure that such warnings in the "package insert" reach the patient.

X. MEDICAL INDEMNITY INSURANCE

Medical Indemnity Insurance [MII] [syn, Medical Malpractice Insurance (MMI] is a contract between the doctor and an insurance company, under which the latter agrees, in exchange for the payment of premiums, to indemnify [reimburse or compensate] the insured doctor against claims resulting from his professional negligence.

XI. EUTHANASIA [MERCY KILLING]

Euthanasia [Gk eu, good; thanatos, death] or ***mercy killing*** means voluntary putting to death of a patient who is suffering from an incurable, terminal and extremely painful condition. Euthanasia was common in ancient Greece and Rome. This practice was challenged by **Hippocrates**, who spoke against it, writing, "I will not prescribe a deadly drug to please someone, nor give advice that may cause his death". Among most famous recent cases of euthanasia, **King George V** [1865 – 1936] was euthanized by his physician **Lord Dawson** by administering a lethal injection of cocaine and morphine. **John Bodkin Adams**, British general practitioner [**ch 40**], is thought to have euthanized several of his patients with morphine.

A. Classification

1. According to Physician's act

a. Active euthanasia

(1) Physician does an act of commission to bring an end to life, eg giving an injection of lethal drugs.

(2) **Case studies:** (i) **Howard Martin** (1935-), a British doctor euthanized 18 patients by giving them morphine injections. In 2005, he was tried for the murder of 3 patients, but was acquitted by the court, because he had no motive to kill other than euthanizing patients who were in great pain. Nevertheless the General Medical Council [GMC] struck his name for life.

b. Passive euthanasia

(1) Physician does an act of omission to bring an end to life, eg

(i) not using measures that would prolong life,

(ii) discontinuing or withholding extraordinary life sustaining measures [O_2, IV fluids etc] to prolong life,

(iii) failure to resuscitate a terminally ill or hopelessly incapacitated patient, or a severely defective newborn [**Table 5**].

(2) Broadly active euthanasia is killing while passive euthanasia is letting die.

(3) **Passive euthanasia is legal in India**.

2. According to the will of the patient

a. Voluntary euthanasia

(1) Patient wants to die and makes a request to this effect [ie patient consent is there].

(2) He must be competent to make this choice [i.e. must be mentally sound].

(3) **Voluntary euthanasia is legal in:** (i) Belgium (ii) Luxembourg (iii) Netherlands (iv) Switzerland (v) U.S. - (a) Oregon (b) Washington.

(4) **Classification** - Divided into two further types according to involvement of patient himself in the dying process.

b. Involuntary euthanasia

(1) Patient does not want to die, yet he is compulsorily killed.

(2) **Examples**:

Soldier has received vital injuries on abdomen "his intestines are hanging out " He is in great pain " requests a doctor to save his life " doctor knows he can not be saved " wants to end his suffering " shoots him.

c. Non-voluntary euthanasia

(1) Ending life of a person who is in coma and cannot make a decision on his behalf [eg persons in irreversible coma, severely defective infants].

(2) **Case studies:** (i) Karen Ann Quinlan’s case [**ch 8**] (ii) **Terri Schiavo**’s case [**ch 8**].

Table 5: Differences between Active and passive euthanasia

S.No	Feature	Active Euthanasia	Passive Euthanasia
1.	Definition	Doing a positive act to kill, eg administering a lethal drug	Discontinuing a life preservation procedure
2.	Nature of act	Act of commission	Act of omission
3.	Procedure	Administration of lethal doses of barbiturates, KCl, muscle relaxants	No longer resuscitating the patient by means of artificial live saving machines, O_2 etc
4.	Characteristic	Uses measures that cause death. Without these measures, patient would not have died	Patient is continuing on life sustaining machinery, without which he would have already been dead. These measures are withdrawn.
5.	Role of nature	Nature would have allowed the person to live. But doctor interfered to cause death	Nature would not have allowed the person to live. doctor was interfering with nature to keep him alive. Doctor simply removes his interference, and allows the nature to take its own course
6.	Acceptance in general public	Less	More
7.	Countries following procedure	Belgium, Netherlands	India, Some US states

B. Dysthanasia

Dysthanasia [Gk dys, bad; thanatos, death] is the converse of euthanasia, and is generally considered undesirable. This term is generally used when a person is kept alive artificially in a condition where, otherwise, he could not have survived, especially done for some sort of ulterior motive.

C. ML Aspects

Indian law – does not allow euthanasia [**S.89**, **S.92, IPC**]. Doctor may be charged **u/s 304, IPC**.

XII. CONSENT IN MEDICAL PRACTICE

Consent means voluntary agreement, compliance, or permission.

Salient features:

(1) **Indian Contract Act, 1872:** (i) S.13 - Two or more persons are said to consent when they agree upon the same thing in the same sense. Patient must understand, what it is given for and what are the risks involved (ii) S.14 - Consent is said to be free when it is not caused by coercion, undue influence, fraud, misrepresentation or mistake.

(2) **Components of a legally valid consent:** (i) Patient >12 y [for general physical examination]; >18 y [for surgery; please see S.88, IPC below]. In

case of child, consent from guardian who should be >18 y (ii) Patient understands that he can either agree or refuse to submit to examination, diagnosis and treatment (iii) Has been provided full information on his disease and treatment options [informed consent]. (iv) Consent should be clear, direct, free, informed, intelligent, personal and voluntary (a) Clear " in unambiguous words (b) Direct " directly conveyed to the doctor by patient, with no intermediaries (c) free " without undue force (d) informed " please see below (e) intelligent " patient should have clearly understood the implications of treatment. No ambiguity. No communication barriers (f) personal " Doctor should not communicate to anyone, what the patient has consented for (g) Voluntary " of his own accord.

(3) **Consent of an arrested person at police request** – not necessary **u/s 53, CrPC** [**ch 11**]**.**

(4) **Consent of an arrested person at his own request:** (i) Before **2008**, it could be done any time during his custody **u/s 54, CrPC,** if he made a request to the magistrate**.** (ii) This section has been amended since 2008; the amended ***S.54*** mandates compulsory medical inspection in all cases of arrest by the police. (iii) A copy of the medical report is to be provided to the arrested person or his nominee. (iv) If the arrested person is a female, her examination can be done only by or under the supervision of a female doctor [**S.54(1), CrPC**]. Please compare with corresponding **S.53(2), CrPC** [**ch 11**].

(5) **Consent of victim in criminal cases** – In victims of assault, rape and in cases involving pregnancy, abortion and delivery, examination of victim is necessary to gather evidence of crime. However if victim does not consent to examination it cannot be done.

(6) **Conflict of consent** – When two persons legally competent to give consent differ. Case must be referred to court, through IO.

(7) **Consent of victim of statutory rape – ch 25**.

(8) **Consent of an alcoholic – ch 40**.

(9) **Consent of spouse:** (i) Legally required - (a) for receiving artificial insemination (b) For donation of ovum or sperm.

Memory Aid 5: Consent of spouse

Consent of spouse legally required: Giving and taking of gametes, because gametes may be construed as belonging to spouse also

(ii) Legally not required but desirable to avoid marital conflict later - (a) MTP (b) Sterilization (c) Any operation that can affect sexual life [eg breast

reduction, penectomy, penis transplantation]

(10) **S.87, IPC** - It is not meant for doctors, because the act done by perpetrator is not done for the benefit of the opposite party. (i) It is applicable to people who undertake risky activities. (ii) To participate in a risky activity, the consenting person must be >18 y. (iii) The act by the perpetrator must neither be intended, nor known to be likely to cause death or grievous hurt.

(11) **S.88, IPC** - It is meant for doctors, because the act done by perpetrator is done in good faith for the benefit of the opposite party. (i) It is applicable to doctors (ii) The consenting person must be >12 y [**S.90, IPC**]. (iii) definition of good faith given in S.52, IPC – please see below.

(12) **S.89, IPC** - Act done in good faith for benefit of child <12 y or a person of unsound mind, by consent of guardian – is not punishable. Following provisos apply (i) this exception does not extend to the intentional causing of death [euthanasia], or to the attempting to cause death; (ii) this exception does not extend to the doing of anything which the doer knows to be likely to cause death, for any purpose other than the preventing of death or grievous hurt, or the curing of any grievous disease or infirmity; (iii) this exception does not extend to the voluntary causing of grievous hurt, or to the attempting to cause grievous hurt, unless it be for the purpose of preventing death or grievous hurt, or the curing of any grievous disease or infirmity; (iv) this exception does not extend to the abetment of any offence.

(13) **What makes consent valueless** – Consent given (i) under fear of injury (ii) under a misconception of fact (iii) Consent of mentally unsound person [**ch 28**] (iv) consent under intoxication (v) Consent of child <12 y [**S.90, IPC**; please also see **ch 25**].

Memory Aid 6: What makes a consent valueless

Forensic **M**edicine **M**aster **I**s **C**oming - Fear, Misconception, Mentally unsound, Intoxication, Child

(14) **S.52, IPC** - Definition of "**Good faith**" — Nothing is said to be done or believed in "good faith" which is done or believed without due care and attention.

(15) in loco parentis [in place of parent] – In emergency if parents are not available, person in charge of child [eg a school teacher, a hostel warden] can give consent.

(16) If inmate of jail, hostel etc refuses treatment, and he is likely to spread disease - he can be forcibly administered treatment. Inmate of a hostel may be

asked to leave hostel.

(17) In state mandated national programmes – eg pulse polio, vaccinations, no consent is needed.

(18) Consent for illegal acts [eg criminal abortion] – is invalid.

(19) Consent is not a defense – in cases of professional negligence.

(20) **Discharge against medical advice** – If a patient wants to leave against medical advice [LAMA], the dangers should be explained clearly in writing; then his wish should be recorded in the case sheet and his signatures obtained. Patient can then be discharged. In the discharge certificate, the doctor should specifically write the dangers of leaving hospital against medical advice.

A. Kinds of Consent

Consent may be classified (a) **According to information provided to patient:** (1) **Uninformed consent** [information completely or partly held back from patient]. Illegal unless it falls within the concept of "therapeutic privilege" [please see above]. (2) **Informed consent** [please see below] (b) **According to the way patient communicates it to the doctor**. It may be (1) **Implied** – when patient's actions imply consent [eg, telephoning, SMSing or emailing a doctor and asking for advice; (2) **Express** – (i) Def - When patient specifically consents to examination either (a) verbally [oral or verbal consent] or (b) in writing [written consent]. (ii) When required – in all procedures beyond routine physical examination [biopsies, Blood transfusions, LP, operations, Pleural and peritoneal tap etc] (iii) When taken – Before the act; not after it (c) **Miscellaneous kinds of consent** - (1) Blanket consent – is one where the doctor takes consent from the patient for any and every possible diagnostic or treatment procedure that might have to be undertaken any time in the future. (i) Since doctor himself is not sure of the kind of treatment that might be undertaken, patient cannot be explained of its risks etc. (ii) Many nursing homes and private hospitals, take blanket consent in the following manner "I hereby authorise staff of this hospital to perform any surgical or anaesthetic procedure on me." (iii) Blanket consent is invalid in the eyes of law. (2) Proxy consent [syn, substitute consent, surrogate consent] – Consent given by a relative, next of kin or friend in case the patient himself cannot give consent [child <12 y, mentally unsound person, intoxication, unconsciousness etc].

B. Informed Consent

Informed consent means that consent of patient for any procedure or treatment can only be taken after fully informing him about the nature of his condition.

Salient features:

Consent should always be informed.

1. Components of an informed consent

(1) Informed consent has five essential components, (i) **disclosure**, i.e. transmission of the information which is material to a decision; (ii) **comprehension**, or substantial understanding of this information; (iii) **absence of any outside control** over the decision; (iv) **competence**; and (v) **actual consent**.

(2) **Disclosure** [component 1] - Patient must be told (i) nature - of his disease (ii) Options available – All treatments, costs, risks, benefits, success and failure rates, after-effects and prognosis of each (iii) In case of surgery – (a) Whether it would be done under local or general anesthesia (b) Possible harmful effects of anesthesia (c) Details of all possible operative procedures, their relative benefits and risks (d) costs of each (iv) Risks - of not receiving any treatment [informed refusal].

(3) **Comprehension** [component 2] - Language - Explanation must be done in patient's own language. If need be, an interpreter may be employed.

(4) **Absence of any outside control** over the decision [component 3] – doctor, nurse, paramedical staff or any relative must not influence the decision. If patient voluntarily asks for advice, it may be given.

(5) **Competence** [component 4]: (i) patient should be above age of consent [>12 y for GPE; >18 y for surgery; ch 3] (ii) should be mentally sound (iii) should not be under influence of alcohol or drugs.

(6) **Actual consent** [component 5]: Written and witnessed - To make it legally acceptable, (a) everything explained must be reduced to writing and (b) signatures of patient obtained (c) in presence of two independent witnesses to prevent any allegation that the consent was forged or obtained under compulsion or pressure.

2. Advantages of taking informed consent [or reasons for obtaining consent]

(1) Patient cannot sue doctor (i) **u/s 350, IPC** (ii) **u/s 351, IPC** [**ch 11**] (iii) for

obtaining consent by misrepresentation of facts [**S.90, IPC**].

(2) Legally to examine, treat and operate upon a patient without consent, even if done in good faith is punishable under above sections.

3. Principles which form the basis of informed consent

(1) Principle of full disclosure or patient's right to truth.

(2) Principle of patient autonomy.

Principle of full disclosure

Principle of full disclosure states that a doctor should reveal all information to the patient, regarding his disease, treatment options etc.

Salient features:

(1) Withholding information from a competent patient is a violation of the doctor's role as a fiduciary [please see above] and is not justified.

(2) **Exceptions to principle of full disclosure** (i) Doctor need not disclose risks of which he is himself unaware (ii) If the risk is so obvious that a person with normal intelligence can easily understand. (iii) Therapeutic privilege [please see above]

4. Exceptions to informed consent

(1) Emergency [**S.92, IPC**]. Exactly same provisos apply for mentally unsound, intoxicated, persons <12 y and persons otherwise incapable of giving consent. Ex. Patient consents to laparotomy " during operation doctor discovers an unanticipated condition which endangers the life of patient " patient is under anesthesia " consent for the new operation cannot be taken " doctor undertakes procedure " protected u/s.92, IPC. This exception however does not extend to causing death. **Ex -** Doctor finds a lesion which would cause excruciating pain to patient, and he is sure, the patient cannot live for more than 6 months with the lesion. He decides to commit euthanasia to save unnecessary suffering. It is illegal.

(2) Therapeutic privilege.

(3) Waiving of rights by patient – Patient expressly waives his right to informed consent to doctor.

(4) **Case studies** – Please see Jackovach v. Yocum below.

C. Informed Refusal

The concept of **informed refusal** states that the doctor must inform the patient about the risks of refusing a particular operation, test, medication, or other medical intervention.

Salient features:

Concept of informed refusal is inherent in informed consent [please see above]. It assumes importance only when the patient does not consent to treatment despite being informed of risks of refusal. To be legally valid, such informed refusal must be reduced to writing with signatures of patient, doctor and witness.

D. Additional Rules of Consent

(1) **Necessity** – Consent is required for all medical examinations. Ordinarily express consent is not required, because implied consent is inherent in patient's behavior.

(2) **Verbal consent** – Must be obtained in the presence of a disinterested third party, eg a nurse.

XIII. HUMAN EXPERIMENTATION

Human experimentation is conduction of medical experiments on humans.

Declaration of Helsinki (1964)

Adopted by the 18th WMA General Assembly held at Helsinki, Finland in **June 1964** and amended in 1975, 1983, 1989, 1996, 2000, 2002 and 2004. Prescribes ethical Principles for **Human Experimentation**.

Memory Aid 7: Declaration of Helsinki

Helsinki"**H**uman experimentation.

XIV. THE WORKMEN'S COMPENSATION ACT, 1923

The ***Workmen's Compensation Act, 1923*** [WCA, 1923] is one of the earliest labor welfare and social security legislation enacted in India.

Salient features:

Aims and objectives: (i) Provides compensation to workmen and their dependents - in case of accidents arising out of and in the course of employment causing death or disablement of workmen. Compensation is payable by employer (ii) lays down various amounts payable in case of an accident, depending upon the type and extent of injury.

XV. CONSUMER PROTECTION ACT, 1986

Consumer Protection Act 1986, [Syn, CPA, COPRA] is a legal statute whose purpose is to provide for better protection of the interests of consumers.

Salient features:

Aims and objectives: to protect the interest of consumers - of different commodities [eg TV, car] and services [eg banking, airline travel].

A. Consumer Disputes Redressal Agencies

There are three agencies:

(1) District Forum
(2) State Commission
(3) National Commission

1. Manner of making complaints

(1) A complaint in relation to any goods sold or delivered or any service provided may be filed with a District Forum by (a) the consumer. If consumer dies, his legal hair or representative (b) any recognized consumer association (c) one or more consumers, where there are numerous consumers having the same interest, with the permission of the District Forum (d) Central or the State Government. Every complaint must be accompanied by a fees prescribed by the State Govt [**S.12**].

(2) **On receipt of a complaint** - The District Forum may allow the complaint to be proceeded with or rejected [after giving an opportunity to the complainant of being heard]. Admissibility of the complaint shall ordinarily be decided within 21 days from the date on which the complaint was received [**S.12(3)**].

(3) **Frivolous or vexatious complaints** – are dismissed after recording the reasons in writing. The complainant may be asked to pay a cost of up to

`10,000 to the opposite party [**S.26**].

2. Procedure

(1) **Speedy trials:**

(i) Every complaint is heard as early as possible and must preferably be decided within 3 months.

(ii) No adjournment are ordinarily granted by DF unless sufficient cause is shown and the reasons for grant of adjournment have been recorded in writing by the Forum [**S.13(3A)**].

(2) If complaint refers to any service, the Forum shall refer a copy of such complaint to the opposite party directing him to give his version of the case within a period of 30 days. An extension of 15 days may be granted [**S.13(2), (a)**]. Notices may be served even by FAX [**S.28(2)**].

(3) It is not necessary for parties to be represented by lawyers.

(4) Every proceeding before the DF shall be deemed to be a judicial proceeding within the meaning of **S.193** and **228** of IPC, and DF shall be deemed to be a civil court.

(5) **Appeals against orders of District Forum:**

(i) Must be made to the State Commission within 30 days from the date of order.

(ii) If there was sufficient cause for appeal later than 30 days, State Commission can entertain it.

(iii) Fees - appellant must deposit 50% of amount ordered by DF or `25,000 whichever is less [**S.15**].

(6) **Appeals against orders of State Commission:**

(i) Must be made to the National Commission within 30 days from the date of order.

(ii) If there was sufficient cause for appeal later than 30 days, National Commission can entertain it.

(iii) Fees - appellant must deposit 50% of amount ordered by SC or `35,000 whichever is less [**S.19**].

(7) **Appeals against orders of National Commission:**

(i) National Commission is empowered to review its orders to avoid rush to the Supreme Court

(ii) However even if review does not give relief to a party, the appeal must be made to the Supreme Court within 30 days from the date of order.

(iii) If there was sufficient cause for appeal later than 30 days, Supreme Court can entertain it

(iv) Fees - appellant must deposit 50% of amount ordered by SC or $ 50,000 whichever is less [**S.23**].

(8) **Ensuring payment of compensation** – To ensure payment of compensation, the Consumer Courts [DF, SC, NC] can attach the property of the opposite party and dispose it to recover amount due to the complainant. District Collector would help the courts to recover this amount.

(9) **Finality of orders** – If no appeal has been preferred against order of a DF, SC or NC, such order shall be final [**S.24**].

3. Limitation period

The District Forum, the State Commission or the National Commission normally will not admit a complaint unless it is filed within 2 years from the date on which negligence was committed. But under special circumstances complaints after 2 years may be entertained if the Forum or Commission is satisfied that there is sufficient reason for doing so, and it must record in writing, the reasons for entertaining such late complaints [**S.24A**].

4. Who is covered under COPRA

(1) All doctors and hospitals who have charged for their service.

(2) All doctors and hospitals [including Govt hospitals, health centers, dispensaries etc] who perform partial charitable services, i.e. who charge from few patients only [rich] and other [poor] are not charged. All [both rich and poor] can get benefit under COPRA.

5. Who is immune from COPRA

(1) Doctors and hospitals [including Govt hospitals] which are doing a completely charitable service not charging anyone [i.e. neither rich nor poor are charged].

(2) Hospitals charging a token amount for registration purpose only – It is not payment for the purposes of COPRA.

XVI. ORGAN DONATION AND TRANSPLANTATION – LEGAL ASPECTS

A. The Transplantation of Human Organs and Tissues Act, 1994 (No. 42 of 1994)

The ***Transplantation Of Human Organs and Tissues Act, 1994*** [***TOHOTA***] provides for (i) regulation of removal, storage and transplantation of human organs for therapeutic purposes and (ii) prevention of commercial dealings in human organs. The Transplantation of Human Organ rules were framed in **1995**. The Act was amended in ***2011*** [27th Sep].

XVII. THE PROTECTION OF HUMAN RIGHTS ACT, 1993

(1) The Act was passed in **1993** and amended in **2006**. It provides for the constitution of a National Human Rights Commission [NHRC (**S.3)**] at the Center, State Human Rights Commissions [SHRC **(S.21)**] in States and Human Rights Courts [**S.30**].

(2) **Purpose** - Better protection of human rights.

XVIII. MEDICOLEGAL ASPECTS OF HIV AND AIDS

A. Civil

1. Discrimination

HIV+ve person can not be isolated, quarantined or discriminated against.

2. Divorce

U/s S.13(1) of Hindu Marriage Act, 1955, a person can get divorce, if spouse has been suffering from **venereal disease in a communicable form** [**ch 23**].

3. Employment

According to India's National Policy on HIV/AIDS [please see above], a person can neither be denied employment because of his HIV+ve status, nor can he be discriminated during service, nor can he be terminated from job solely due to HIV+ve status.

B. Criminal

Marriage or intercourse:

(1) HIV +ve person A knowingly marries B [or performs intercourse] without

informing B of his status " B catches infection " A can be sued **u/s 269, IPC**. **S.269** may apply when the accused (a) did not know he was HIV+ve and (b) did not understand how HIV is transmitted

(2) If accused disclosed his HIV+ve status to the person at risk [or honestly believed the other person was aware of his status], there may be no case.

C. Rights of HIV+ve Patients

(1) An AIDS patient has the **right to life** and **personal liberty [Art 21, Constitution of India]**. It means that he has the right to treatment of his disease like any another person.

(2) They have the **right to privacy**. It means that they have the same right as other patients with regard to the disclosure of their disease status to other people and general public as a whole. In other words, one (doctor or hospital) **can't do things like putting labels on their beds indicating their HIV status**.

(3) They cannot be isolated or quarantined on the basis of their disease.

(4) They cannot be discriminated in any sphere of life, including their job, due to their HIV status.

D. Duty of Doctors

(1) **Confidentiality:**

(i) Normally a doctor must maintain professional secrecy, but in case of HIV+ve patients, he can reveal the HIV+ve status to the prospective bride.

(ii) In case of married couple, the status of married partner can be told to the spouse. The latter can then apply for divorce under provisions of the Hindu Marriage Act, 1955.

(2) **Careful screening of blood:**

(i) for HIV status - before blood donation, transfusion, surgical operation or organ transplantation.

(ii) Precautions - (a) screen living donors for HIV as close to the time of organ recovery and transplantation as possible, using sensitive tests for both chronic and acute infections, namely, serology and **nucleic acid testing** [NAT] (b) inform transplant candidates of the potential risks for disease transmission (c) advise donors that they have an obligation to avoid behaviors that would put them at risk for acquiring HIV before organ

donation.

(iii) Arguments in favor of routine screening (a) Charges of negligence – may be brought by the patient, if patient discovers after the operation that he is HIV +ve. He may allege that the infection was transmitted through these procedures. Preoperative or pretransfusion screening will protect the doctor from such charges

(iv) Arguments against routine screening (a) The accepted ethical standards of autonomy, confidentiality, and informed consent are broken.

E. Miscellaneous

Multiple tattooing with same needle may cause HIV transmission.

3. Identification

I. INTRODUCTION

Identification is the determination of the individuality of a person – living or dead.

A. Case Study

1. Bhowal Sanyasi case [1921]

A rich person **Kumar Ramendra Narayan Roy** [**Fig 3.1**] allegedly died of biliary colic in **1909** in Darjeeling. In **1921**, a Sadhu (Bhowal Sanyasi) arrived in Dacca, and **claimed that he was Kumar Ramendra**, and asked for his share in property. When denied, he filed a suit in a **Dacca court** in **1930**, for restoration of his legal rights. Most of his **identification data** was **exactly the same** as that of Ramendra including pink and white complexion, brown wavy hair, light moustache, twisted lip, sharp angle at the rim of ear, ear lobes pierced and not adherent to cheeks, broken left upper first molar, a fleshy point in the right lower eyelid, size 6 for shoes, irregular scar on left ankle, syphilitic ulcers, boil mark on the head and back, operation mark near the groin, tiger claw mark on the right arm and even a minute mole on the dorsum of the penis. He was **declared by the**

court to be **Kumar Ramendra Narayan Roy** and **entitled to the property**. However the Bhowal Sanyasi died shortly after the decision of the court in **July 1946**.

Fig. 3.1: Ramendra Narayan Roy, the prince of Bhawal Estate, whose true identity was in question in early 20th century.

II. CORPUS DELICTI

Corpus delicti [from Latin corpus, body; delictum, crime] is the legal principle requiring ***positive proof of crime*** before someone can be convicted of committing that crime. Also known as the ***body of crime, foundation of crime*** or ***essence of crime***.

Salient features:

(1) Corpus delicti includes anything which positively prove a crime.

(2) In case of homicide, it would include the **dead body**, **photograph** of **dead body**, **blood stained clothes showing weapon marks**, **blood stained weapon**, **bullet**, **severed body parts (eg head)** etc.

III. IDENTIFICATION DATA

Table 1 enumerates important identification data. Please also see **Fig 3.2**.

Table 1: Important Identification data

(A) Race (B) Religion (C) Sex (D) Age (E) Stature (F) Complexion (G) features (H) Hair (I) Deformities and other peculiarities such as moles, birthmarks (J) Scars (K) Tattoos (L) Occupation marks (M) Anthropometry (N) Dactylography (O) Footprints (P) Other prints, such as palatoprints, and lip prints (Q) Superimposition (R) Reconstruction of features from the skull (S) Identification from teeth and bite marks [forensic odontology] (T) Clothes and personal articles (U) Molecular methods (V) Miscellaneous methods -

1. **Biometric methods -** (a) Physical characteristics (i) Bone comparisons (ii) DNA profiling (iii) Frontal sinus pattern (iv) Iris scans (v) Nails (vi) Retina scans (vii) Skull suture pattern (viii) vascular grooves (ix) Stains (x) Stomach pictures (xi) Veins on the back of hands (xii) X-rays (b) Behavioral characteristics - (i) Gait (ii) Handwriting (iii) Mental power, memory and education (iv) Speech and voice (v) Tricks of manner and habit.
2. **Non Biometric methods -** (a) Facial composites. Some of these data can only be used in living [eg behavioral ch], some only in dead [eg superimposition], and some in both [eg dactylography]. Except dactylography, no single feature is completely reliable, so a set of 4-5 criteria [or more] may be taken for identification. More the number of criteria, better is the identification.

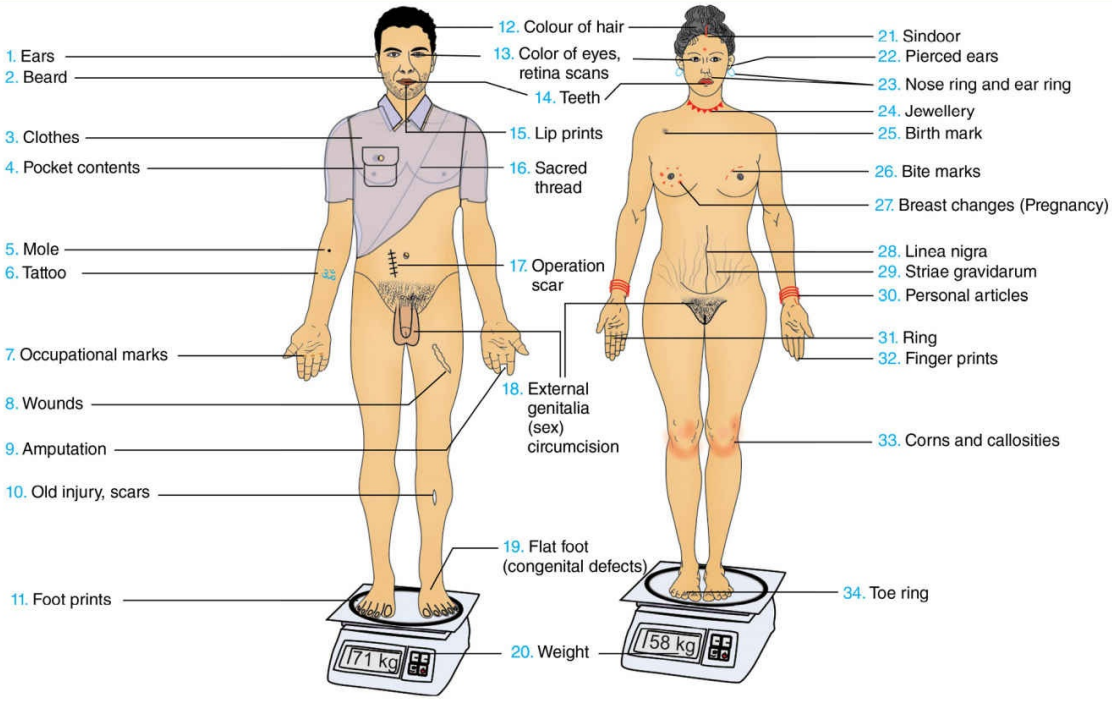


Fig. 3.2: Identification data at a glance.

A. Race

Three races are recognized: (i) **Caucasoids** (ii) **Mongoloids,** and (iii) **Negroids**. Race – as mentioned above - can be determined by:

1. Skin

(1) **Caucasoids** are white skinned**,**
(2) **Mongoloids** yellow skinned**,** and
(3) **Negroids** black skinned
(4) **Indians** - brown
(5) Skin color changes in **advanced decomposition** and **burns** -> determination of race may become difficult.

2. Eyes

(1) Caucasoids have **blue eyes**.
(2) **Black** in other races.
(3) People of mixed races (e.g. Indians) may have **brown** eyes.

3. Hair

(1) **Caucasoids** have light colored, reddish or golden hair. They are curly or wavy and cross section is **oval**.

(2) **Mongoloids** have dark colored hair. They are straight, and cross section is **round**.

(3) **Negroids** have dark colored hair. They are wooly and arranged in tight spirals, and cross section is **kidney shaped** (Fig 3.3).

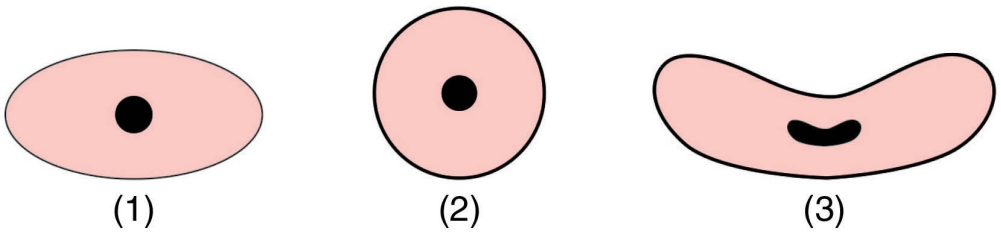


Fig. 3.3: Cut section of hair in (1) Caucasoids; (2) mongoloids and (3) negroids

4. Skeleton

Racial differences in the skeleton are enumerated in Table 2. (1) **Prognathism** – A quick test is to occlude the lower jaw in its proper dental occlusion, and with a pencil attempt to touch the base of the nasal aperture and chin at the same time. This can be done in Caucasoid skulls, but not in Negroid and Mongoloid skulls.

Memory Aid 1: Cusp of Carabelli

Cusp of Carabelli is present in Caucasoids.

Table 2: Racial differences in skeleton in various races

S.No	Trait	Caucasoids	Mongoloids	Negroids
1.	Skull	Rounded	Square	Narrow and elongated
2.	Prognathism [protrusion of the alveolar regions of the mandible and maxilla]	Least common, least pronounced	Midway between caucasoids and negroids	Common, pronounced
3.	Forehead	Raised	Inclined	Small and compressed
4.	Face	Small proportionately; Malar bones prominent	Large and flattened; teeth set obliquely	Malar bones and jaw projecting
5.	Orbits (Fig 3.4)	Triangular	Rounded	Square
6.	Nasal aperture (Fig 3.4)	Narrow and elongated	Rounded	Expanded, Broad
7.	Palate (Fig 3.4)	Triangular	Rounded or horse shoe shaped	Rectangular
8.	Shape of upper incisors (Fig 3.4)	Straight (no concavity)	Shovel shaped	Straight (no concavity)
9.	Cusp of Carabelli	Present. Please also see below under the heading "race from teeth"	Absent	Absent
10.	Upper and lower limbs	Normal in proportion to body	smaller	Longer

a. Skull

Cephalic index

Cephalic index (CI) [syn ***index of breadth***] is the ratio of the maximum width (B) of the head divided by its maximum length (L), multiplied by 100. CI as a criterion of race was introduced in **1842** by **Anders Retzius** (1796 – 1860). It is now known that besides race, local environmental conditions also influence the CI. Its validity in race determination has been challenged.

$$C.I. = \frac{\text{Maximum breadth of skull (B)}}{\text{Maximum length of skull (L)}} \# 100$$

Memory Aid 2: Sex related indices

All sex related indices **[Table 3]** are **higher** in **females** except **C**hilotic line index and **C**orporobasal index, which are more in males. Both start with **C**.

Salient features:

(1) **Measurements:** (i) Maximum length of skull = Summit of glabella to furthest occipital point.

(ii) Maximum breadth of skull = greatest breadth, at right angles to sagittal plane. (iii) measured by sliding calipers. If not available, use osteometric board.

(2) As seen from the top, a longer skull is dolichocephalic and a more rounded skull is brachycephalic. Brachycephaly can occur in any other race also if the coronal suture fuses prematurely [flat head syndrome].

(3) Races can be determined to some extent by CI [**Table 3**]. An average Indian has a **mesaticephalic skull**.

Table 3: Various important indices and their application in forensic medicine

Race	Sex	Human or animal origin
[A] **Related to skull** (1) Cephalic index [B] **Related to limbs** (2) Brachial Index (3) Crural Index (4) Intermembral Index (5) Humerofemoral Index	[A] **Related to skull** (1) Nasal index [B] **Related to hip bone** (2) Ischiopubic index (3) Sciatic notch index (4) chilotic line index (5) ilium index [C] **Related to sacrum** (6) Sacral index (7) Kimura's base-wing index (8) corporobasal index	[A] **Related to hair** (1) Medullary index

Memory Aid 3: How to remember indices

There are about **15 important indices** **[Table 3]**, all of which can be remembered using the following **3 simple rules**:

1. Think up 2 most natural dimensions of the related bone
2. Keep smaller dimension in numerator; larger in denominator

3. multiply with **100**. Only one index – **medullary index** – is not so multiplied. Strictly speaking it is a co-efficient. Conventionally co-efficient is measured between **0** and **1**. When it is expressed as a percentage, it is called an **index**.

Example 1 – Remembering **cephalic index**

1. Cephalic is skull. 2 most natural dimensions are **length** and **breadth**.
2. Breadth is smaller so we get B/L.
3. Multiply above by 100.

Example 2 – Remembering **sternal index**

1. Sternal refers to sternum. 2 most natural dimensions are **height of manubrium** [M] and **height of body** [B]
2. M is smaller so we get M/B.
3. Multiply above by 100.

With some practice, all indices can be similarly derived. In fact the **definition of index** is that it is "*the percentage expression of the ratio of a smaller dimension over a larger one*".

(4) **Use of indices** – In forensic medicine, indices are used for solving 3 major questions – race, sex and species origin [human or animal] **[Table 3]**.

b. Long bones

(1) **Brachial index** [Latin Brachium, arm]

[syn, radio-humeral index] = $\frac{\text{Length of radius}}{\text{Length of humerus}} \# 100$

(2) **Crural index** [Latin crus, leg] [syn, tibio-femoral index] = $\frac{\text{Length of tibia}}{\text{Length of femur}} \# 100$

(3) **Intermembral index** = $\frac{\text{Length of (humerus + radius)}}{\text{Length of (femur + tibia)}} \# 100$

(4) **Humerofemoral index** = $\frac{\text{Length of humerus}}{\text{Length of femur}} \# 100$

B. Religion

(1) Hindu males are not circumcised and wear a sacred thread.

(2) Muslim males are circumcised, have corns and callosities on lateral aspects of knees and feet and have a black spot on their forehead [From making sujood, (placing of the forehead on the ground in prayer)].

(3) Hindu females wear sindoor on head, nose rings and ear rings.

Memory Aid 4: Racial differences in cephalic index. Pl. correlate with Table 4**.**

Du MB Horse

Table 4: Racial differences in cephalic index

S.No	Type of skull	Cephalic index	Race
1.	Dolichocephalic (long headed)	70-75	Caucasoids (some, eg. Pure Aryans), Negroids [Aborigines and Negroes]
2.	Mesaticephalic (medium headed)	75-80	Caucasoids (some, eg Europeans), Mongoloids (some, e.g. Chinese)
3.	Brachycephalic (short headed)	80-85	Mongoloids (some, e.g. Mongolian)
4.	Hyperbrachycephalic	85-90	Kyushu of Japan

Table 5: Long bone Indices in different races

S. No	Indices	Indians	Caucasoids (Europeans)	Negroids
1.	Brachial Index	76.49	74.5	78.5
2.	Crural Index	86.49	83.3	86.2
3.	Intermembral Index	67.27	70.4	70.3
4.	Humerofemoral Index	71.11	69.0	72.4

C. Sex

Normally sex determination is easy from external examination (**Table 6**), but it may be difficult in cases if (i) Advanced decomposition (ii) Concealed sex (iii) Hermaphroditism (iv) Skeleton.

Table 6: Sex differences in male and female sex. (Presented roughly from above downwards)

S.No	Trait	Male	Female
1.	Build	Larger, stouter. Greater muscular development	Smaller, lesser muscular development
2.	**Adam's apple (larynx)**	**Prominent. Length 4.8 cm**	**Not prominent. Length 3.8 cm**
3.	Shoulders	Broader than hips	Narrower than hips
4.	Thorax	Larger. Less rounded	Shorter. More rounded
5.	Limbs	Longer	Shorter
6.	Arms	Flatter in cross section	More rounded in cross section
7.	Wrists and ankles	Stouter. Less delicate	More delicate
8.	Head hair	Shorter, thicker and coarser	Longer, thinner and finer
9.	**Body hair**	**Present on face (moustache, beard) and chest**	**Absent**
10.	**Pubic hair**	**Thick. Area containing pubic hair rhomboidal in shape (extends upwards to the umbilicus)**	**Thin. Area containing pubic hair triangular in shape (ends abruptly in a horizontal line at mons veneris)**
11.	Trunk	Abdominal segment smaller	Abdominal segment longer
12.	**Breasts**	**Not developed**	**Well-developed (for lactation)**
13.	Waist	Ill defined	Well-defined
14.	Thighs	Cylindrical	Conical (shorter femur, -deposition of fat)
15.	**Gluteal region**	**Flatter**	**Full and rounded**
16.	**External Sex organs**	**Penis, scrotum**	**Vulva, vagina**
17.	**Internal sex organs**	**Prostate, seminal vesicles etc**	**Uterus**
18.	**Gonads**	**Testes**	**Ovary**

1. Intersex

An ***intersex*** is an intermingling in one individual of characters of both sexes in varying degrees, including physical form, reproductive organs, hormonal profile and sexual behavior.

Salient features:

(1) Incidence is **0.018%** without the inclusion of Klinefelter's syndrome and Turner's syndrome, and **1.7%** with their inclusion.

(2) Intersex may be classified in a number of ways. The simplest classification is according to presence of gonads and accompanying external sexual characteristics [**Table 7**].

Table 7: Classification of intersex according to presence of gonads and accompanying external sexual characteristics

S. No	Presence of gonads and external sexual characteristics	Type of intersex
1.	Gonads absent	Gonadal agenesis
2.	Gonads [of only one sex] present, but fail to develop at puberty	Gonadal dysgenesis
3.	Gonads of both sexes present	True hermaphrodite
4.	Gonad of one sex present, but external sexual characteristics are of opposite sex	Pseudohermaphrodite

a. Gonadal agenesis

In ***gonadal agenesis***, the testes and ovaries are entirely absent.

Salient features:

Sex chromatin is absent (46, XY).

b. Gonadal dysgenesis

In ***gonadal dysgenesis***, the testes and ovaries are present, but they fail to develop normally.

Salient features:

(1) Gonads are mainly composed of fibrous tissue (**streak gonads**).

(2) Gonadal dysgenesis are divided into two types: (i) **Klinefelter's syndrome** (ii) **Turner's syndrome**.

i. Klinefelter's syndrome

Klinefelter's syndrome, "***47, XXY***", or "***XXY syndrome***" is a condition in which the affected individual has two X chromosomes and one Y chromosome.

Salient features:

A. **Anatomical structure** - male; nuclear sexing – female; sex chromosomes [gonosomes]– 47, XXY [XXY Males] Most common; 48,XXXY, 48,XXYY, and

49,XXXXY have also been described. B. **Incidence** - Occurs in 1:600 males. Most common sex chromosome disorder. C. Named after American endocrinologist **Harry Klinefelter** (1912 -1990), who described the condition in **1942** D. **Signs and symptoms:** (1) **Diagnosis** - Usually diagnosed when there is (i) a delay in onset of puberty, (ii) behavioral disorders and (iii) mental retardation [in contrast to Turner's syndrome, where intellectual function is intact]. (2) **Body dimension related** - (i) Height -ed [due to extra copy of **SHOX** gene. Please see below] (ii) narrow shoulders, (iii) broad hips. (3) **Sex related features** - (i) **Testicles** are small [microorchidism] (ii) .testosterone (iii) **Leydig cell function**"normal to moderately reduced (iv) Azoospermia [absent spermatogenesis] (v) **Sterility** [most common genetic cause for male sterility] (vi) Gynecomastia. (4) **Hair** - (i) Axillary and pubic hair absent. (ii) Hair on chest and chin are reduced. (5) **Serum and urinary gonadotrophins**- - Pathophysiology

s
Primary hypogonadism"serum testosterone."FSH and LH-. (6) **Miscellaneous** - (i) Diabetes mellitus (ii) Osteoporosis (iii) thromboembolic disease (iv) varicose veins E. **Histology** – Testicular atrophy with hyalinization of seminiferous tubules.

ii. Turner's syndrome

Turner's syndrome (also known as ***Ullrich-Turner syndrome***) is a condition in which the affected individual has only one X chromosome and no Y chromosome [monosomy X].

Salient features:

[A] **Anatomical structure** - female; nuclear sexing – male; sex chromosomes – 45, XO. [B] **Incidence** - Occurs in **1:2500** girls [C] **Signs and symptoms** – Turner syndrome manifests itself differently in each female; no two individuals display the same symptoms. (1) **Diagnosis** - Can be recognized at birth by (i) **low birth weight**, (ii) **edema** of dorsum of hands and feet and (iii) loose skin folds in the nape of the neck (**Webbed neck** or pterygium colli). Other signs and symptoms are (2) **Stature related** - (i) Short stature [most common feature; becomes evident by about age 5]. (ii) **slow growth** (iii) **Micrognathia** [small lower jaw] (iv) **shortened 4th metacarpal** (3) **Sex related** - (i) **Premature ovarian failure** [very common]. (ii) **Rudimentary ovaries gonadal streak** [underdeveloped gonadal structures] (iii) **Amenorrhoea** [primary] (iv) **Sterility**

(v) Lack of development of primary and secondary sexual characteristics (vi) Serum and Urinary **gonadotrophins**- [pathophysiology same as in KS] (4) **Face related** - (i) Ptosis (ii) Visual impairments [glaucoma, etc] (iii) Ear infections and hearing loss (iv) high-arch palate [narrow maxilla] (v) Low-set ears (vi) Low hairline (5) **Chest related** - (i) Broad chest [**shield chest**] (ii) **Poor breast development** (iii) **widely spaced nipples** (6) **Upper limb related** - (i) Cubitus valgus [-ed carrying angle (>15°) of the elbow] (ii) palmar crease (iii) Small soft upturned fingernails (7) **Body related** - (i) High waist-to-hip ratio [hips are not much bigger than the waist] (ii) -ed wt, obesity (iii) pigmented moles (8) **Anomalies of heart** – (i) Bicuspid aortic valve, (ii) Coarctation of aorta (iii) septal defects (9) **Other anomalies** - (i) Horseshoe kidney [other renal defects also] (ii) spina bifida. (10) **Endocrine** - (i) Hypothyroidism (ii) Diabetes mellitus (iii) Cushing's syndrome (11) **Intelligence** - Most patients have normal intelligence [E] **Histology** – Ovaries do not contain primordial follicles.

c. True hermaphroditism

True hermaphroditism refers to a condition in which the affected individual has both ovarian and testicular tissue.

Salient features:

(1) Very rare condition

(2) **Gonads** - There may be an ovary on one side and a testis on the other, but more commonly the gonads are fused in the form of **ovotestis**.

(3) **Position of gonads** – Abdominal, inguinal or labio-scrotal

(4) **Function** - Neither gonad is completely functional.

(5) **External genitalia** – ambiguous.

(6) **Internal sex organs** – Uterus may be present in some cases

(7) **Karyotypes** - are mosaics [46XX/46XY, or 46XX/ 47XXY].

d. Pseudohermaphroditism

Pseudohermaphroditism, is the condition in which the affected individual has gonads of one sex, but external genitalia of opposite sex**.**

Salient features:

(1) **Two types** are seen – Male pseudohermaphroditism and female hermaphroditism

(2) **Male pseudohermaphroditism:** (i) **Genetics and external sex** - Nuclear

sex is XY, but external sexual characteristics and internal sex organs (some or all) are female. (ii) **Causes** - (a) Androgen insensitivity syndrome (AIS) [earlier name "testicular feminization syndrome"]- The gonads are testes [due to the influence of the Y-chromosome]. No ovaries or uterus. The absence of uterus in fact is regarded as a condition for the diagnosis of testicular feminization. (b) 5-**a**-reductase deficiency [5-ARD] –

(3) **Female pseudohermaphroditism:** (i) **Genetics and external sex** - Nuclear sex is XX, but external sexual characteristics and internal sex organs are male (ii) **Causes**- (a) Congenial Adrenal hyperplasia [CAH].

In **2014**, the Supreme Court directed the Center and State govts that **they must recognize transgenders as 3rd gender**. All documents including voter/Aadhar card and passports etc will have a third category called "transgenders". They will be considered as OBCs and shall be provided reservation in educational institutions and jobs. Govt must provide separate toilets in public places for them and separate facilities for them for HIV/Sero-surveillance measures. The court clarified that their verdict pertained only to **eunuchs** and **not to other sections of society like gay, lesbian and bisexuals.**

2. Concealed sex

Concealed sex is hiding one's sex by wearing dress of the opposite sex.

Salient features:

(1) Criminals often do it to avoid being caught by the police. This can be detected by physical examination.

(2) Concealed sex is different from transvestism (eonism), in which also the person wears clothes of the opposite sex. But in the latter, there is a psychological compulsion instead of a motivation to avoid detection (see also chapter 25).

(3) **Colonel Sir Victor Barker's** case is one of the most amazing cases of concealed sex. Barker (1895 – 1960) was born a **female** (Valerie Lilias Barker), **who acted like a man between 1923 and 1929**. She married twice to males and once to a female (posing as a male)! During the period she posed as male, she played for a cricket club, moved about in the streets in Khaki shorts with a shirt open at the neck and dropped in at pubs for drinks. She was sentenced to nine months imprisonment for making a false statement in the marriage register.

3. Determination of sex

Determination of sex [in living and dead in medico legal practice] can be done by

(1) Examination of external genitalia

(2) Sex chromatin

(3) DNA based methods

(4) Skeleton

a. Examination of external genitalia

Useful in cases of concealed sex. In other cases, may be unreliable and confusing. Please see intersex above.

b. Sex chromatin

A normal person has 46 chromosomes (males 46, XY; females 46, XX). ***Sex chromatin*** is the condensed chromatin of the inactivated X chromosome in females (***Barr body***). While **sex chromosomes** refer to both X and Y chromosomes, **sex chromatin** refers only to inactivated X chromosome. [A] **Sex chromatin** is: (i) characteristic of females [found in about **30% to 40% of female cells**] (ii) **shape** - planoconvex in shape (iii) **size** - about 1**µ**, in diameter (iv) **location** - Lies against the inner surface of the nuclear membrane (v) **Stage seen in** - present only during interphase; disappears during mitosis (vi) **Tissues seen in** – Visible in all nucleated cells (not seen in RBC), but easily seen in (a) buccal smears (b) saliva, (c) skin (d) hair follicles (e) dental pulp and (f) bone marrow. **Karyotyping** (study of chromosomal constitution of cell nuclei for sex determination) of fetus is done from (g) amniocytes (h) lymphocytes and (i) fibroblasts (vii) **Stain** – (a) **X chromosome** – demonstrated by **Feulgen reaction** using acriflavine Schiff reagent [X chromosome seen as **bright yellow spot**].

Memory Aid 5: Feulgen reaction

XY FAST -

X chromosome looks bright **Y**ellow in **F**luorescent **F**eulgen reaction using **A**criflavine **S**chiff reagen**T**

Memory Aid 6: Feulgen reaction

Feulgen reaction is +ve in **F**emales

(b) **Y chromosome** – demonstrated by **quinacrine dihydrochloride** [Y chromosome seen as **bright fluorescent body** – commonly referred to as an **f-body**] (viii) **discovered in** - 1948 by Barr and Bertram [hence the name Barr body] (ix) **Putrefied bodies** – Sex chromatin may be demonstrated in **putrefied**

bodies, but not if putrefaction has reached up to cellular level. Uterus and prostate resist putrefaction for a long time; in advanced putrefaction, sex can be determined by identifying uterus and prostate.

Memory Aid 7: Quinacrine staining

MCQ - **M**ale **C**hromosome colors with **Q**uinacrine

[B] **Davidson's bodies:** (i) **Location** - Barr bodies seen on the nucleus of **neutrophils**. Appears like a drumstick (**Fig 3.5**). (ii) seen in **3%** female neutrophils. (iii) **Diameter** - of the round head at the end of the drumstick is 1.5**µ**. (iv) **Stain** – Demonstrated by Jenner-Giemsa method or Feulgen method (v) Named after **William M. Davidson** of King's college hospital medical school, London, who first described these bodies in **1954**.

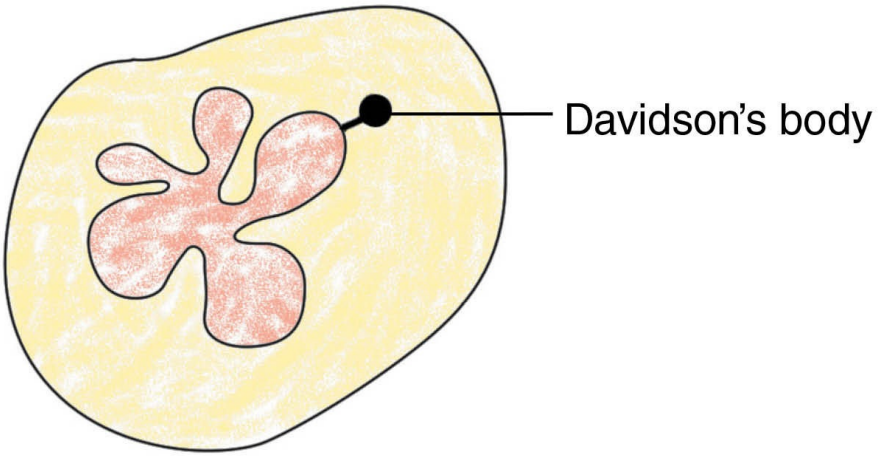


Fig. 3.5: Davidson's body

Table 8: Various cellular level tests for male and female features, and their positivity

S.No	Test	Male%	Female%
1.	Barr body [sex chromatin]	0-4%	30-40%
2.	Davidson's body	0%	3%
3.	Feulgen reaction	0-2%	50-70%
4.	Quinacrine Dihydrochloride	45-80%	0%

c. Sexing of the skeleton

(1) Human bones display **sexual dimorphism** [different shapes in sexes]. Recognizable sex differences do not appear until after puberty except in pelvis, in which sexual dimorphism is present since fetal life.

(2) There are two methods for sex determination from skeleton (i) morphologic [roughness, shapes etc, more subjective] and (ii) metric [measurements; more objective]. Sex differences in skull are typically morphologic, while sex differences in femur are typically metric. An admixture of both is mostly used.

Normal "carrying angle" of the elbow varies between 5 to 15 degrees. This angle permits the forearms to clear the hips in swinging movements during walking, and is important when carrying objects. This is more in girls.

(3) **Sex differences in minor bones:** (i) Metacarpals - Metacarpal 2 is best for sex determination; metacarpal 3 is worst. (ii) Patella - (a) Vol – Male > 15 cc; Female < 11 cc. Measured by putting patella in a container of water.

(4) **Accuracy of sexing adult skeletal remains** – According to American anthropologist **Wilton Krogman** (1903–1987) the percentage accuracy of determining sex when one or more bones are given is as follows [**Table 20**].

(5) **Medullary index of bones** – Medullary index of long bones [tibia, humerus, radius, ulna etc] is

$$\frac{\text{Diameter of medulla}}{\text{Diameter of shaft}}$$

100. Attempts have been made to determine age and sex from this, but have been largely unsuccessful.

(6) **From bone marrow** – DNA based methods of determination of sex [please see above].

Table 9: General sex differences in human skeleton

S.No	Criteria	Male	Female
1.	General size	(i) Larger (ii) more massive	(i) Smaller (ii) less massive
2.	Depressions, muscular markings, processes and ridges on bones	More prominent	Less prominent
3.	Length	On an average bones about 8% longer	Shorter
4.	Shaft	Rougher	Smoother
5.	Medullary cavity	Shallower	Relatively wider
6.	Metacarpals	Longer and broader	Shorter and narrower
7.	Bone Mineral Density [BMD]	Higher, especially in the forearm bones,	Lower
8.	Weight of dry skeleton [please see ch 6 – Fragmentary remains also]	4.5 kg	3.0 kg

Table 10: Sex differences in human skull

S.No	Criteria	Male	Female
1.	General appearance	(i) Larger (ii) heavier (iii) more massive	(i) Smaller (ii) lighter (iii) less massive
2.	Architecture	Rugged	smooth
3.	Walls	Thicker	Thinner
4.	Surface	Rough	Smooth
5.	Muscular ridges	More marked esp at base and in occipital and temporal areas	Less marked
6.	Capacity	1500-1550 ml	1350-1400 ml
7.	Forehead	Steeper, less rounded	Vertical, fuller and rounder
8.	Glabella	More prominent	Small or absent
9.	Frontonasal junction	Distinct angulation	There is smooth curve
10.	Orbits	(i) Size - relatively smaller (ii) shape - square (iii) position - set lower on face (iv) margins -rounded	(i) Size - relatively larger (ii) shape - Rounded (iii) position - set higher on face (iv) margins - sharp
11.	Supraorbital ridges	Prominent	Less prominent or absent
12.	Zygomatic arch [cheek bones]	(i) Heavier and more prominent (ii) Laterally arched	(i) Lighter and lessprominent (ii) Lateral arching less; more compressed
13.	Nasal aperture	Higher and narrower	Lower and broader
14.	Bony ridge along the upper border of external auditory meatus	Prominent	Less prominent or absent
15.	Frontal eminences	Small	Large
16.	Parietal eminences	Small	Large
17.	Frontal sinuses	More developed	Less developed
18.	Maxillary sinuses	Significantly larger	Smaller
19.	Occipital protuberance	Very prominent	Less prominent
20.	Mastoid process	(i) size - Large (ii) tip – Blunt (iii) M type	(i) size – small (ii) tip – pointed (iii) F type
21.	Digastric groove	Deeper	Shallower
22.	Occipital condyles	Larger	Smaller
23.	Palate	(i) Larger and broader (ii) U-shaped	(i) Smaller and narrower (ii) Parabolic in shape
24.	Foramina	Larger	Narrower
25.	Foramen magnum	Larger and oblong	Smaller and rounder
26.	Teeth	Larger	Smaller

Memory Aid 8: Medicolegal and anatomical angles of mandible. Pl co-relate with (Fig 3.6)

1. Remember **MA**ndible

Medicolegal angle is upper angle

Anatomical angle is lower angle

2. Remember **MAAS**

Medicolegal angle deals with

Age

Anatomical angle deals with **S**ex. Further, it behaves the same way as subpubic angle, i.e. it is - in females

3. Remember 75 " 100 " 125 " 140

75º " Subpubic angle in males

100º " Subpubic angle in females

<125º " Anatomical angle in males

>125º " Anatomical angle in females

>140º " medicolegal angle in infants and old age.
[90º in adults]

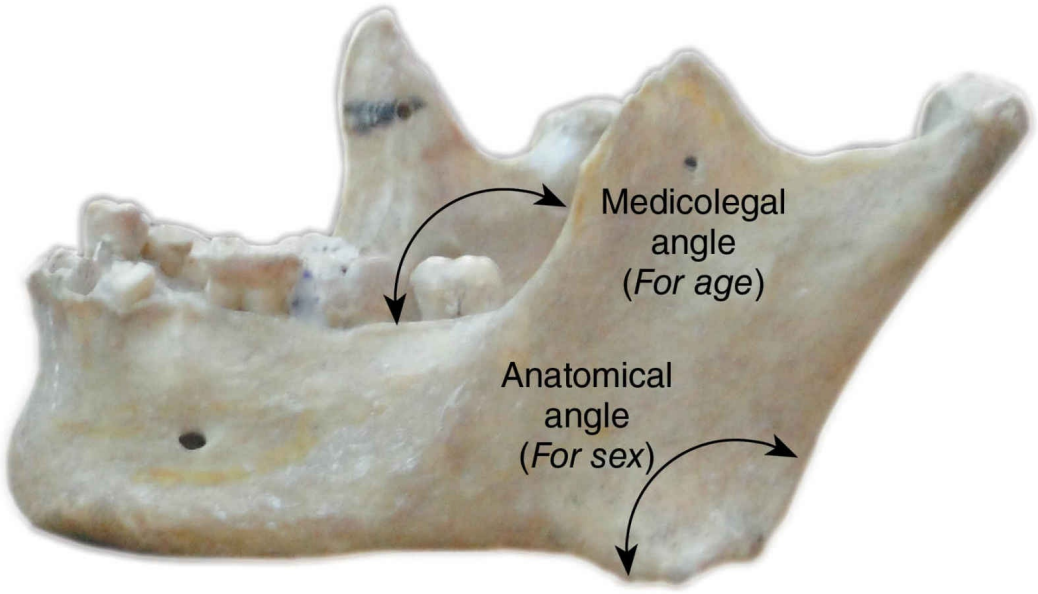


Fig. 3.6: Mandibular and anatomical angles of mandible (pl co-relate with **Memory Aid 8**)

Table 11: Sex differences in human mandible. Pl co-relate with (**Fig 3.7**) [major differences in male and female mandible]; and also with **Table 28** [differences in mandible with age]

S.No	Criteria	Male	Female
1.	General size	Larger and thicker	Smaller and thinner
2.	Chin [Symphysis menti]	Square	Rounded
3.	Body height at symphysis	greater	Smaller
4.	Breadth of ascending ramus	Greater	Lesser
5.	posterior border of ascending ramus	Shows indentation at the level of occlusal surface of the molars	No such indentation
6.	Angle between **lower border of body** and **posterior border of ascending ramus** [angulus mandibulae, anatomical angle, gonial angle]. For medicolegal angle pl. see **Table 28**	Less obtuse [<125°].	More obtuse [>125°].
7.	Angles	everted	Less everted or inverted
8.	Condyles	Larger	Smaller
9.	Mental tubercle	Large and prominent	Insignificant

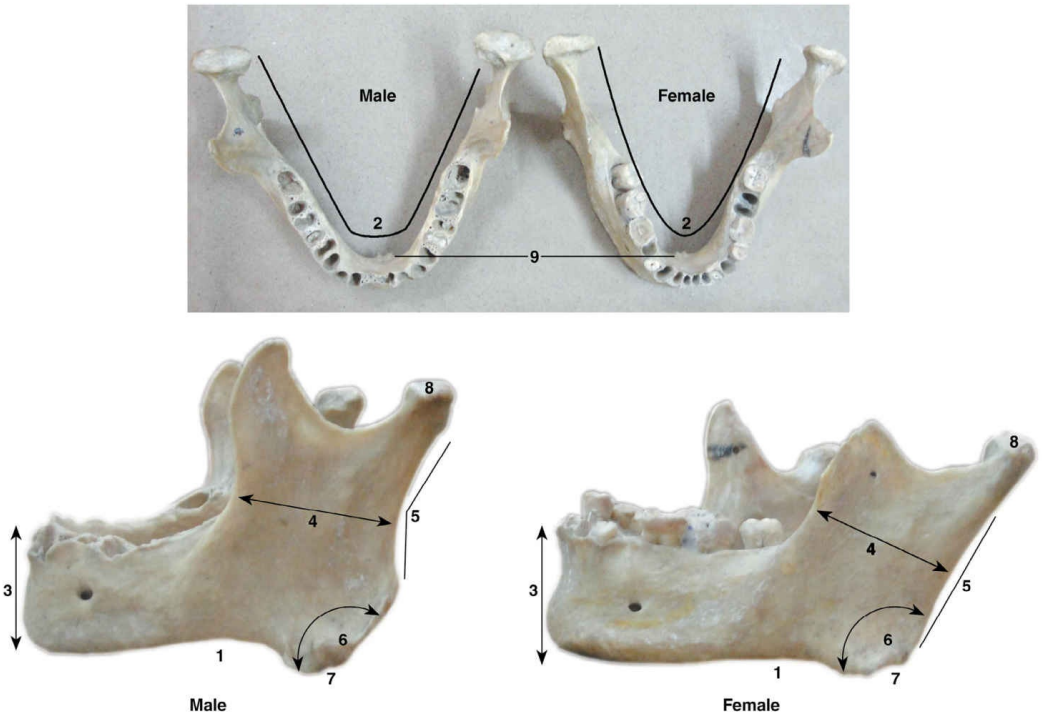


Fig. 3.7: Major differences in male and female mandible. Pl co-relate numbers with **Table 11** [Sex differences in human mandible]. Also co-relate with **Table 28** [differences in mandible with age] and **Fig. 3.24** [appearance of mandible at different ages].

Table 12: Sex differences in human pelvis

S.No	Criteria	Male	Female
1.	Bony framework	(i) Massive (ii) More erect (iii) Muscular markings more marked (iv) Rougher (v) Stands higher	(i) less massive (ii) less erect (iii) muscular markings less marked (iv) smoother (v) stands lower
2.	Bones	Tough, thick and heavy	More delicate, thin and light
3.	General shape	Deep funnel shaped	Flat bowl shaped
4.	Ilium	(i) Curve of iliac crest reaches higher level (ii) More prominent (iii) Less vertical (iv) Iliac fosse deep (v) Distance between iliac crests is more [26.7 cm] (vi) Curves of iliac crest less marked	(i) curve reaches lower level (ii) less prominent (iii) more vertical (iv) Iliac fosse shallow (v) Distance between iliac crests is less [25.4 cm](vi) Curves of iliac crest well marked
5.	Preauricular sulcus [attachment of **anterior sacroiliac ligament**]	Not prominent [sometimes not even visible], narrow, shallow	More frequent, broad, deep
6.	Acetabulum	Large [dia 52 mm], directed laterally	Small [dia 46 mm], directed anterolaterally
7.	Obturator foramen	Large, oval with base upwards	Small, triangular with apex forwards
8.	Greater Sciatic Notch	Smaller, narrower, deeper	Larger, wider, shallower
9.	Average width of greater sciatic notch	46.5 mm	51.5 mm

10.	Average depth of greater sciatic notch	32 mm	31 mm
11.	Sciatic notch index $\left[\frac{\text{Width of sciatic notch}}{\text{depth of sciatic notch}} \times 100\right]$	**145** [in adults] **4-5** [in fetuses]	**166** [in adults] **5-6** [in fetuses]
12.	Iliopectineal line [syn, linea terminales; divides pelvis into greater (false) and lesser (true) pelvis]	Rough, well marked. Less vertical	Smooth, rounded. More vertical
13.	Ischial spines	Closer together, inverted	More separated, everted
14.	Ischial tuberosity	Inverted	Everted; more widely separated
15.	Chilotic line [line extending backwards from the iliopectineal eminence to the nearest point on the anterior margin of the auricular surface and then to the iliac crest. The auricular point divides it into anterior (pelvic) and posterior (sacral) segments]	Sacral part prominent	Pelvic part prominent
16.	Chilotic line index: $\frac{\text{Sacral part of chilotic line}}{\text{Pelvic part of chilotic line}} \times 100$	>100	<100
17.	Body of pubis	(i) Narrow, (ii) triangular. (iii) No parturition pits [syn, parturition scars]	(i) Broad, (ii) square or quadrangular, (iii) depressions, pits or scars on posterior surface, if borne children **[parturition pits]**
18.	Superior ramus of pubis	It is like continuation of body of pubis	Short and thick. Has a constricted or narrowed appearance
19.	Ischiopubic rami	Distinctly roughened, heavier. More everted [attachment of crus penis]	Smoother, lighter, less everted [attachment of clitoris]
20.	Ischiopubic index [Washburn's index]: $\frac{\text{Pubic length}}{\text{Ischial length}} \times 100$	73-94 [average - 75]	91-115 [average - 100]
21.	Symphysis pubis	Higher, narrower	Lower, wider
22.	Margins of pubic arch [formed by both ischiopubic rami]	Everted	Not everted
23.	Distance between two pubic tubercles [syn, pubic spines]	Lesser	Greater
24.	Pelvic brim (inlet)	Heart shaped	Circular or elliptical. All diameters longer. More spacious.
25.	Pelvic cavity	Conical and funnel shaped	Broad and round
26.	Pelvic outlet	Smaller	Larger
27.	Subpubic angle	V-shaped. Angle 70°-75°	U-shaped. Angle 90°-100°
28.	Sacroiliac joint surface	Large. Angulation is less sharp	Small. Angulation is sharp; almost L-shaped
29.	Coccyx	Less moveable	More moveable

Table 13: Sex differences in the human sacrum

S.No	Criteria	Male	Female
1.	Size and shape	Longer, narrower	Shorter, wider
2.	curvature	more evenly distributed.	Curvature not seen in the upper half; lower half curves suddenly
3.	Sacral Promontory	well marked.	less marked.
4.	Body of first sacral vertebra	larger	smaller
5.	Sacroiliac articulation	Large. Extends to 2½ - 3 vertebrae	Small. Extends to 2-2½ vertebrae
6.	Sacral index: $\frac{\text{Anterior Superior Breadth of Sacrum}}{\text{Anterior length}} \times 100$	105	115
7.	Kimura's base-wing index [Alar index]: $\frac{\text{Width of wing}}{\text{Breadth of 1st Sacral Vertebra}} \times 100$	65	80
8.	Corporobasal index: $\frac{\text{Breadth of 1st Sacral Vertebra}}{\text{Anterior Superior Breadth of Sacrum}} \times 100$	45	40

Memory Aid 9: Indices relating to sacrum

1. **S**tate **B**ank **C**orporation - **S**acral, **B**ase-Wing, **C**orporobasal.

2. For males, figures are in a neat sequence - 105"65"45. Female indices are greater for first 2 and lesser for 3rd. The differences are 10, 15 and 5 - another easily remembered sequence.

	M	Diff	F
Sacral	**105**	10	**115**
Base-Wing	**65**	15	**80**
Corporobasal	**45**	-5	**40**

3. For **base-wing index**, remember **WBC**, i.e. **W** comes at top and **B** at bottom. Another way to remember is reading the index backwards i.e. **B**ase - **W**ing"W/B

4. Pl also note that ***numerator of 1st index*** and ***denominator of 2nd index*** interchange their position in the 3rd index.

Table 14: Sex differences in the human sternum

S.No	Criteria	Male	Female
1.	**Ashley's rule**	Total length>149 mm [also sometimes known as Ashley's "149 rule". Enunciated by Ashley in 1956]	Total length<149 mm
2.	**Hyrtl's law**	Body is longer and more than twice the length of manubrium	Body is shorter and less than twice the length of manubrium
3.	Breadth	more	Less
4.	Length of the mesosternum [Middle piece or body of the sternum]	>95 mm	<80mm
5.	Level of Upper margin	in level with lower part of body of T2	in level with lower part of body of T3
6.	Sternal [manubrio-corpus] index $\frac{\text{Length of manubrium}}{\text{Length of body}} \times 100$	46.2	54.3
7.	width of the 1st sternebra at its waist	27 mm [approx]	24 mm [approx]
8.	width of the 3rd sternebra at its waist	32 mm [approx]	29 mm [approx]
9.	Sternal foramen	Twice as common as in females	Less common

Table 15: Sex differences in human ribs

S.No	Criteria	Male	Female	S.No	Criteria	Male	Female
1.	Composition	Thicker	Thinner	2.	Curvature	Lesser	Greater
3.	Obliquity [cranio–caudal inclination]	Less oblique. cranio–caudal inclination of ribs is .	More oblique. cranio–caudal inclination of ribs is -	4.	Length of ribs in relation of body height	Less [ribs are relatively shorter]	More [ribs are relatively longer – better accommodate abdominal distension during pregnancy]

Table 16: Sex differences in human thoracic bones

S.No	Criteria	Male	Female
1.	Shape of thorax	Longer and narrower	Shorter and wider
2.	Volume	More	Less [about 10% less than that of males having the same height]
3.	Clavicle	Longer, broader, heavier, less curved	Smaller, narrower, lighter, more curved

Table 17: Sex differences in human vertebral column

S.No	Criteria	Male	Female
1.	Breadth of atlas	7.4-9.9 cm	6.5-7.6 cm
2.	Length of vertebral column	73 cm	60 cm

Table 18: Sex differences in human femur

S.No	Criteria	Male	Female
1.	Head	(i) Larger (ii) Forms 2/3 of a sphere (iii) vertical dia >46 mm	(i) Smaller (ii) Forms <2/3 of a sphere (iii) vertical dia <42 mm
2.	Angle of Neck with shaft [*caput-collum-diaphyseal angle or CCD angle]*	125°[Obtuse angle]	<125° [less obtuse angle]
3.	Popliteal length	>145 mm	<106 mm
4.	Bicondylar width	>78 mm	<72 mm
5.	Trochanteric oblique length	>450 mm	<390 mm
6.	Angulation of shaft with condyles	80° **Memory Aid: 10** Both angles - (i) Neck with shaft and (ii) Shaft with condyles are - in males [just opposite to subpublic angle]	75°

Table 19: Sex differences in human scapula

S.No	Criteria	Male	Female
1.	Height of scapula	>157 mm	<144 mm
2.	Height of Glenoid cavity	>39.2 mm	<32.6 mm

Table 20: Accuracy in sexing adult skeletal remains

S.No	Bone	Accuracy
1.	Entire skeleton	100%
2.	Pelvis + Skull	98%
3.	Pelvis alone	95%
4.	Skull alone	90%
5.	Long bones alone	80%

D. Age

1. Age estimation in the living

a. Methods of determination

Age of an individual can be determined from (i) teeth (ii) bones (iii) secondary sexual characters and (iv) general development (v) Miscellaneous features.

i. Teeth

For deduction of other parameters from teeth please see below under the heading "Teeth and bitemarks [forensic odontology]". Criteria from which age can be determined are (a) Eruption and calcification of teeth and root resorption (b) Aspartic acid racemization (c) Tooth Development stages [TDS].

(a) Eruption and Calcification

1st Deciduous teeth

(1) Deciduous [Latin – de, down; cadere, to fall] teeth are also known as **baby teeth**, **milk teeth** or **temporary teeth**.

(2) Total number of deciduous teeth are 20.

(3) The dental formula of children is

$$\frac{2102}{2102}$$

.

(4) **Ages of eruption of deciduous teeth** - can be remembered by two different mnemonics:

Memory Aid 11: Eruption of temporary teeth

Mnemonic (1) South Indian Idlis, mothers can make.

1. South"Indicates lower teeth appear first (especially incisors)
2. **I**ndian"(i) **I**ncisors [1st], central (lower)"6 months (ii) **I**ncisors [1st], central (upper)"8 months
3. **I**dlis"(i) **I**ncisors [2nd], lateral (upper)"10 months [Lateral incisors are an exception; upper appear first] (ii) **I**ncisors [2nd], lateral (lower)"12 months
4. **M**others"**M**olars [1st]"14 months
5. **C**an"**C**anine"18 months
6. **M**ake"**M**olars [2nd]"24-30 months.

The ages start at 6 months. After this go on adding 2 months to go to next step. Nothing appears at 16, 20 and 22 months. Counting of teeth is always done from medial to lateral, i.e. 1st incisor is medial, 2nd lateral and so on.

Mnemonic (2) It is a visual mnemonic. Draw 10 temporary teeth of one half of mandible in the form of a graph [**Fig 3.8**] other half need not be drawn, as it is identical. (i) Incisors would appear in a clockwise fashion starting from lower central incisor [6 m]. (ii) Teeth erupt from medial to lateral with a gap of 6 [except 1st molar which erupts at 14 m]. Best depicted in lower jaw with the sequence 6, 12, 18, 24 (iii) Double headed arrows indicate that upper and lower teeth erupt at same age.

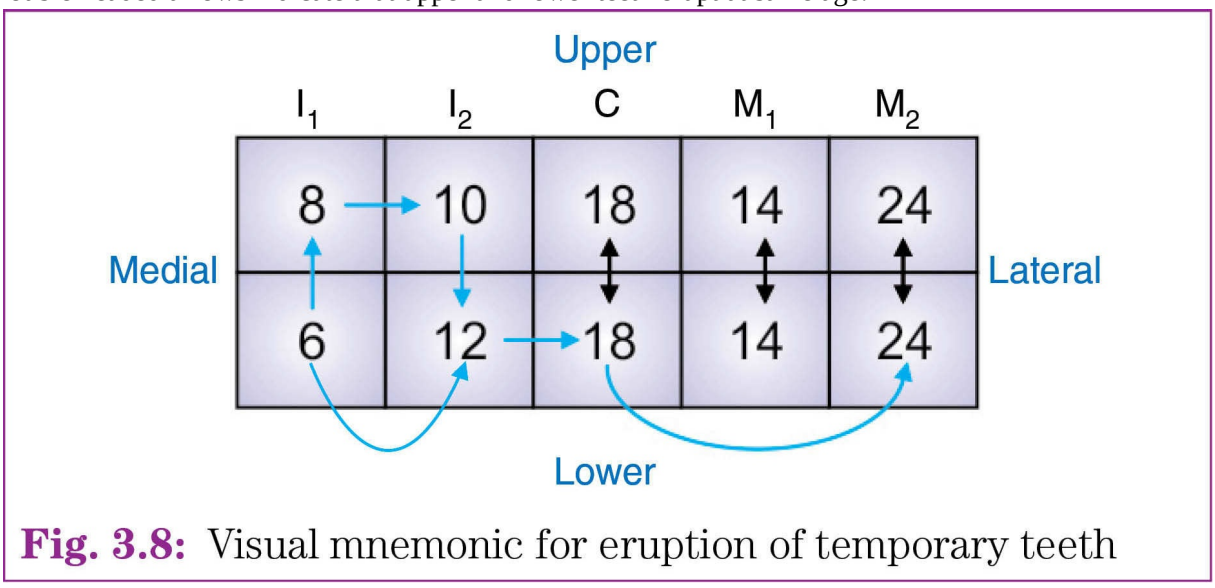


Fig. 3.8: Visual mnemonic for eruption of temporary teeth

Ages of eruption and calcification of deciduous teeth are summarized in **Table 21**.

2nd Permanent teeth

(1) Total number of permanent teeth are 32.

(2) The dental formula of adults is

$$\frac{2123}{2123}$$

(3) **Ages of eruption of permanent teeth** -**Table 22**.

Memory Aid 12: Eruption of permanent teeth

Mnemonic

1. Order of eruption of permanent teeth is in the following sequence

Mama Is In Pain; Papa Can Make Medicine

Mama	Molar, 1st	**Papa**	Premolar, 2nd
Is	Incisor, 1st	**Can**	Canine
In	Incisor, 2nd	**Make**	Molar, 2nd
Pain	Premolar, 1st	**Medicine**	Molar, 3rd

Mnemonic

2. **Mother is in bed; baby comes Monday morning. Same as above, except that B**"Bicuspid

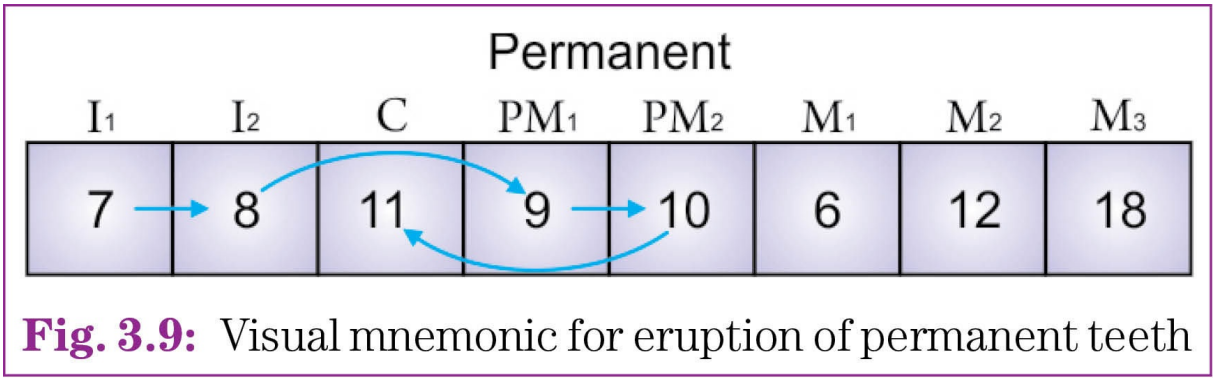


Fig. 3.9: Visual mnemonic for eruption of permanent teeth

Mnemonic

3. It is a visual mnemonic. (i) Draw all 8 permanent teeth of one quadrant in the form of a graph [**Fig 3.9**]. (ii) First fill up ages of eruption of molars which are a table of 6 [6,12,18 y]. (iii) Now fill up ages for rest of teeth. Teeth appear from medial to lateral with a gap of 1 y except canine which appears at 11 y.

Table 21: Ages of calcification and eruption of deciduous teeth.

S.No	Tooth	Calcification begins	Eruption	Calcification of root complete	Resorption of root begins
1.	Central Incisor – Lower	14wk IU	6m	1½ - 2 y	4th y
2.	Central Incisor – Upper	14 wk IU	8m	1½ - 2 y	5th y
3.	Lateral Incisor – Upper	16 wk IU	10m	1½ - 2 y	5th y
4.	Lateral Incisor – Lower	16 wk IU	12m	1½ - 2 y	5th y
5.	1st molar	15.5 wk IU	14m	2 - 2½ y	6th y
6.	Canine	17 wk IU	18m	2½ - 3 y	8th y
7.	2nd molar	18-19 wk IU	24-30m	3 y	7th y

Table 22: Ages of calcification and eruption of permanent teeth

S.No	Tooth	Calcification begins	Eruption	Calcification of root complete
1.	1st Molar	At birth	6y	9y
2.	1st [Central] Incisor	4m	7y	10y
3.	2nd [lateral] Incisor	1y	8y	11y
4.	1st Premolar	1½y	9y	12y
5.	2nd Premolar	2y	10y	13y
6.	Canine	4m	11y	13y
7.	2nd Molar	2½-3y	12y	15y
8.	3rd Molar	8-10y	18-25y	18-25y

Memory Aid 13: Calcification of permanent teeth

Remember following steps:

1. For **beginning of calcification** of permanent teeth – remember 0,6,12,18,24,30,36.

2. Sequence of teeth remain as earlier [as in Memory aid 12].

3. Now allot numbers to teeth, keeping first molar at birth [0m]

4. M_1=0m

I_1=6m

I_2=12m

P_1=18m

P_2=24m

C=6m [because **C**anine and **C**entral incisors begin with same letter]

M_2=30-36m; M_3=8-10 y [**Mnemonic - 8th** tooth calcifies at **8 y**].

5. For greater accuracy, change **6 m** to **4 m** for both **I_1** and **C**.

6. For **completion of calcification**, just add **3 y** to the ages of eruption for all teeth.

(4) **Dentition [permanent] is delayed in:** (i) Local conditions – Same as in deciduous teeth. Additionally (a) Ankylosis of deciduous teeth (b) Apical periodontitis of deciduous teeth (c) Impacted primary tooth (d) Injuries to primary teeth (e) Lack of resorption of deciduous tooth (f) Premature loss of primary tooth (ii) Systemic conditions – Same as in deciduous teeth

(5) **Mixed dentition** – Between 6-11 y, the child has both temporary and permanent teeth. This is the period of mixed dentition. In mixed dentition, the total number of teeth are always 24 [**Table 23**]. During this period following formula gives age:

Number of permanent teeth = (Age in years – 5) x 4.

(6) **Successional and superadded teeth** [**Fig 3.10**] – ***Successional teeth*** are those permanent teeth which replace some other teeth. Thus their eruption does not change the total number of teeth of the individual. In contrast ***superadded teeth*** are those which are added to the existing set of teeth; they do not replace any teeth. Thus their eruption always -es the number of teeth. Incisors, canines and premolars are successional teeth while all molars are superadded teeth.

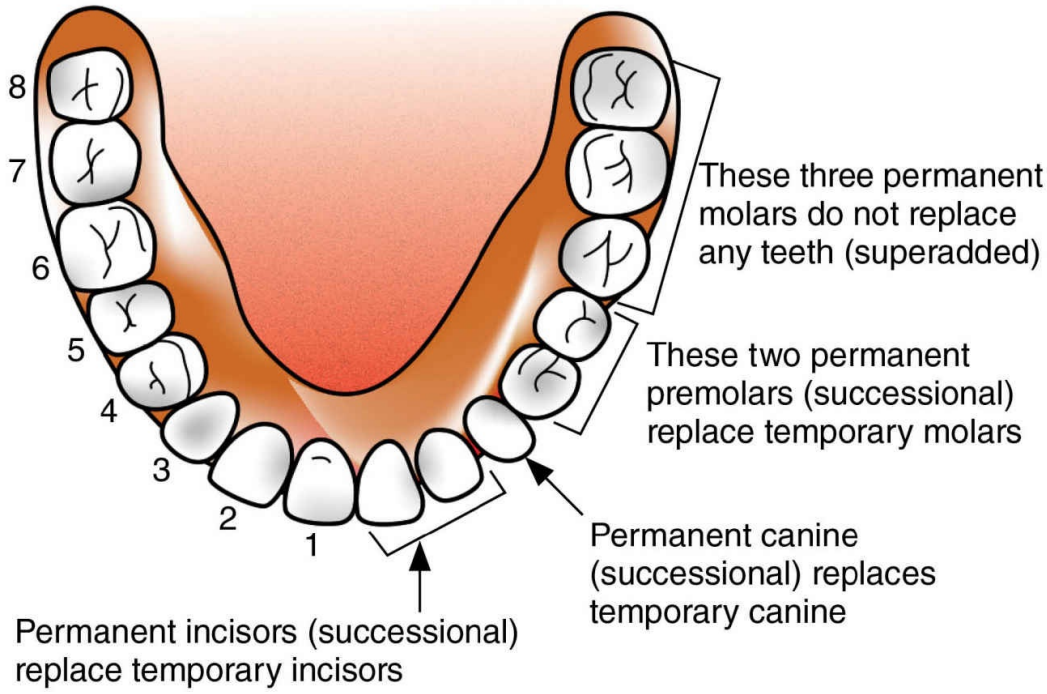


Fig. 3.10: Successional and superadded teeth

Table 23: Number of teeth in relation to age. Ages of mixed dentition have been shown in light blue.

Age	Temporary teeth	Permanent teeth	Total teeth
6m	2	0	2
8m	4	0	4
10m	6	0	6
12m	8	0	8
14m	12	0	12
18m	16	0	16
2½-5y	20	0	20
6y	20	4	24
7y	16	8	24
8y	12	12	24
9y	8	16	24
10y	4	20	24
11y	0	24	24
12y	0	28	28
18-24y	0	32	32

ii. Bones [determination of age in the living]

[A] **General:** (1) **Formation of bones** - In a fetus bones form in two ways: (i) Intramembranous ossification (ii) endochondral ossification [formation in hyaline cartilage]. (2) **Primary and secondary centers:** (i) Initial site of ossification is referred to as the **primary center** [eg shaft of humerus, femur, tibia]. This center lays down most of the bone. Typically, a long bone [eg femur] is already ossified throughout its shaft [**diaphysis**] at birth. (ii) The one which appears afterwards is the **secondary center** (eg head of humerus, head of femur

etc). Generally they appear towards the bone ends [eg each side of the long bone]. Both primary and secondary centers appear in X-rays initially as ***white spots***. As bone lays around them, the whitish area -s in area, till only a black plate remains between primary and secondary centers. This is the **growth plate** [please see below] (iii) Secondary center may be single [eg head of humerus, femur] or multiple [eg lower end of humerus, which shows 4 secondary centers – medical epicondyle, trochlea, capitulum and lateral epicondyle]. (iv) Metacarpals, metatarsals, phalanges, clavicles and ribs possess an epiphysis at one end only. (v) Not all bones have secondary centers; some bones ossify from a single primary center only [eg all carpals and most tarsals]. (vi) The earliest bone to begin ossification is **clavicle** (**45 days**, or **1½ months**). (vii) Most primary centers appear during fetal development [with some exceptions eg. most carpals and tarsals]. (viii) **Number of ossification centers** – (a) End of 11th IU wk – **806 centers**. Many of these unite with one another (b) At birth - Approx **450 ossification centers** remain. (c) These gradually enlarge throughout childhood and united together to form **206 bones of the adult**. (3) **Ossification of a typical bone:** Growth plate [growth cartilage, metaphysis] – is a layer of hyaline cartilage that persists between the diaphysis and epiphysis. In an X-ray it appears as a black line, which may be confused by an inexperienced person with a fracture line. In the dead, it appears as a circular groove, and cuts with the consistency of tough wax. In very old skeletal materials, growth plates putrefy completely causing separation of epiphysis and diaphysis. This may be confused with fractures by inexperienced persons. To demonstrate them adequately in a living person, X-rays of all joints should be taken both in AP as well as lateral views.

(a) Long bones

1st—Ages of appearance

[I] **Upper end of humerus** [**Fig 3.12**]"Remember the arithmetic series 1, 3, 5 : Head of humerus appears at 1; Greater tuberosity at 3; Lesser tuberosity at 5. Remember that in English alphabet g comes before l, and in arithmetic 3 comes earlier than 5: G"L 3"5 All three centers unite by 6 y, i.e. 1 y after the last centre comes. [II] **Lower end of humerus** [**Fig 3.12**]"There are four major centers to remember: Medical epicondyle, trochlea, capitulum and lateral epicondyle.

Memory Aid 14: Ossification centers of lower end of humerus

1. **C**apitulum - appears at one [remember "**c**" for **c**hild. Children are younger so it comes very early, i.e. at 1]

2. **T**rochlea - appears at **T**en (remember "**T**")

3. **L**ater**al** **Epicondyle** - appears at 11 (Remember "**l**later**al**". Both beginning and ending letters are "l". If we keep them together, they look like 11)

4. **M**edial **E**picondyle - appears at **5-7** [**F**ive-**S**even].

[**M**y **E**xcellent **F**orensic **S**cience]

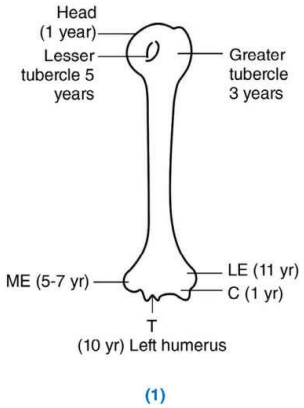
5. **LTC** (**L**ateral epicondyle, **T**rochlea and **C**apitulum) join together to form a **conjoint epiphysis**, which then joins with the shaft.

6. **Medial epicondyle** joins the **shaft of humerus** separately **[Table 24]**.

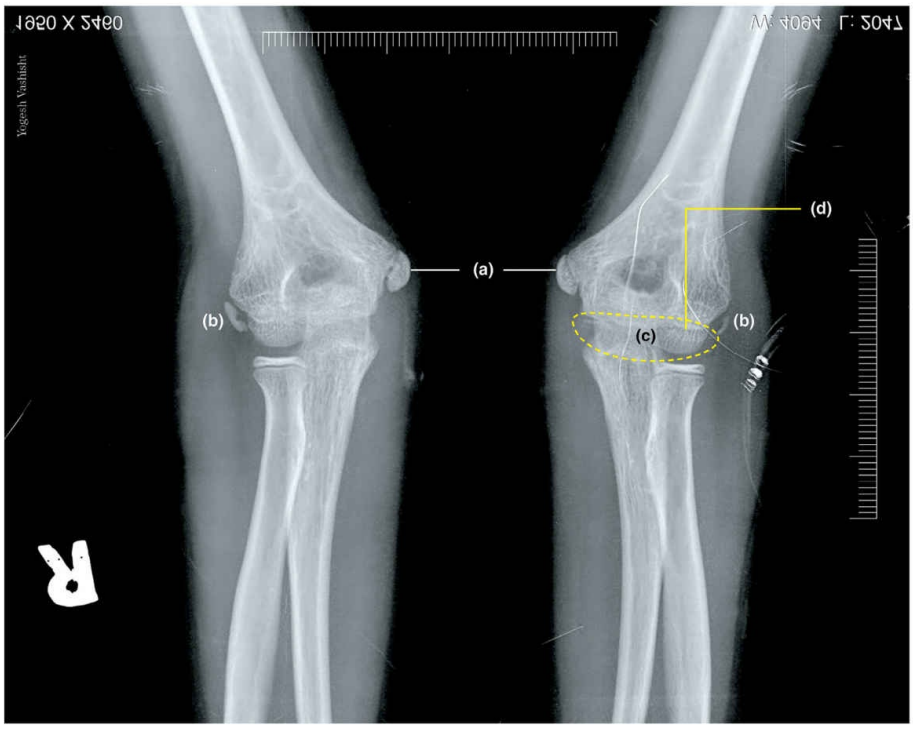
Table 24: Ossification of lower end of humerus

Criteria	Females	Males
Conjoint epiphysis forms	12	13
Conjoint epiphysis unites with shaft	14	15
Medial epicondyle unites with shaft	15	16

Fig. 3.11: (1) Femur of a male slightly older than 16 years. Please note metaphyseal grooves at the base of head and greater trochanter. These are seen as running across the epiphyses. At 17 years, as epiphysis would have united with the diaphysis, they would have disappeared. (2) Lower end of the same femur. Please note that the lower end, which normally unites at 18 years has completely come off due to maceration, which was used to prepare this bone. The shaft is thus showing only the lower end of diaphysis; epiphysis is missing. Please note the rough surface of diaphysis. During life it was in opposition with the metaphysis (3) This rough surface is more clearly seen in this view. Please note numerous pits.



(1)



(2)

Fig. 3.12: (1) Ages of appearance of ossification centers of humerus (left); (2) X-ray of 13 y old girl. **(a)** Medial epicondyle appeared (>7 y); **(b)** Lateral epicondyle appeared (>11 y). **(c)** Conjoint epiphysis has formed (>12 y), but has not united with shaft (<14 y). **(d)** This black line represents metaphysis between conjoint epiphysis and shaft of humerus, showing non-union.

[III] **Radius and ulna**: Make a representation of the two bones [**Fig 3.13**]. Make a capital "N" beginning from the lower end of radius as shown, and write numerals 2"5"6"9 (1) Lower end of radius appears at 2 years (2) Upper end of radius appears at 5 years (3) Lower end of ulna appears at 6 years (4) Upper end of ulna appears at 9 years [IV] **Femur**: Remember **1** -> **4** -> **14** [formed by the first two digits]. Head of femur appears by **1** year, greater trochanter by **4** years and lesser trochanter by **14** years [**Fig 3.14**].

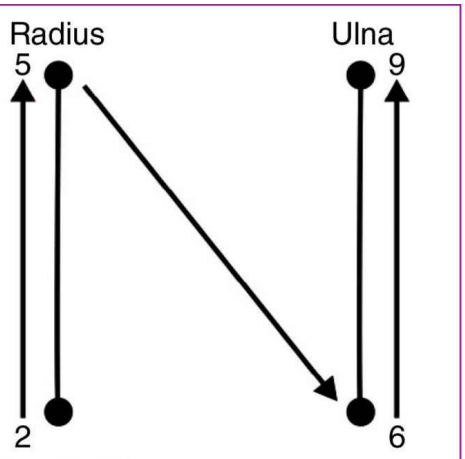


Fig. 3.13: A simple way to remember ossification centers of radius and ulna. Pl correlate with X-rays in **Fig 3.9** [**visual Memory Aid – 15**]

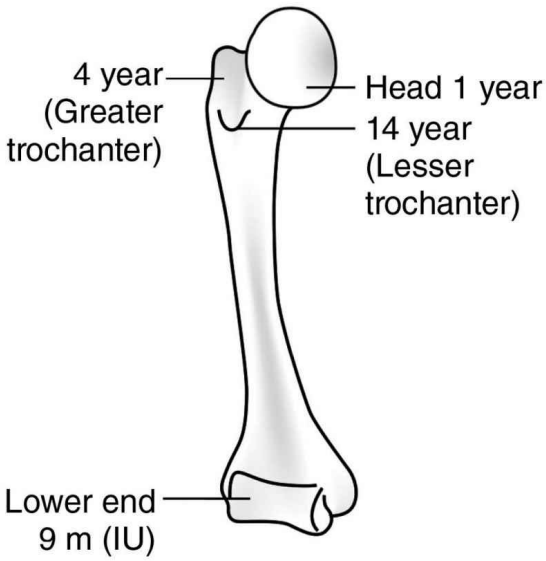


Fig. 3.14: Ossification centers in femur

Angle of neck with shaft 150° in newborn. Angle in adults is different depending on sex [**Table 18**]. [V] **Tibia and fibula**: Make the bones (as in case of radius and ulna) and write a capital "U". Now write the numerals 0"1"1"4 **[Fig 3.15]** (1) Upper end of tibia appears at 0 years, i.e. just at birth [in **80%** cases**;** in rest it appears soon after birth] (2) Lower end of tibia appears at 1 year (3) Lower end of fibula appears at 1 year (4) Upper end of fibula appears at 4 years.

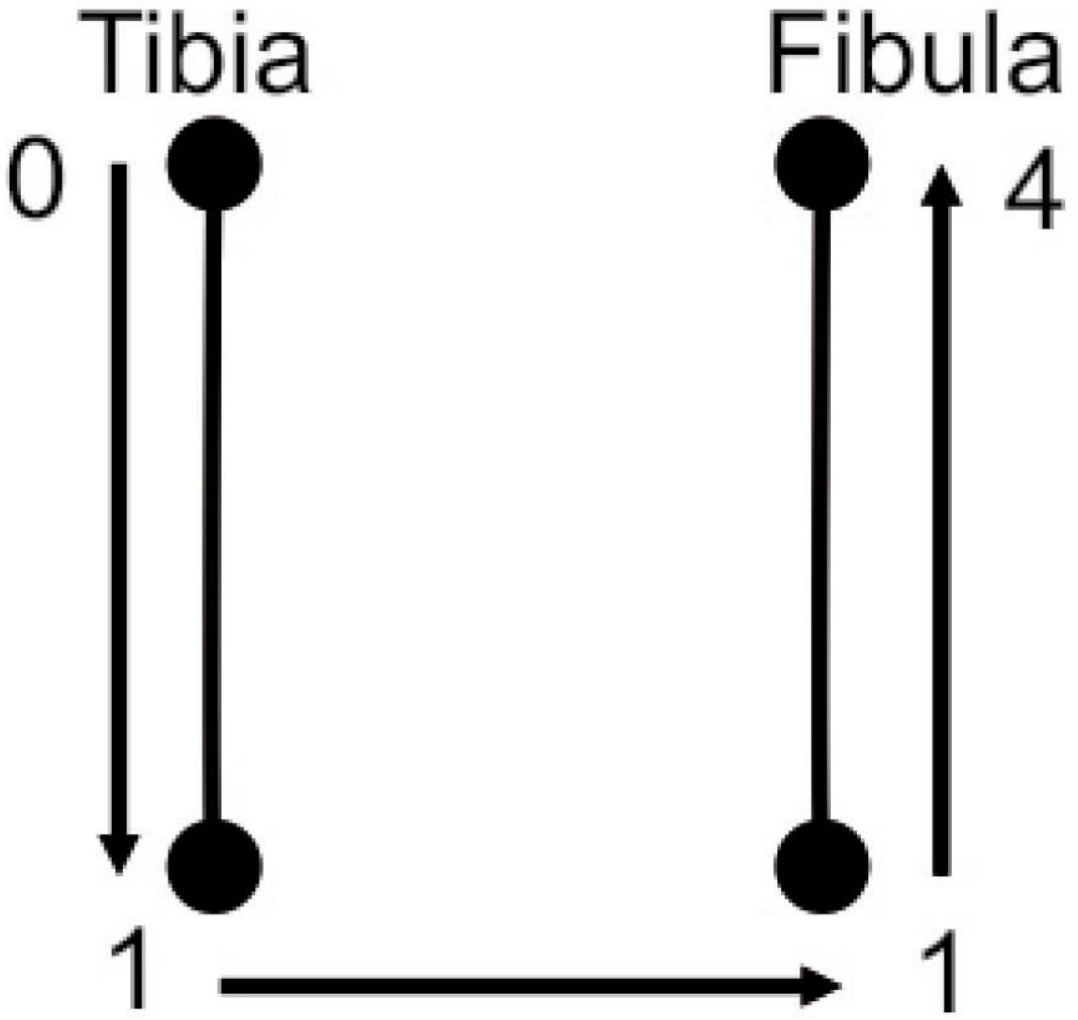


Fig. 3.15: A simple way to remember ossification centers of tibia and fibula [**visual Memory Aid – 16**]

2nd—Ages of fusion

Age of fusion may be remembered by an easy mnemonic. Imagine a person reclining on a sandy beach [**Fig 3.16**]. His elbows are sinking in the sand

somewhat. Draw four lines parallel to the horizontal plane. The lowest one passes through the elbow, the next through the hip and ankle joints, the next through the shoulder and knee joint, and the uppermost through the wrist joint. Epiphyses falling on a particular line fuse at a particular age. All epiphyses around the elbow joint complete fusion by 16 years, all epiphyses around hip and ankle joints by 17 years, all epiphyses around shoulder and knee by 18 years, and all epiphyses around the wrist by 19 years.

Following three centres fall upon the lowest line – ossification centres around elbow joint.

(1) Medial epicondyle unites with the shaft at 16 y
(2) Upper end of radius unites with the shaft at 16 y
(3) Upper end of ulna unites with the shaft at 16 y. Following three centers fall on the second line from below.
(4) Head of femur unites with the shaft at 17 y.
(5) Lower end of tibia unites with the shaft at 17 y.
(6) Lower end of fibula unites with the shaft at 17 y. Following four centers fall on the third line from below.
(7) Head of humerus unites with the shaft at 18 y.
(8) Lower end of femur unites with the shaft at 18 y.
(9) Upper end of tibia unites with the shaft at 18 y.
(10) Upper end of fibula unites with the shaft at 18 y. Following two centers fall on the fourth line from below.
(11) Lower end of radius unites with the shaft at 19 y.
(12) Lower end of ulna unites with the shaft at 19 y.

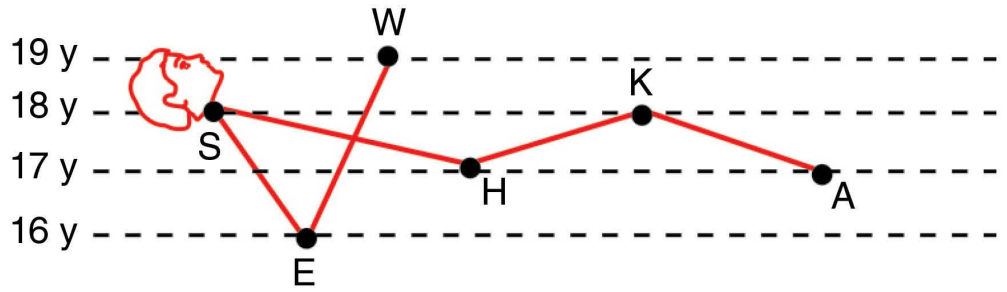


Fig. 3.16: A simple way to remember ages of fusion in some major bones [with **visual Memory Aid 17**].

3rd—Epiphyseal scar

Epiphyseal scar is a radiodense band seen in X-rays at the junction of the epiphysis and metaphysis, which represents closure. It signifies recent union, and disappears a few years after. Rarely multiple transverse lines are seen at diaphyseal ends. They often indicate growth disturbances due to scurvy, rickets etc [**scars of arrested growth**].

(b) Skull

(1) Determined by X-rays. Changes given in detail in a subsequent section.
(2) For suture closure, x-ray requested should be
(i) Anteroposterior (Towne's) X-ray of skull
(ii) Posteroanterior X-ray of skull
(iii) Submentovertical X-ray of skull.

(c) Hyoid

(1) Hyoid ossifies from 6 centers - 2 for the body and one for each cornu.
(2) Ossification begins in
(i) Greater cornu"10 m [IU life].
(ii) Body"just after birth.
(iii) Lesser cornu"16 y.
(3) Completion of ossification -
(i) Apices of greater cornu"20-30 y
(ii) Greater cornu of hyoid unites with the body at 40-60 y.

(d) Sternum

(1) **Anatomy** - Sternum consists of three parts – the manubrium sterni, the body of the sternum and the xiphoid process. The body of the sternum has four sternebrae. Thus the whole bone may be visualized as being composed of 6 parts [manubrium sterni + 4 sternebrae + xiphoid process].
(2) Centers appear from above downwards, and unite from below upwards. The union occurs in two different "waves" so to say – hence the two upward arrows.
(3) The centers for the manubrium sterni and the sternebrae appear during the IU life. Remember the figures 5m"5m"7m"7m"10m"3y. All months refer to intrauterine period. The center for xiphisternum appears at 3 years of age.
(4) In the first "wave" of union, the four sternebrae unite from below upwards. The figures are:

(i) Union of 3rd and 4th sternebrae at **15** years

(ii) Union of 2nd and 3rd sternebrae at **20** years

(iii) Union of 1st and 2nd sternebrae at **25** years.

(5) In the second "wave" of union:

(i) xiphisternum unites with the body at **40** years.

(ii) Manubrium sterni unites with the body at **60-70** years, and quite often remains ununited.

(e) Clavicle

(1) Clavicle is the first bone to begin ossification.

(2) **Primary centers** -Two centers appear in the shaft between the 5th-6th weeks of IU life, and fuse at 45th day.

(3) **Secondary centers:**

(i) Medial end appears between 11-19 y [average – 15 y] and unites with the shaft between 18-25 y [average - 22].

(ii) There is no lateral end of ossification in clavicle.

Memory Aid 18: Ossification centers of sternum

Sternum – 1st piece, i.e. Manubrium sterni appears at **5** [**S** looks like **5**]. Last piece. i.e. 4th sternebra appears at **t**en – second letter of sternum is **t**]. All other pieces appear in "pairs", i.e. MS and 1st sternebra at **5**; 2nd and 3rd sternebrae at **7**.

(f) Scapula

(1) **Coracoid** - 1 y [A]; 15 y [F]

(2) **Subcoracoid -** 16 y [A]; 17 y [F]

(3) **Acromion** - 16 y [A]; 20 y [F]

(4) All epiphyses of the scapula join the bone - **20 y**.

(5) **30-35 y** " Lipping starts on the ventral margin of glenoid cavity

(6) **35-40 y** " Irregular lipping around clavicular facet and inf surface of acromion process

(7) **45 y** " Localized bony atrophy

(8) **50 y** " Cristae scapulae.

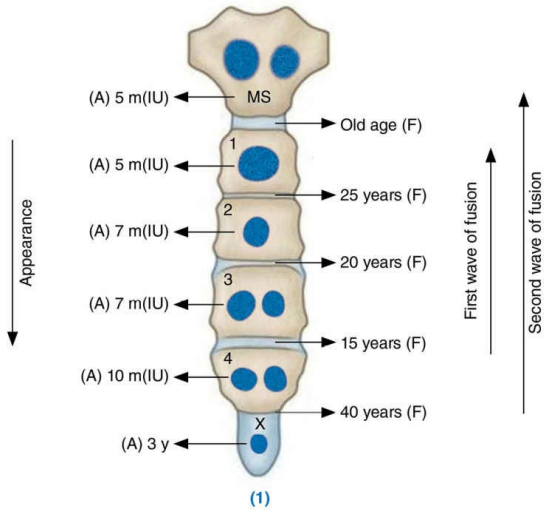


Fig. 3.17: (1) Ages of appearances and fusion of ossification centers of sternum; (2) Sternum of a 7 month old viable baby, incised in the midline. Pl note that the centers of manubrium and upper 3 sternebrae have appeared, but not that of 4th sternebrae and xiphisternum (3) Sternum of a 22 y old male. Pl see that S2 and S3 have united [>20 y], but not S1 and S2 [<25 y]. Pl also see (Fig. 3.20), where sternum is viewed against bright light, another technique to visualize sternal centers.

(g) Carpals

Write down the first letters of all carpal bones as shown.

Memory Aid 19: Carpal bones

Mnemonic 1 - She **L**ooks **T**oo **P**retty; **T**ry **T**o **C**atch **H**er. [lateral to medial; proximal to distal]

Mnemonic 2 - Teacher **t**eaches **C**unning**h**am; **S**tudents **l**ike **t**o **p**lay [lateral to medial; distal to proximal]

Now encircle the pisiform bone. Start from capitate bone and make a full circle as shown. The ages (all extra uterine) are:

(1) **Capitate**"**2** months
(2) **Hamate**"**3** months
(3) **Pisiform**"We pass this bone, as this is encircled
(4) **Triquetral**"since this starts with “tri” we cannot assign the next number. It has to be “tri” or **3**. So Triquetral appears at **3** years
(5) **Lunate**"**4** years
(6) **Scaphoid**"**4-5** years
(7) **Trapezium** and **trapezoid**"**4-5** years (Trapezium comes slightly earlier than Trapezoid)
(8) **Pisiform**: We finally land on this bone. It appears between **9-12** years. It is the last carpal bone to appear.

(h) Vertebrae

(1) **At birth** - Vertebral body shows a series of deep radial furrows both on the

upper and lower surfaces

(2) **birth-10 y** – Furrows -in prominence with age

(3) **10-25 y** – Furrows start . ing in prominence

(4) **>45 y** – Osteoarthritic changes, lipping, osteophytes.

(i) Symphysis pubis

(1) For changes in symphysis pubis [SP], please see below In the living, CT can demonstrate these changes.

(j) Hipbone

(1) Ischiopubic ramus unites by **7-8** years.

(2) For iliac crest and pubis, remember **14**"**20;** i.e. both iliac crest and pubis appear at 14 years of age and unite by 20 years.

(3) Iliac crest - union occurs from before backwards, i.e. the crest begins uniting at anterior superior iliac spine and the union gradually proceeds backwards.

(4) For ischium, remember **16**"**20;** i.e. ischium appears at 16 and unites at 20.

(5) For acetabulum, remember **13**"**15;** i.e. the acetabular cup assumes the shape of a triradiate cartilage, which becomes noticeable in radiographs by 13 years. This is generally taken as the “age of appearance” of triradiate cartilage.

(6) The triradiate cartilage disappears by **15** years.

(k) Sacrum

(1) **20 y** - Bodies of sacral vertebrae begin to unite with one another. They unite at margins but not in the central area.

(2) **25 y** -Sacrum is one bone, even though central area may remain unossified up to **50 y**; sometimes even later.

(3) The union occurs from below upwards. Sometimes a gap may remain between S1-S2 due to **lapsed union** [please see above].

(l) Patella

(1) Centers in **males** – Appear between **1½ -4** y.

(2) In **females** - between **2½ -6** y [Significantly the centers appear later in females].

(3) Patella assumes adult contours by **14** years in females and **16** years in males.

(4) Sometimes additional ossification centers appear which remain ununited, producing bipartite or multipartite patella. This is more common in sporting persons.

(m) Tarsals

(1) For secondary centers of calcaneum remember 7 Appearance ->16 (F)usion.

(2) Navicular appears at 3 years (just like xiphisternum in the upper half of the body).

(3) Cuneiforms - Lay down:

(i) cuboid

(ii) lateral cuneiform

(iii) intermediate cuneiform and

(iv) medial cuneiform in their anatomical fashion. Ossification centers in these bones appear from lateral to medial side [skip IC]. The years are 0, 1, 2, 3.

iii. Secondary sexual characters

Not very helpful. Give a very vague idea about the age. Best known staging of secondary sexual characters is by **James Tanner** (1920-2010). He was a British Pediatrician, who worked at the Institute of Child Health at the University of London. Developed formal stages of sexual development in puberty [Sexual Maturity Ratings or SMR].

(a) Tanner's stages of breast development [girls]

(1) **Tanner stage 1 [B_1, prepubertal, before 9-10 y]**:

No discernible breast tissue in prepubertal girls.

(2) **Tanner stage 2 [B_2, Breast bud stage, thelarche, 10-11 y]:**

(i) Breast development begins with an -in the pigmentation and diameter of the areola and the development of a small amount of breast tissue.

(ii) Breast development not seen beyond areola.

(3) **Tanner stage 3 [B_3, 12 y]:**

(i) Breast development occurs beyond areola;

(ii) areola experiences early changes [pigmentation, Montgomery glands].

(iii) One mound stage - Further breast enlargement, but there is no separation of the contours of the areola from the breast. This is all one mound.

(4) **Tanner stage 4 [B_4, 13 y]:**

Two mound stage - Formation of a double mound; secondary mounding of the areola above the plane of the breast, and of papilla [nipple] above areola.

(5) **Tanner stage 5 [B_5, final or mature stage]:**

(i) One mound stage again - Recession of the areola into the general contour of the breast. Projection of nipple only. Loss of double mound

(ii) further growth of the breast.

(b) Tanner's stages of genital development [boys]

(1) **Tanner stage 1 [G_1, prepubertal, 9-10 y]:**

(i) Testes, scrotum, penis are small sized.

(ii) No rugosities in scrotum.

(iii) Testicular volume 4-5 cc each. Testicular volumes can be measured by orchidometers, ultrasonography or simply by measuring length [L] and width [W] of testes" $\pi/6 \times L \times W_2$, testis being regarded as an ellipsoid.

(2) **Tanner stage 2 [G_2, prepubertal, 10-11 y]:**

(i) Skin of scrotum reddens

(ii) Scrotum shows changes in texture (slight rugation).

(iii) Enlargement of penis, scrotum and testes

(iv) Testicular volume 6-7 cc each

(3) **Tanner stage 3 [G_3, prepubertal, 12 y]:**

(i) Further growth of scrotum, testes and penis. The latter -es at first mainly in length; very slight -in breadth

(ii) Testicular volume 15 cc each.

(4) **Tanner stage 4 [G_4, prepubertal, 13-14 y]:**

(i) Scrotal skin darkens.

(ii) Glans develops.

(iii) Further enlargement of penis, scrotum and testes

(iv) Testicular volume 20 cc each.

(5) **Tanner stage 5 [G_5, prepubertal, 15-16 y]:**

(i) Genitalia adult in size and shape.

(ii) Testicular volume 25-30 cc each.

(c) Tanner's stages of pubic hair development [both sexes]

(1) **Tanner stage 1 [PH_1, prepubertal, <12 y]:**

(i) No pubic hair [< 12 y]

(2) **Tanner stage 2 [PH_2, prepubertal, 12-13 y]:**

(i) Sparse growth of long, slightly pigmented downy hair, straight or very slightly curled, chiefly at base of penis or along labia. [12-13 y]

(3) **Tanner stage 3 [PH_3, prepubertal, 13-14 y]:**

(i) Darker, coarse extending on to mons pubis, pigmented, start curling and spread around

(4) **Tanner stage 4 [PH_4, prepubertal, 14-15 y]:**

(i) Cover most parts but do not go up to thighs

(5) **Tanner stage 5 [PH_5, pubertal, >15 y]:**

(i) Mature pubic dense hair extend to the inner thighs (>15 years).

(d) Secondary sexual features in males according to age

(1) **14 y:**

(i) fine downy hair begin to appear around the pubes.

(ii) Testes become larger and firmer.

(iii) Penis begins to enlarge

(2) **15 y:**

(i) Hair moderately grown on pubes.

(ii) Begin to appear in axilla

(3) **16 -18 y:**

(i) Hair on pubes well grown.

(ii) Begin to appear on face [moustache, beard]

(iii) Adam's apple becomes more prominent

(iv) Voice – becomes hoarse

(v) External genitalia have an adult appearance.

(4) **20 y:**

(i) Color of hair becomes darker

(ii) Hair become thicker

(iii) Hair begin to appear on the inner sides of the thigh, scrotum and anus.

Memory Aid 20: Appearance of body hair

Hair on the body appear from below upwards

(e) Secondary sexual features in females according to age

(1) **13 y**"

(i) Breasts - begin to develop

(ii) fine, pale downy hair appear on mons veneris [about 2 m after breast change]

(iii) Labia – develop

(iv) Menstruation – starts

(2) **14-15 y**"
(i) Pubic hair is well-grown
(ii) Hair appears in axilla.

(f) Fallacies of secondary sexual characteristics

(1) **Sexual precocity**-(i) **McCune–Albright syndrome** [syn, **Albright syndrome**] – Secondary characteristics appear earlier than average age.

(2) **Breast in males** [**Gynecomastia**]– (i) **Causes** – Pl see memory aid.

Memory Aid 21: Causes of Gynecomastia

CT SCAN – **C**imetidine, **T**estosterone, **S**pironolactone (antiandrogen), **C**annabis, **A**lcohol, **N**ifedipine

b. Procedure for determination of age

(1) **Request** - Age is determined by a doctor on
(i) private request, [eg when a couple want to marry in court and require an age certificate]
(ii) police request [eg age determination in a girl to determine if it was a case of statutory rape]
(iii) court order [eg to know if a child is juvenile]

(2) **Preliminaries** – please see **ch 1**"Medicolegal reports" Preliminaries. Important additions are (i) Age – record both as alleged by subject as well as that alleged by police or contesting party

(3) **Body of report** – please see **ch 1**. Note especially
(i) ht and wt
(ii) Secondary sexual characters – eg distribution of hair on body, development of genitals, breasts etc, voice

(4) **Opinion and subsidiaries** – please see **ch 1**. Opinion should be based on physical, dental and radiological examination. If there is a conflict between them generally the levels of preference should be as follows; greatest regard to radiological data followed by "dental followed by"physical findings.

c. Age of the fetus

i. Stages of development of fetus

Pl. see **ch. 26**.

ii. Month wise development of fetus

Following dates are from LMP. The months are lunar (28 days each).

(1) **End of 1st month:**

(i) Length – 1cm

(ii) Wt - 2.5g

(iii) Eyes – seen as 2 dark spots

(iv) Mouth – seen as cleft

(v) Nucleated red cells - begin to form in placenta

(2) **End of 2nd month:**

(i) Length – 4 cm

(ii) Wt - 10g

(iii) Hands and feet – Webbed

(iv) Anus – seen as a dark spot

(v) ossification centers – in clavicle and maxilla

(vi) Placenta begins to form

(3) **End of 3rd month:**

(i) Length – 9 cm

(ii) Weight – 30 g

(iii) Eyes – (a) closed (b) pupillary membrane appears

(iv) neck is formed

(v) Early fingernail development.

(vi) Intestines in abdomen.

(4) **End of 4th month:**

(i) Sex: distinguishable externally.

(ii) Neck: Well defined.

(iii) Head: erect.

(iv) Brain: convolutions begin to form

(v) Lower limbs: well developed.

(vi) Placenta and fetus are equal in weight [17 wks].

(vii) Meconium: begins to form [13th week] In the first trimester, placental growth is more rapid than that of the fetus. It is at beginning of small intestine [duodenum].

(5) **End of 5th month:**

(i) Ears stand out from head.

(ii) Vernix caseosa appears.

(iii) Early toenail development.
(iv) Meconium: at beginning of large intestine

(6) **End of 6th month:**

(i) Head and body (lanugo) hair visible.
(ii) Skin wrinkled and red.
(iii) Eyebrows and eyelashes – appear
(iv) Skin – red, wrinkled. S/c fat begins to be deposited
(v) Meconium – at transverse colon
(vi) Testes – close to kidneys

(7) **End of 7th month** (**age of viability**):

(i) wt 1100 g.
(ii) Fingernails – present and thick.
(iii) Lean body.
(iv) Eyes - (a) partially open (b) pupillary membrane - disappears.
(v) Meconium – in entire large intestine
(vi) Testes – at external inguinal ring
(vii) Ossification center – appears in talus. This is the most imp center medicolegally since it appears at age of viability. For all IU ossification centers, please see above.
(viii) Neonate born at this age has a 90% chance of survival without physical or neurological impairment.

(8) **End of 8th month:**

(i) Eyes open.
(ii) Scalp hair – thicker, length -1.5 cm
(iii) Skin slightly wrinkled.
(iv) Nails – (a) Fingernails reach tip of fingers (b) Toenails present.
(v) Body filling out.
(vi) Left testis – in scrotum.

(9) **End of 9th month:**

(i) Fingernails reach fingertips.
(ii) Skin pink and smooth.
(iii) Scrotum – wrinkled, contains both testes

(10) **End of 10th month:**

(i) Length – (a) Crown-Heel – 50 cm [48-52 cm] (b) Crown-Rump – 30 cm [28-

32 cm] (c) Umbilicus – placed midway between pubis and xiphoid cartilage

(ii) Weight – (a) 3.4 kg [2.5-5 kg]. (b) male infant weighs about 100 g more than female (c) Weight is more variable than length

(iii) Circumference of head – 33-38 cm [Head circumference at any given time is roughly equal to 2/3rd of CH length – same is true of CR length also please see below]

(iv) Head =1/4 x body length

(v) Scalp hair – dark, 3-5 cm long

(vi) size of anterior fontanelle – 4 x 2.5cm

(vii) face – not wrinkled

(viii) Lanugo hairs – absent, except on shoulders

(ix) skin – covered with vernix caseosa

(x) Nails - (a) Fingernails - extend beyond fingertips. (b) Toenails – just reach toe tips. (c) Nose and ears – cartilages have formed

(xi) Umbilical cord – Length 50-55 cm. Thickness 1 cm.

Memory Aid 22: Age of fetus [Pl co-relate with Table 25 below.]

Write figures **1-10** in a **vertical row** as in **Table 25**. These represent lunar months (end of). Now write features appearing at each month as below:

(1) **At 1m** 2 dark spots; at **2m** 1 dark spot

(2) **1 imp thing happens at 1m** - nucleated **RBC** in placenta

(3) **2 things are webbed at 2m** - **hand** and **feet** [mentioned later also in a different mnemonic]

(4) By now we have 2 features each for 1 and 2 months; total **4 features**. Use following mnemonic now:

Post **M**ortem **S**o **T**ough – Arrange them in front of figures **3, 4, 5 and 6** from above downwards, so that P comes in front of 3; M in front of 4; S in front of 5 and T in front of 6. Now proceed as below.

(5) **Pupillary membrane** - **3 , 7** [appearance, disappearance]

(6) **Meconium** - **4, 5, 6, 7**: 4-duodenum, 5-beginning of **LI [cecum]**, 6-Beginning of **TC**, 7-entire **LI.**

(7) **S kin related** - **5, 6**: 5-vernix caseosa appears; 6-head and body hair visible, eyebrows, eyelashes appear.

(8) **Testes** - **6, 7, 8, 9** [start **2 m** after meconium]: **6**-Close to kidneys, **7**-external inguinal ring, **8**-left testis descends [Mnemonic to remember that left descends first - **L**ecture **T**heatre; or co-relate with left umbilical vein - Left is left], **9**-both testes in scrotum.

(9) **Fingernails** - [start **2 m** after testes]- 8,10 : **8**-Fingernails reach tips of fingers, **10**-Fingernails beyond tips; **T**oe nails just at tips. Remember: **T**oe nails are at **T**ips at **T**en.

(10) **Now weights** – In front of numerals 1, 2, 3, write **2.5 g**; **10 g**; **30 g** respectively [Note that for

3 months, just 0 has to be added]. At 7 months [viabIl Ity]– **1100 g. [Take 2 vertical "I"s from viability and add 2 "0"s 1100]**

(11) **Now first formations** [Mostly mentioned in earlier mnemonic also]: **1**- **Nucleated RBC in placenta**; **2** - **Webbing of hand and feet**; **3** - **Neck**; **4** - **Brain convolutions**; **5** - **Vernix caseosa**; **6** - **Hair** [eyebrows, eyelashes]. Nothing much forms after **6m**. In fact disappearance starts at **7m pupillary membrane disappears**.

(12) **4 Imp things happen at 4** m:

(i) **sex** – distinguishable [most imp]

(ii) **neck** – well defined [begins to form at **3 m**]

(iii) **brain convolutions** - begin to form

(iv) **Placenta and fetus** – equal in wt [17 wks]

(13) **Talk of 10 months now** –

(i) **length** -50 cm

(ii) **umbilical cord** - 50 cm

(iii) **umbilicus** - midway between xiphoid and pubis

Table 25: Age of fetus [pl co-relate with **Memory Aid 22 above**.]

Lunar month	Features
1	2 dark spots appear [eyes]; nucleated **RBC** in placenta; wt = 2.5g
2	1 dark spot appears [anus]; **hand** and **feet are webbed**; wt = 10g
3	Pupillary membrane appears; wt = 30g; neck begins to form
4	Meconium at duodenum; brain convolutions begin to form; sex is distinguishable; neck well defined; placenta and fetus are equal in weight [17 wks]
5	Skin related-vernix caseosa appears; Meconium at beginning of large intestine [cecum]
6	Testes close to kidneys: meconium at beginning of transverse colon; head and body hair visible; eyebrows, eyelashes appear
7	Pupillary membrane disappears; Meconium in the entire large intestine; testes at external inguinal ring; wt=1100g
8	Left testis descends; fingernails reach tip of fingers
9	Right testis descends; both testes in scrotum
10	Fingernails beyond tips; toe nail just at tip; CH length – 50 cm; umbilical cord – 50 cm; umbilicus - midway between xiphoid and pubis

iii. Rule of Haase

[A] ***Rule of Haase*** states that during the first 5 months of development of fetus, the crown-heel (CH) length in cm is equal to the square of its age in lunar months; afterwards it is equal to 5 multiplied by its age in lunar months. Thus

CH length of a fetus from **1-5 lunar months** is: 1 m"1^2= 1 cm; 2 m"2^2= 4 cm; 3 m"3^2= 9 cm; 4 m"4^2= 16 cm and 5 m"5^2= 25 cm. From **6-10 lunar months** it is: 6 m"6x5= 30 cm; 7 m"7x5= 35 cm; 8 m"8x5= 40 cm; 9 m"9x5= 45 cm; 10 m (full term)"10x5= 50 cm; (1 lunar month = 28 days). [B] **CR length** is **medicolegally more important** than CH length because of (1) The variability in the length of the legs (2) Difficulty of maintaining them in extension. Despite this, Rule of Haase deals with CH length [**Memory Aid 23:** H for

H

aase"thus C

H

], because that is easier to remember. Karl Friedrich **Haase** (1788-1865), a German gynaecologist reported the birthlength statistics for the Berlin Charité for **1875**. In a footnote he added this famous mnemonic. CR length at any given time is roughly equal to 2/3rd of CH length.

iv. Development after birth

(a) Length

Length (i) at birth = 50 cm;

(ii) at end of 6 months = 60 cm;

Memory Aid 24: - in infant length

At 6 m, add a "0" after 6 to get length " 60 cm

(iii) at end of 1st year = 68 cm; (iv) at end of 4th y =100 cm [double the length at birth].

Memory Aid 25: Infant measurements

1. **HS** - **H**eight is **s**low to increase.
2. Double the double, i.e. 4
3. Now write 4 5
4. Now write bigger unit first (y) and smaller later (m) in front of **4** and **5**. Joining them together gives circumference of head at 1 y [**45 cm**]
5. **4 y** height doubles; **5 m** weight doubles.
6. Head circumference till **1 y** is **2/3 of CH** length.
7. At **1 y**, when height is **68 cm** and circumference is **45 cm**.

(b) Weight

Birth weight (i) at birth = 2500 gm (average); (ii) at 5 months = doubles.

Memory Aid 26: - in infant weight

At **5 months**, wt is **5 kg**.

(iii) at 1 year = triples [7.5 kg].

(c) Head circumference

Head circumference (i) at birth = 33 cm (average); (ii) 1 year = 45 cm.

2. Age Estimation in the Dead

a. Methods of determination

Virtually all criteria used in the living can be used in the dead also. But certain additional criteria become available in the dead.

i. Teeth

(a) Gustafson's Method

In 1950, Swedish forensic odontologist **Gösta Gustafson** (1906 - 2001) developed an objective method for determining age from teeth in the dead.

[A] **Criteria** – Six criteria are used [**Fig 3.18**]:

Memory Aid 27: Gustafson's Method
[Mnemonic 1]
SCRIPT

Secondary dentin, **C**ementum apposition

Root resorption, Attr**I**tion

Periodontosis, **T**ransparency of root

OR

[Mnemonic 2]

Always **P**urchase **S**econd **C**lass **R**ailway **T**icket

Attrition, **P**eriodontosis

Secondary dentine, **C**ementum apposition

Root resorption, Root **T**ransparency

(1) **Secondary dentin:**

(i) With advancing age, dentin develops within the pulp cavity decreasing its size [**Fig 3.18**]

(ii) Initially it is deposited in the upper part of the pulp cavity. Gradually it extends lower down till the apex of the root, filling up the entire pulp cavity

(iii) Dentine forms at an average rate of 4 **µ**/day for man.

(iv) Scoring : S_0 – No deposition; S_1 –Deposition in the upper part; S_2 – Pulp

cavity half filled; S_3 – More than half to whole pulp cavity filled.

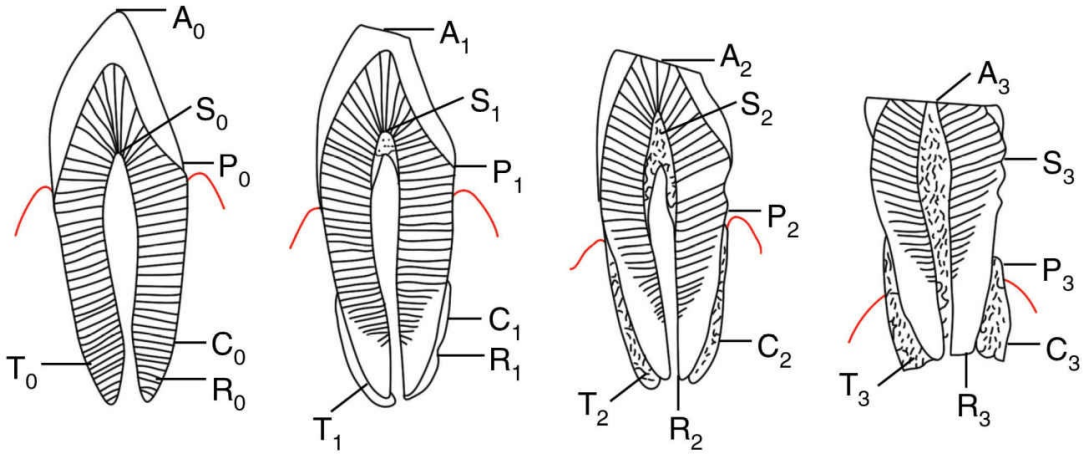


Fig. 3.18: Determination of age by Gustafson's Method. Subscripts refer to values for the assessment of age changes in (S) Secondary dentin, (C) Cementum, (R) Root Resorption, (A) Attrition (P) Periodontosis, (T) Transparency of the root.

(2) **Cementum apposition:**

(i) Cementum is a specialized calcified substance covering the root of a tooth.

(ii) It is light yellow and slightly lighter in color than dentin.

(iii) Cementum is formed continuously throughout life; a new layer of cementum is deposited to keep the attachment intact as the older layer of cementum ages. It can thus be used to calculate age. (iv) Scoring: $\mathbf{C_0}$ –Normal layer of cementum; $\mathbf{C_1}$ – Deposition slightly greater than normal; $\mathbf{C_2}$ – Great layer of cementum; $\mathbf{C_3}$ – Heavy layer of cementum

(3) **Root resorption:**

(i) Resorption of root starts at the apex, and extends upwards

(ii) Shows characteristically as sharp grooves

(iii) Scoring: $\mathbf{R_0}$ – No root resorption; $\mathbf{R_1}$ – Root resorption only on small isolated spots; $\mathbf{R_2}$ – Seen over a large area; $\mathbf{R_3}$ – Great areas of both cementum and dentin affected.

(iv) It is the least reliable of all criteria.

(4) **Attr Ition:**

(i) Wearing down of incisal or occlusal surface due to mastication

(ii) Scoring: $\mathbf{A}_0$ – No attrition; $\mathbf{A}_1$ – Attrition within enamel; $\mathbf{A}_2$ –Attrition reaching dentin; $\mathbf{A}_3$ – Attrition reaching pulp

(5) **Periodontosis:**

(i) Regression of gums and periodontal tissues surrounding the teeth occurs as age advances

(ii) Gradually necks and roots are exposed

(iii) Finally teeth may become lose and fall off

(iv) Scoring: $\mathbf{P}_0$ – No periodontosis; $\mathbf{P}_1$ – periodontosis just begun. Less than $1/3^{rd}$ of root exposed; $\mathbf{P}_2$ – $1/3^{rd}$ to $2/3^{rd}$ of root exposed; $\mathbf{P}_3$ – More than $2/3^{rd}$ of root exposed.

(6) **Transparency of root:**

(i) Cause of transparency - (a) C anals in the root are at first wide and filled with air. Light cannot pass through them as it is diffracted (b) As age advances, they are filled by mineral, making the entire area uniform. No diffraction of light occurs and root becomes visible (c) Transparency first appears in the apical region and then ascends upwards

(ii) Scoring: $\mathbf{T}_0$ – No Transparency; $\mathbf{T}_1$ – Transparency over apical region; $\mathbf{T}_2$ – Transparency over apical $1/3^{rd}$ of root; $\mathbf{T}_3$ – Transparency over apical $2/3^{rd}$ of root.

(iii) Most objective criteria, because area of root transparency can directly be measured by microscope

(iv) It is the single most reliable criteria followed by secondary dentin formation.

[B] **Technique:**

(1) **Most suitable teeth** – are anterior teeth (incisors). Merit .s as one goes back from incisors to molars. Molars are quite unsuitable

(2) **Observation of paradentosis** – Before extraction of incisors, paradentosis is measured, and score noted

(3) Tooth extracted, cleaned under running water, disinfected in 3% H_2O_2 and dried at room temperature.

(4) Embedded in resin, and ground down perpendicular to the axis

(5) When the thickness of tooth is reduced to **1mm**, transparency is estimated

(6) Tooth is now further ground down to **100µm** and other changes noted. Scoring of all 6 criteria done as mentioned earlier. All scores are added. Minimum score can be **0** [No change (0) in any criteria. On an average this

happens **up to 15 years**] and maximum **18** [maximum change (3) in all criteria] (7) Age can directly be read from an already plotted regression line [made from known cases] on a graph. (8) **Rate of error** is ±4 to 7 years. It -s after 50 years of age.

(b) Boyde's method

(1) **Ameloblasts** produce enamel at the rate of 4 µ/day.

(2) Therefore the enamel of the crowns of teeth shows **incremental striae** in histologic sections [striae of Retzius].

(3) When viewed microscopically in cross-section, they appear as concentric rings [similar to the annual rings on a tree]. In a longitudinal section, they appear as a series of dark bands.

(4) The **neonatal line** is the darkest band, which represents the **disrupted enamel** formation due to the stress of being born.

(5) Occasionally systemic disturbances in the human body [eg fever] can cause some lines to appear darker than those surrounding them.

(6) **Boyde** in **1963** suggested that counting the **enamel rings** outside of neonatal line can reveal age of a child.

Fig. 3.19: (1) Alan Boyde and **(2)** Enamel rings used by him in estimating age.

(c) Age from size and weight of teeth

Stack's method - Size and weight of anterior deciduous teeth (incisors and canines) are known to -with age. Thus age of a child can be calculated by measuring exact height and weight of anterior teeth.'

ii. Bones [determination of age in the dead]

Please also see above [Bones "determination of age in the living]. In the dead, following additional parameters become available.

(a) Ossification centers during IU life

Demonstration of ossification centers:

(1) Naked eye - All ossification centers on naked eye examination would appear as **brownish red spots** surrounded by **bluish white** cartilage. Significantly the same centers appear as ***whitish spots*** in radiographs. (a) **Sternum** - (I) **Sectioning** - Place sternum flat on a wooden board and cut with a cartilage

knife in its long axis in midline. **1-5 ossification centers** are seen depending upon the age of fetus. (II) **Visualizing against bright light** – Alternatively hold sternum against bright light. Centers of ossification will appear as dark spots within the bone [**Fig 3.20**]. [**Table 26**]. (b) **Lower end of femur and upper end of tibia** – (I) Leg is flexed against the thigh (II) A transverse incision is made in the knee joint (III) Patella removed (IV) End of femur pushed forward through the wound (V) Make a number of parallel cross-sectional incisions through the epiphysis starting from the articular surface and continuing till diaphysis is reached. Look for brownish red centers (VI) Diameters of ossification center - (1) At 36 wks, when the center appears, it is pinpoint in size (2) At 37-38 wks - diameter is 4-5 mm (3) At full term – diameter is 6-8 mm (VII) Examine upper end of tibia in same way (c) **Tarsals** – (I) Grasp foot behind the heel in left hand, with toes pointing towards the dissector (II) Make incision between the interspace of 3^{rd} and 4^{th} toes backwards through the sole of the foot and heel (III) Centers for talus and calcaneum should be seen as brownish red spots; if not seen, make thin incisions on either side till they are visible. The upper one is talus; lower is calcaneum. If not visible, it is interpreted as not appeared.

(2) **By staining - Alizarin Red** is used as a marker to stain centers of ossification.

Fig. 3.20: Sternal centers visualized against bright light. Centers for MS, S1 appeared [>5 m]; S2 and S3 appeared [>7 m]; S4 not appeared [<10 m]. Age 7-10 IU months. Pl also see (**Fig. 3.17**), where sternum is sectioned in the middle to see centers – another technique to see sternal centers.

Fig. 3.21: **(1)** Skull of a 1 year old infant. Please note ununited anterior fontanelle **(2)** Skull of a 1 month old infant. Please note ununited posterior fontanelle.

Table 26: Major ossification centers appearing during IU life with **Memory Aid – 28** The figures represent IU month [IUM].

CSI CAST TAKES SAFE CUSHION

- **C**"Clavicle"1½ IUM [followed by maxilla a few days later]
- **S**"Shafts of long bones"2 IUM
- **I**"Ischium"3 IUM
  [If one reads backwards from "I" it reads as "**Isc**", first 3 letters of "**ischium**". It also reads as "**SC**", **S**econd **C**ervical, which appears at **4th month**]
- **S**econd **C**ervical Vertebra"4 IUM
- **Ca**"Calcaneus"5 IUM
- **St**"Sternum (manubrium)"**6** IUM [by a previous mnemonic, manubrium sterni is taken to come at 5. It is okay to consider it as **5-6** months now]
- **Ta**"Talus"7 IUM [Age of viability]
  Kes"It stands for nothing
- **Sa**"Sacrum"**8** IUM
- **Fe**"Femur (lower end)"**9** IUM
- **Cu**"Cuboid"**10** IUM [or just before birth]

(b) Skull

(1) **Number of bones:**

(i) Calvaria [8]– 1 frontal, 2 Parietal, 2 temporal, 1 occipital, 1 ethmoid and 1 sphenoid.

(ii) Face and jaw [14] – 2 inferior nasal concha, 2 lacrimal, 2 maxillae, 2 nasal, 2 palatine, 2 zygomatic, 1 mandible and 1 vomer.

(2) Fontanelles:

(i) Posterior fontanelle closes by 2 months
(ii) anterior by 2 years.
(iii) Two postero-lateral fontanelles [mastoid fontanelles] close shortly after birth
(iv) Two antero-lateral fontanelles [sphenoidal fontanelles] at 6 months.

Memory Aid 29: Closure of fontanelles.
Fontanelles close from back to front.

(3) **Ossification:**
(i) Basiocciput unites with basisphenoid – 18-21 y
(ii) Condylar portions of occipital bone unite - (a)with squama – 3 y (b)with basiocciput – 5 y.
(iii) Inferior nasal concha - ossifies from a single center, which appears at 5th month of IUL in the lateral wall of the cartilaginous nasal capsule.

(4) **Suture closure:** [**Fig 3.22**]
(i) Skull sutures obliterate earlier in males than in females.
(ii) Suture closure begins at the inner table [5 y earlier] and progresses outwards.
(iii) Metopic suture – closes at 3 y. Persists in 5-10% cases
(iv) Other sutures - **Table 27**.

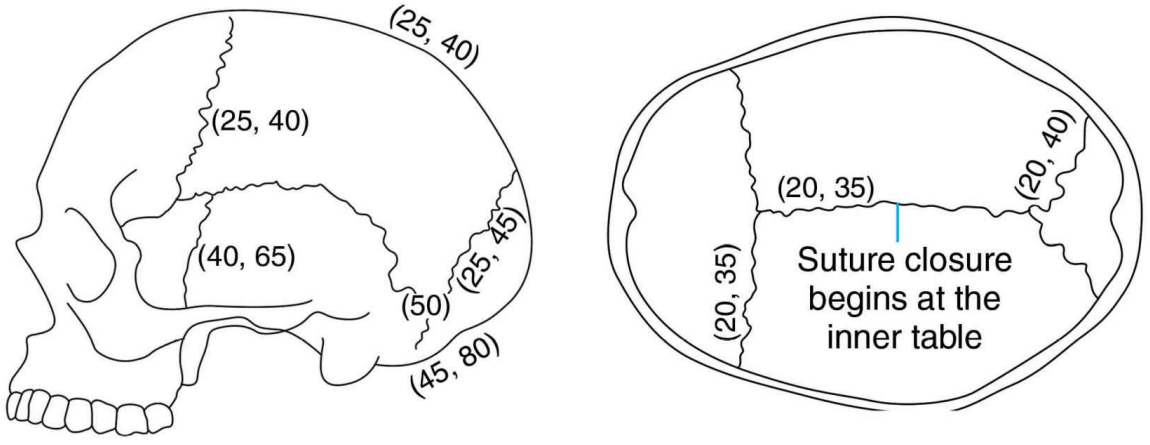


Fig. 3.22: Ectocranial, endocranial and palatine suture closure with age. Numbers represent ages of commencement and completion of suture closure

Table 27: Ages of skull suture closure.

Suture	Commencement	Halfway closed	Completion
Spheno-occipital synchondrosis	-	-	20
Coronal suture	25	30	40
Sagittal suture	25	30	40
Lambdoid suture	25	30	45
Pterion	40	-	65
Masto-occipital suture	45	-	80
Asterion	-	-	50

Memory Aid 30: Skull suture closure. Please correlate with Table 27.

1. Spheno-occipital is the earliest to fuse (20 years)
2. Coronal, sagittal and lambdoid start fusing together at 25 years. The frontal ones (coronal and sagittal) finish at 40 years while posterior one (Lambdoid) finishes a little later – at 45 years (remember "back benchers")
3. Two minor sutures are Pterion (at front) and Masto-occipital (at back)
4. Front one (pterion) starts when front sutures close (40 years)
5. Back one (Masto-occipital) starts when back sutures close (45 years)
6. Pterion takes 25 years to close (65 years)
7. Masto-occipital takes 35 years to close (80 years) [remember "back benchers" again]
8. Asterion closes by 50 years.

(5) **Other age criteria in skull:**

(i) 7 y - Vault almost reaches adult dimensions.

(ii) <10 y - skull is smooth and ivory like on both outer and inner surfaces

(iii) 25 y – Muscular markings become more prominent especially (a) Masseteric attachments (b)Nuchal lines and (c) Temporal line

(iv) 40 y - the surface begins to assume a "matted" granular rough appearance.

(v) 45-50 y - (a) On the inside of skull, pacchionian depressions for arachnoid granulation bulges - in number as well as depth, even to the point of penetrating the outer table of the vault. (b)Groove for middle meningeal artery becomes deeper

(vi) >50 y – (a) Diploe becomes less vascular (b) Thins out, as it is gradually replaced by bone (c) There is no consistent change in the thickness of skull.

(c) Mandible

(1) Mandible ossifies in dense fibromembranous tissue

(2) Each half ossifies from one center which appears near mental foramen at 6th week of life (just after the appearance of primary center for the clavicle)

(3) Two halves of mandible – Start uniting from below upwards in the 1st year. Completely unite by 2 y.

(4) Head of mandible completely ossifies by 30 y.

Memory Aid 31: Circle of 2.

Nature has made an interesting circle around the skull, which we call the "**circle of 2**" **[Fig. 3.23]**. As many as 5 structures unite at 2 (or a related figure) along this circle.

A. Symphysis menti unites at 2 years.

B. Metopic suture unites at 2 years (more correctly 2-8 years).

C. Anterior fontanelle unites at 2 years.

D. Posterior fontanelle unites at 2 months (more correctly 2-6 months).

E. Basisphenoid unites at 20 years.

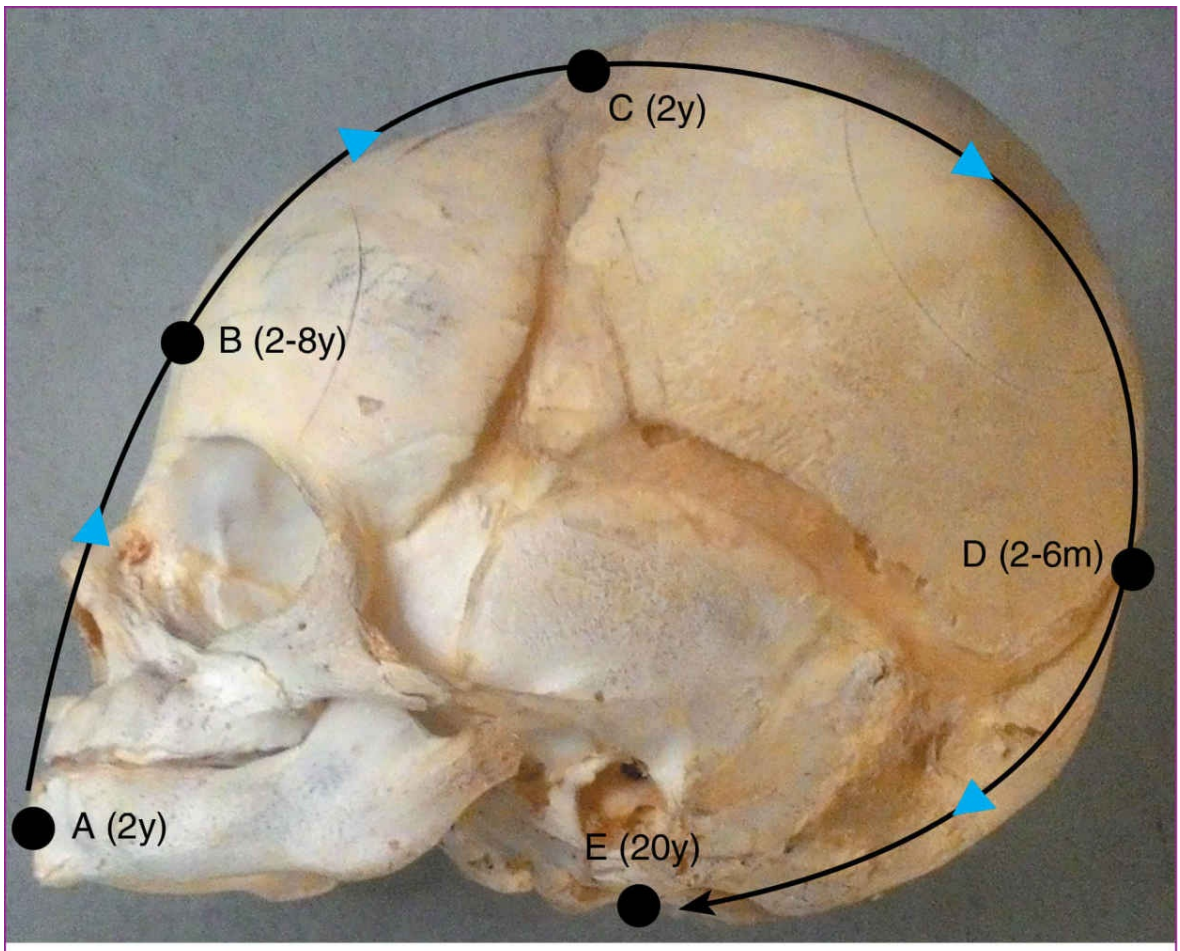


Fig. 3.23: Skull of a newborn. Please note both mastoid and sphenoid fontanelles are ununited. Please also note "circle of 2".

(d) Ribs

(1) It was reported in **1984** that sternal ends of ribs, particularly 4th rib show age related changes similar to pubic symphysis [please see below].

(2) surface is billowy in adolescence, while margins are sharp and cup shaped in middle aged adults, and irregular in old age

(3) For age determination right 4th rib is chosen.

Table 28: Differences in mandible in infancy, adult life and old age [Pl co-relate with **Fig. 3.24** (appearance of mandible at different ages); and also with **Fig. 3.17** and **Table 11** (both dealing with differences in mandible in sexes)]

S.No	Feature	Infancy	Adult	Old age
1.	Body	Shallow	Thick and long	Shallow
2.	Ramus	Short and oblique	Longer	Longer
3.	Angle between upper border of body and anterior border of ramus **[medicolegal angle]** (Fig on p. 49). [For anatomical angle, pl. see **Table 11**]	Obtuse. >140°	Almost a right angle	Obtuse [about 140°]
4.	Mental foramen	Towards the lower margin	Midway between upper and lower margin	Near the alveolar margin [It is useful to visualize mental foramen as traveling upwards during life]
5.	Condyloid process	At a lower level than coronoid process	Elongated and projects above coronoid process	At a lower level than coronoid process

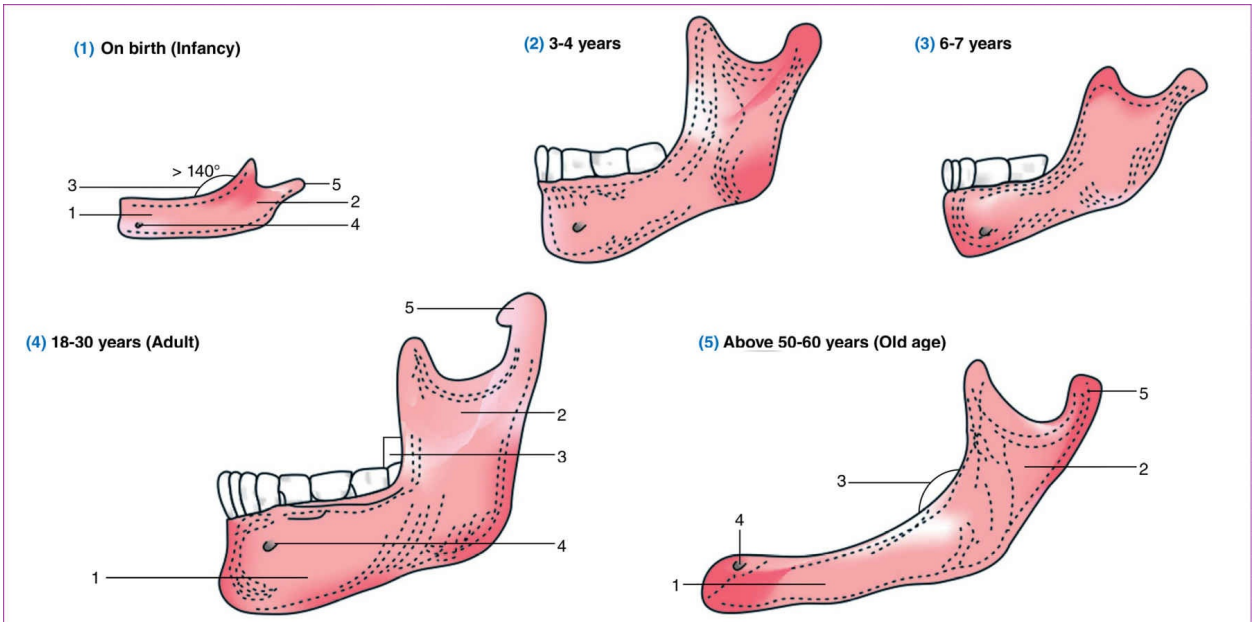


Fig. 3.24: Appearance of mandible at different ages. (please correlate nos. with **Table 28**).

(e) Symphysis pubis [SP]

(1) SP is the single best criterion for determining age after 25 y. Method is similar to that of rib end

(2) It requires bones free of cartilage

(3) While pubic symphyses of humans shows age changes, those of other primates metamorphose more quickly and usually synostose with advancing age.

(4) The young adult human SP has a rugged surface traversed by horizontal ridges and intervening grooves. This surface loses relief with age and is

bounded by a rim by age 35 Subsequent erosion and general deterioration of the surface are progressive changes after this age [**Fig 3.25**].

(5) SP changes were first observed by **Todd** in 1920, based on a series of **306 males** of known age. He noted evidence of billowing, ridging, ossific nodules, and texture on the symphyseal surface. Using these observations on his sample of known ages, Todd recognized **ten phases** of SP age, ranging from 18 y to 50+ y.

(6) In **1955**, **Brooks** modified Todd's system.

(7) In **1957**, **McKern and Stewart** modified Todd's system in their own way, using skeletal remains of 349 males who had died in the Korean War. Their ages were documented from military records. They divided the symphyseal surface longitudinally into two halves, or "components":

(i) the "**dorsal demiface**" [**Fig 3.25**].

(ii) the "**ventral demiface**" and added a 3rd component

(iii) the "**symphyseal rim**."

Six developmental stages [from 0-5] were recognized for each of the **3 components** [**Fig 3.25**]. Developmental stage for each component is calculated and added. The resultant score gives the age of the specimen. This system, like that of Todd, was derived from an all-male sample.

(8) **Drawbacks of McKern and Stewart's 3-component system:**

(i) The components do not vary independently

(ii) The system is unduly complex.

(9) Because of these drawbacks, in **1986**, **Darryl Katz** and **Judy Suchey** modified the Brooks' 1955 system. This is now widely known as **Suchey–Brooks (SB) system**, and is currently the most widely accepted system, although even this system continues to evolve and is yet to reach its final form. Like McKern and Stewart, they also suggested 6 phases, but with some differences. Significantly they made **casts of SP** [of all phases] which could be compared with the unknown sample. This method was obviously better than comparison with photographs. The casts made by **Diane France**, consist of a set of 12 models – 2 from each of the 6 phases; one illustrating an early pattern and one showing a later pattern.

(10) **Changes** [McKern and Stewart's method] – Changes are complex and must be attempted by an expert only. Broadly they are:

(i) **20 y** – Symphyseal surface is markedly irregular and uneven. Ridges run

transversely across the articular surface

(ii) **20-40 y** – (a) Ridges gradually disappear (b) Surface has a granular appearance (c) Ventral and dorsal margins form

(iii) **40-45 y** – symphyseal surface has an oval smooth surface, with raised upper and lower ends

(iv) **45-50 y** – A narrow beaded rim develops on the margins

(v) **51-60 y** – (a) Symphyseal surface erodes (b) breakdown of ventral margin begins

(vi) **61-70 y** – Surface is irregularly eroded.

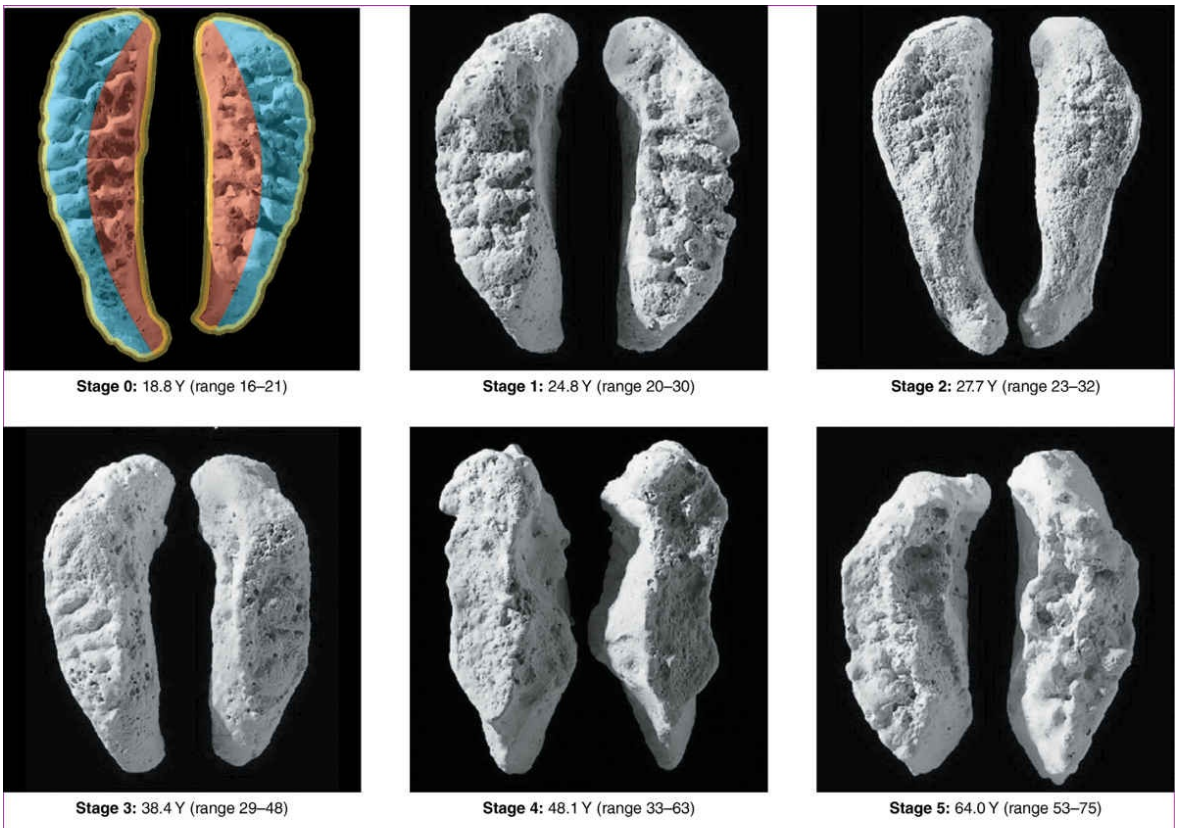

Stage 0: 18.8 Y (range 16–21)
Stage 1: 24.8 Y (range 20–30)
Stage 2: 27.7 Y (range 23–32)
Stage 3: 38.4 Y (range 29–48)
Stage 4: 48.1 Y (range 33–63)
Stage 5: 64.0 Y (range 53–75)

Fig. 3.25: Age changes in pubic symphysis. The figure at the top left depicts the 3-component system of McKern and Stewart. Blue depicts **dorsal demiface**; red depicts **ventral demiface** and yellow depicts the **symphyseal rim**. Note the prominent ridges and grooves in stage 0 and how they gradually disappear due to wear and tear.

3. ML importance of age

Memory Aid 32: MLI of age

CIRCŁE MIKE JIM [MERE MCI KI JAI - is another useful mnemonic, where 'A' becomes 'Abortion']

C-CRIMINAL RESPONSIBILITY, **C**ONSENT

I-INFANTICIDE

R-RAPE
C-CRIMINAL ABORTION
L – [dummy letter]
E-EMPLOYMENT
M-MAJORITY
I-IMPOTENCE AND STERILITY
K-KIDNAPPING
E-EVIDENCE
J-JUDICIAL PUNISHMENT
I-IDENTIFICATION
M-MARRIAGE CONTRACT

(1) **Criminal Responsibility:**

(i) **7 y** - Any act which is done by a child under 7 years of age is not an offence [**S.82, IPC**]. This is because such a child does not have mens rea [**ch 28**]. **Corollary** - A male child <7 y cannot be accused of rape [**ch 25**].

(ii) **7-12 y** - A child >7 y of age is presumed to have "attained sufficient maturity of understanding to judge the nature and consequences of his conduct". However if a child between 7 and 12 years can show that he did not attain such understanding, his action would not be considered an offence [**S.83, IPC**].

(iii) **12-18 y** – A child between 12 and 18 years would be tried only by a Juvenile Justice Board [JJB] [Children's court in layman's terms]

(iv) Railways Act [RA] 1989 - (a) **u/s 168(1)**, a child ***of any age below 12 years*** can be convicted. But no punishment will be given to such a child. Instead the father or guardian will have to execute a bond for such amount and for such period as the court may direct for the good conduct of such person.

Memory Aid 33: Section of Railways Act 1989, denoting age of criminal responsibility

S.168 – Remember 16. Half of it is 8. Put that in front. A child of **any age** can be convicted according to this section.

(b) If child repeats offence, the bond amount shall be forfeited [**168(2)**]. (c) If a father or guardian fails to execute a bond he shall be punishable with `50 fine [**168(3)**]. (d) The offences for which child may be convicted are (I) maliciously wrecking a train [S.150], (II) causing damage or destruction of railway property [S.151], (III) maliciously hurting persons travelling by railway [S.152], or (IV) endangering safety of persons traveling by railway willfully [S.153] or (V) negligently [S.154]. (e) It is wrongly taught

sometimes that under the Indian Railways Act, the age of criminal responsibility is 5 years. This is not true. A child of any age can be convicted. The important diff is that in conviction of children <12 y, the father or guardian will execute a bond of good conduct of the child; no punishment will be awarded to the child.

(2) **Consent:**

(i) **12 years** – A child >12 y can give consent for general physical examination [Act not intended to cause death, done by consent in good faith for person's benefit – **S.88, IPC**, **S.90, IPC**]

(ii) **18 years** – A person >18 y can give consent for undergoing surgery. (iii) **S.87, IPC - ch 2**.

(3) **Infanticide** – For a charge of Infanticide, infant must be proved to be <1 y. In India there is no specific law on infanticide [**ch 27**].

(4) **Rape:**

(i) sex with consent with wife of <15 y"Marital rape

(ii) sex with consent with any other girl <18 y"Statutory rape [**ch 25**].

(5) **Criminal Abortion** – Woman who has passed the child bearing age [usually 45y] cannot be charged of procuring criminal abortion.

(6) **Employment** -

(i) child <14 y cannot be allowed to work in any factory [please see below under MLI of **14 y**].

(ii) Young person 14-18 y – (a) can work in a factory only when a certificate of fitness has been given to him by a certifying surgeon (b) The certificate must be in custody of manager. (c) Such young person must carry a token giving a reference to such certificate [**S.68** The Factories Act, 1948]

(7) **Majority** – A person attains majority when he/she attains the age of 18 years [**S.3**, Indian Majority Act, 1875]. If person is under guardianship, the age of majority is 21 y [please see below under MLI of 21 y].

(8) **Impotence And Sterility:**

(i) Males - Before puberty, a boy is sterile, but not impotent.

(ii) Females - become sterile after menopause [approx 45 y]

(9) **Kidnapping:**

(i) Kidnapping from lawful guardianship — Taking away a boy <16y or a girl <18y or person of unsound mind away from a guardian without his consent [**S.361, IPC**]

(ii) Abduction — Taking away a person by force or deceitful means [**S.362, IPC**].

(iii) Punishment for kidnapping — 7 y + fine consent [**S.363, IPC**].

(10) **Evidence** - Persons of all ages are competent to testify as witnesses [**S.118, IEA**][**ch 1**].

(11) **Judicial Punishment:**

(i) "**Juvenile**" or "**child**" - means a person who has not completed 18th year of age [**S.2(k)** The Juvenile Justice (Care and Protection of Children) Act 2000, or JJA 2000]

(ii) **Juvenile in conflict with law** - means a juvenile who is alleged to have committed an offence and was <18 y as on the date of commission of such offence [**S.2(l), JJA 2000**]

(iii) **Punishments that *cannot be given* to a juvenile who has committed an offence** - (a) death (b) life imprisonment (c) imprisonment in default of payment of fine or in default of furnishing security [**S.16, JJA 2000**]

(iv) **If juvenile is between 16-18 y** - and JJB thinks that offence committed by him is so serious or his conduct and behaviour is such that it would not be in his interest or in the interest of other juveniles in a special home to send him to such special home, it may order the State Govt to keep such juvenile in a safe place [**S.16, JJA 2000**]

(v) **Punishments that *can be given* to a juvenile who has committed an offence** - (a) allow the juvenile to go home after advice, admonition and counselling. Parent or guardian is also counselled. (b) direct the juvenile to participate in group counselling and similar activities; (c) order the juvenile to perform community service; (d) order the parent of the juvenile or the juvenile himself to pay a fine, if he is over fourteen years of age and earns money; (e) direct the juvenile to be released on probation of good conduct. (f) Release the juvenile on probation of good conduct and place him under the care of any fit institution for up to 3 y. (g) send him to special home for up to 3 y [**S.15, JJA 2000**]

(vi) **Borstal school** - Please see below.

(12) **Identification** – Approximate age is important in all identifications, eg when a newborn is lost, it cannot be replaced by an older child, say of 2-3 months.

(13) **Marriage Contract** – age of marriage of female is 18 y and of males 21 y

[**ch 23**].

(14) **Miscellaneous** – use of children in pornographic materials is punishable.

4. Various ages of medicolegal importance

a. 12 y

(1) **Crime committed by a child between 7-12 years of age** - is not an offence, if he hasn't attained sufficient maturity of understanding [**S.83, IPC**]. Judge decides the issue.

(2) **General physical examination** – Age of consent is 12 y [**S.89, IPC**].

(3) **Consent** – intended by any section of IPC is not a valid consent, if age of child is <12 y [**S.90, IPC**]

(4) Indian Oaths Amendment Act 1939 - Unsworn evidence of a child <12 y is admissible if the court thinks he does not understand the nature of an oath.

(5) Bombay Shops and Establishments Act 1948 - Child <12 y cannot be employed.

(6) Exposure or abandonment of a child < 12 y by parents or person having care of it - 7 years and/or fine [**S.317, IPC]**

(7) Man rapes his own wife of <12 y - Punishment as if he raped any other girl of <12 y. [10 y-life]. If age of wife is 12-15 y - punishment of 2 y [maximum] or fine or both [**S.376(1), IPC]**. This clause may appear paradoxical as the minimum legal age for marriage in India is 18 years for girls. But despite this law, many marriages of young girls are going on in India [for details, please see **ch 23**].

b. 16 y

(1) **Child passports** - In many countries, different rules apply for a child <16 y for getting passport.

(2) **Age of voting** - in Austria [Since **1st July 2007**. It became the first member of the European Union, and the first of the world's leading democracies, to adopt a voting age of 16 for all purposes], Brazil, Cuba, Ecuador, Nicaragua.

(3) **Juvenile is between 16-18 y of age**"has committed an offence so serious in nature or his conduct and behaviour have been such that it is neither in his interest nor in interest of other juveniles to keep him in a special home"the Board may order the juvenile to be kept in such safe place that it thinks fit and

report the case to the State Government [**S.16** Juvenile Justice Act 2000].

(4) **Kidnapping of boy**– Taking away boy <16 y [and girl <18 y, or any person of unsound mind] from lawful guardianship without the consent of such guardian [**S.361, IPC]**

(5) **Selling intoxicating drugs** - A licensed vendor is not permitted to sell any spirit or intoxicating drug to persons <16 y [**S.22**, United Provinces Excise Act, 1910].

(6) **Childhood**, **Minority:** (i) **Child** - (a) means a boy or girl <16 y. If child commits an offence, he is called a **youthful offender**, and cannot be sentenced to death [The Bombay Children Act 1948]. (b) means a person <16y [**S.2(aa)** The Immoral Traffic (Prevention) Act, 1956]. (ii) **Minor** - (a) means any person <16 y [Bombay Prevention of Hindu Bigamous Marriage Act 1946]. (b) means a person 16-18 y [**S2(cb)** The Immoral Traffic (Prevention) Act, 1956].

(7) **Kidnapping or maiming a minor boy for the purpose of begging** - If merely kidnapped [10 y+fine]. If also maimed [life+ fine]. For the purposes of this section a minor is a male <16 y (or a female <18y). Imp because ages for minors are different for males and females [**S.363A, IPC]**.

c. 18 y

(1) **Age of marriage** - for girls [(i) **S.5(iii)**, Hindu Marriage Act 1955; (ii) **S.4(c)**, Special Marriage Act, 1954 (**ch 23)**].

(2) **Kidnapping of girl**- Whoever takes or entices any girl <18 y [or boy <16 y, or any person of unsound mind] out of the keeping of the lawful guardian without his consent fine [**S.361, IPC]**

(3) **Adulthood**, **Majority** etc:

(i) Adult - Person >18 y is an adult [**S.2(a)** The Factories Act, 1948; **S.2(1) (b)** The Mines Act, 1952]

(ii) Child – A female <18 y, and a male <21 y [**S.2(a)** The Prohibition of Child Marriage Act, 2006]

(iii) Juvenile - (a) Person <18 y is a “juvenile” or “child” [**S.2(k)** JJA 2000]. (b) If juvenile is accused of committing an offence and an enquiry is going on he must be kept in “Observation homes” [**S.8** JJA] (c) If after enquiry, juvenile is proved to have committed an offence, he is called a “juvenile in conflict with law”. Irrespective of what his current age is, if at the time of

commission of offence, he was <18 y he is “juvenile in conflict with law” [**S.2(l),** JJA]. He must be kept in “special homes” instead of jails [**S.9,** JJA]. He may be kept in **special home** for maximum of 3 y [**S.15(1) (g),** JJA] (d) Juvenile cannot be sentenced to death or life imprisonment, or committed to prison in default of payment of fine or in default of furnishing security [**S.16(1),** JJA].

(iv) Major - (a) A person attains majority when he/she attains the age of 18 years [**S.3**, Indian Majority Act, 1875]. If person is under guardianship, the age of majority is 21 y [please see below]. (b) Major means a person >18 y [S.2(ca) The Immoral Traffic (Prevention) Act, 1956]. Person between 16-18 y is a minor [S.2(cb) of same Act].

(v) Minor – is a person <18 y [(I)**S.4(a)** The Hindu Minority and Guardianship Act 1956 (II)**S.2(hb)** The Transplantation of Human Organs and Tissues Act, 1994].

(vi) Young person - person <18 y [**S.2(d)** The Factories Act, 1948].

(4) **S.87, IPC** -A person <18 y cannot give valid consent to suffer any harm which may result from an act not intended or not known to cause death or grievous hurt [example: boxing, cricket, fencing]. 17 y old boy [i.e.<18y] asks an older boy to do boxing with him "Older boy explains it could injure him"younger boy says, it doesn’t matter"They start boxing"Younger boy receives fatal injury"dies"Older boy is responsible. He cannot take plea that younger boy had consented. If however the younger boy was >18 y, the older boy could take the plea of consent.

(5) Age of consent for **undergoing surgery** [please see MLI of age above].

(6) **Procurement of girl <18 y for illicit intercourse** – Whoever procures such girl is punishable [10 y+fine; **S.366A, IPC]**

(7) **Selling person <18 y for prostitution** - Whoever sells, lets to hire, or otherwise disposes of any person <18 y for the purposes of prostitution or illicit intercourse with any person or for any unlawful or immoral purpose is punishable [10 y+fine; **S.372, IPC]**

(8) **Buying or hiring person <18 y for prostitution** - Whoever buys, hires, or otherwise obtains possession of any person <18 y for the purposes of prostitution or illicit intercourse with any person or for any unlawful or immoral purpose is punishable [10 y+fine; **S.373, IPC]**

(9) **Abetment of suicide of child <18 y** - Death + fine [**S.305, IPC]**

(10) **Culpable homicide** -is not murder if the person who died was >18 y and he risked death with his own consent [**S.300, IPC, exception 5]**

(11) **Children's home** - A child [Under Children's Act 1960] is sent to children's home on commission of an offence, but is not retained there beyond the age of 18 years.

(12) **Children and Young Person's Act 1933 of England** - A person <18 y can't be sentenced to death.

(13) **Kidnapping or maiming of a minor girl for the purpose of begging** - If merely kidnapped [10 y+fine]. If also maimed [life+ fine]. For the purposes of this section a minor is a female <18 y (or a male <16 y). [**S.363A, IPC]**

(14) **Age for voting** - in India and most other countries [Australia, UK, USA]

(15) **Statutory rape** - Sexual intercourse with a woman <18 y even with consent is rape [S.375, IPC].

(16) Age of consent for MTP [S.34. (a) MTP Act 1971].

(17) **Divorce by a girl between 15-18 y** - A wife may divorce her husband without any ground if her marriage (whether consummated or not) was solemnized when she was <15 y, and she repudiated the marriage after attaining that age but before the age of 18 y [S.13(2)(iv) The Hindu Marriage Act 1955].

(18) **Donation of organs** - (a) Age of giving consent for organ donation (b) A "donor" is a person>18 y who voluntarily authorizes removal of any of his human organ or tissue or both for therapeutic purposes. [S.2(f) **The Transplantation of Human Organs and Tissues Act, 1994**].

E. Hair [Forensic Trichology]

Study of hair is known as ***trichology***, and its application for administration of law and justice, ***forensic trichology***.

Salient features:

(1) **Composition** - Human hair is composed of proteins [<65-90%], water [<15-35%], and lipids [<1-9%].

(2) **Growth:** (i) Rate - Hair grows at the rate of 0.4mm/day [1cm/month]. Nails at the rate of 0.1mm/day.

Memory Aid 34: Growth phases of hair

ACT

1. **A**nagen phase: **2–3 years** (occasionally much longer)

2. **C**atagen phase: **2–3 weeks**

3. **T**elogen phase: **2–3 months**

1. Structure of hair

a. Longitudinal section

The structure of hair is quite like that of a pencil.

(1) It consists of a shaft

(2) and root

(3) ending in a bulb.

b. Transverse (cross) section

(1) **Cuticle:**

(i) This is the outermost layer [**Fig 3.26**].

(ii) consists of - (a) thin, non pigmented scales [flat overlapping cells arranged in a root-to-tip direction]. (b) Each cuticle cell [scale] is 5–10 µm long and 0.3–0.5 µm thick. (c) The cuticle layer in human hair is approximately 5–10 scales thick.

(iii) Function – to protect the cortex

(iv) Scales [cuticular cells] - are broadly divided into two types - (a) Coronal – when each scale encircles the shaft completely and (b) Imbricate, when the scales do not encircle the entire shaft. Each type is further divided into several subtypes.

(v) Examination of cuticular scales – An impression from the surface of hair is made in a thin film of cellulose acetate or finger nail varnish. A drop of sol^{n} is spread thinly on a glass slide. Hair is cleansed in alcohol and is laid over the wet film. After the film has dried [in about 10 m], the hair is stripped off the slide quickly and the scale impressions are examined microscopically.

(2) **Cortex:**

(i) Middle layer.

(ii) Consists of longitudinally arranged elongated cells without nuclei.

(iii) Within these cells, fibrils are present

(iv) granules of pigment lie over the fibrils

(3) **Medulla:**

(i) innermost layer

(ii) Consists of keratinized remains of cells and air sacs.

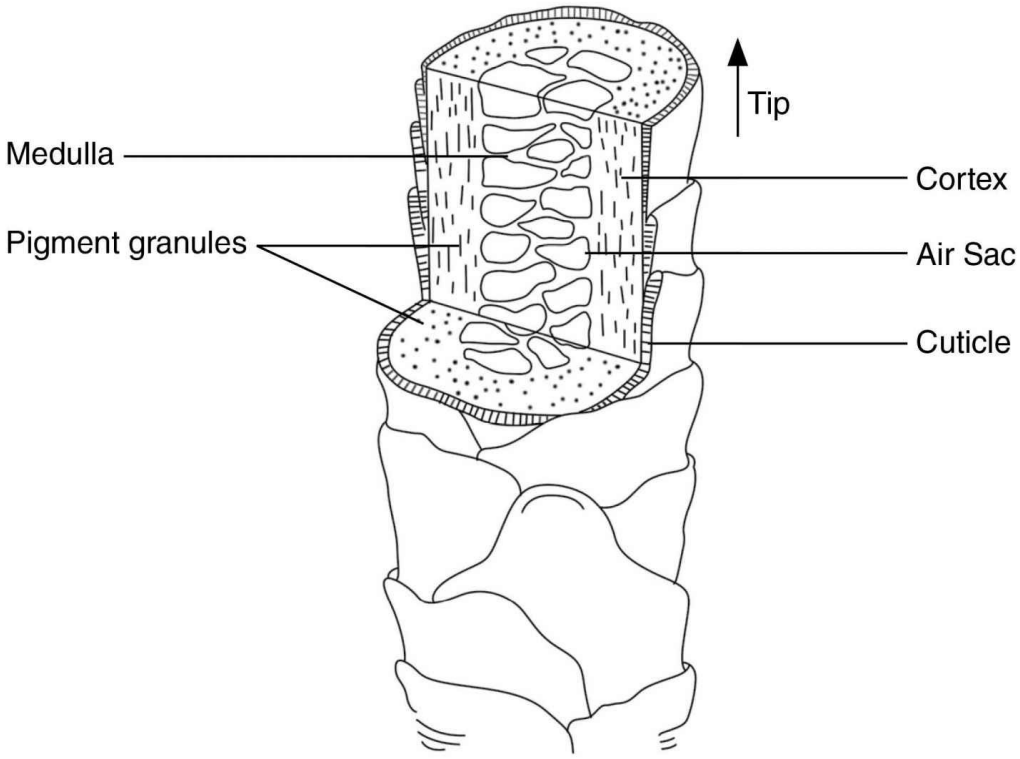


Fig. 3.26: Diagrammatic structure of a typical hair shaft. Shows both longitudinal and transverse sections

2. Information from hair

Hair may be examined to reveal following information:

a. Hair or some fibre

Structure - Look under the microscope for structure. (I) Hair - Hair structure would be unique as illustrated above. (II) Textile fibers - are divided into two categories: natural or synthetic. Natural fibres are cotton, jute, linen, silk and wool. Synthetic fibres include acetate, acrylic, aramid, nylon, polyester, and rayon. Each fibre will show its own unique structure [**Fig 3.27**]. Characteristics such as relative thickness of cell walls and lumen, cell length, and the presence, type, and distribution of dislocations are important in identification of all natural fibres.

(1) **Coir:** (i) Comes from the husk of coconut. (ii) Very dense and stiff fibre. (iii) Dark brown or opaque (iv) chemical tests for lignin - Can be used for all plant

derived fibres.

(2) **Cotton:** (i) These are fibers from the cotton plant's seed pod (ii) Solubility – Insoluble in alkali. Soluble in 75% Sulphuric acid. (iii) Most commonly encountered natural fiber (iv) The cross section of the fiber is bean-shaped, swelling almost round when moisture absorption takes place. (v) Each cotton fiber is composed of concentric layers (vi) Fibres show double refraction when observed in polarized light.

(3) **Jute:** (i) Comes from flax just like linen but the plants are processed slightly differently (ii) Jute fibres are smooth without transverse lines (iii) The cell cavity is not uniform (iv) The ends are blunt.

(4) **Linen:** (i) Comes from flax, a bast fiber taken from the stalk of the plant (ii) Has a narrow lumen (iii) Fibres show cross lines or folds about which the fibre is often swollen.

(5) **Silk:** (i) These are fibers from the cocoon of the silkworm (ii) Shows crossover marks and a triangular cross section with rounded corners. (iii) Consists of long clear threads without any cells (iv) Smooth and finely striated.

(6) **Wool:** (i) These are fibers from animal coats: Sheep, goats, rabbits etc (ii) Show an outer layer of flattened cells with overlapping margins (iii) Interior is composed of fibrous tissue.

(7) **Synthetic fibres** – Differentiated by noting (i) their solubility in solvents like HCl, HNO_3, acetone, and dimethyl sulfoxide. (ii) Physical characteristics like density, refractive index, melting/softening point (iii) infrared spectroscopy. For more on fibres, please see ch 30 – Forensic Science Laboratory.

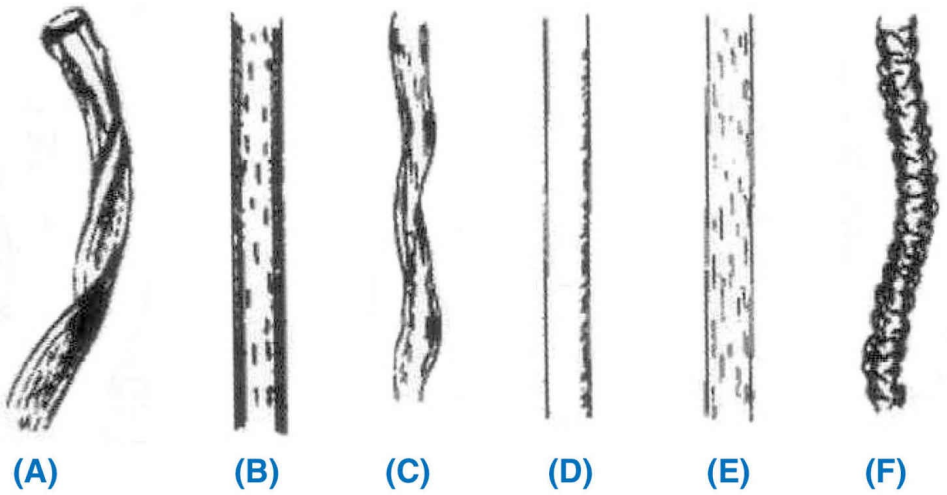


Fig. 3.27: Fibres as seen under the microscope. **(A)** Cotton **(B)** Dacron **(C)** Natural silk **(D)** Nylon **(E)** Viscose **(F)** Wool

b. Human or animal

(1) **Issue of human or animal hair arises** in

(i) Killing of an animal protected under the Wildlife Protection Act, 1972

(ii) Bestiality. It can be differentiated from human hair [**Table 29. Fig 3.28**].

(2) **Medullary index:**

(i) Medullary index =

$$\frac{\text{Diameter of medulla}}{\text{Diameter of whole hair shaft}}$$

(ii) **Value** – Humans<0.3; Animals>0.5 [**Table 29**]. Value differs in hair from different parts of the body; thus sometimes useful to know the part of the body, hair is derived from.

c. From what part of body derived?

(1) **Medullary index** – please see above

(2) **Morphology** [**Table 30**]:

(i) Head – long, soft, taper gradually from root to tip. Often traces of oil found smeared on them

(ii) Beard and moustache – thicker than the hair from any other part of body. Traces of shaving cream, or aftershave may be found on surface

(iii) Eyebrows, eyelashes – stiff, thick and taper to a point. In females, traces of

eye cosmetics may be found smeared on them.

(iv) Nostrils – stiff, thick. May show nasal mucus smeared on them.

(v) Beard, axilla, chest, arms, legs and pubis - **Table 30**.

Table 29: Differences between human and animal hair

S.No	Criteria	Human hair	Animal hair
1.	**General appearance**	Fine and thin	Coarse and thick
2.	**Cuticle and cuticular scales**	Cuticular scales are (1) short and thin (2) irregularly annular	Cuticular scales are (1) very large (2) have step like or wavy projections
3.	**Cortex**	(1) Thick (2) 4-10 times as broad as medulla	(1) Thin (2) 2 times as broad as medulla
4.	**Medulla**	(1) Narrow (2) discontinuous, fragmented [sometimes even absent]	(1) Wide (2) Continuous
5.	**Medullary index**	<0.3	>0.5
6.	**Pigment**	Evenly distributed	Mostly concentrated towards medulla.
7.	**Banding**	No banding seen	Alternate dark and light bands are seen along the length of hair in some animals. Measurement of the length of bands can help in identification of species.
8.	**Precipitin test**	Specific for humans	Specific for animal concerned

Table 30: Human hair characteristics from various body parts

S.No	Body part	Length	Shaft diameter	Tip	Medulla	Cross-section
1.	Scalp	1-100 cm	20-130 µ. Less variation along length	Tapered	Different types	Circular, elliptical or oval
2.	Eyebrows and eyelashes	<1 cm	Sharp change along length	Pointed	Broad	Circular
3.	Nostrils	<1 cm	Sharp change along length	Pointed	Variable	triangular
4.	Beard	5-30 cm	Broad	Blunt; sharply cut	Discontinuous	Triangular
5.	Axilla	1-5 cm	Broad	Round or frayed	Discontinuous	Circular
6.	Chest	>3 cm	Appreciable variation along length	Fine	Different types	Circular
7.	Arm and leg	<1 cm	Sharp change along length	Fine	Complex	Circular
8.	Pubis	1-5 cm	Curly, kinky, large variation along length	Round or frayed	Broad	Irregular

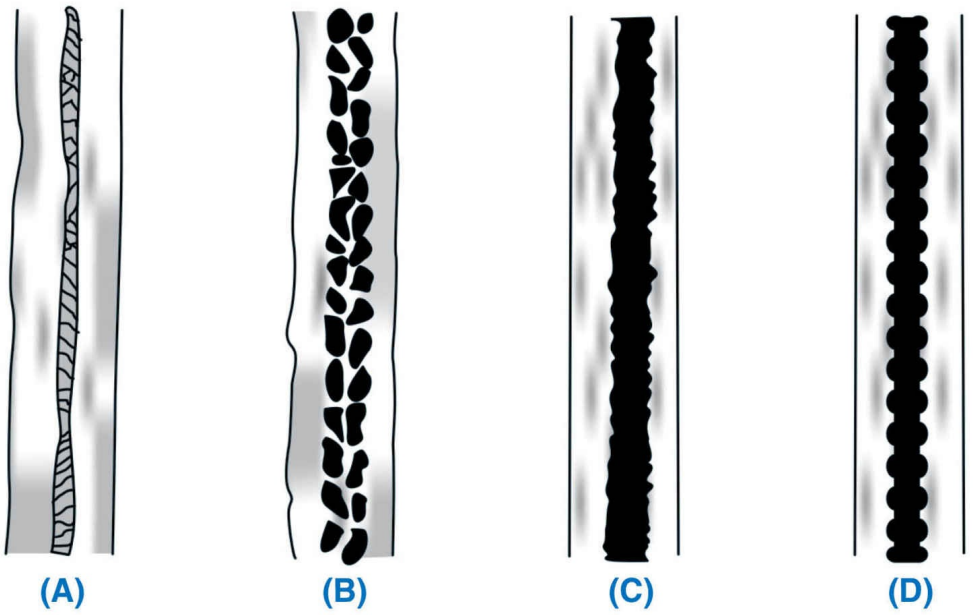


Fig. 3.28: Hair as seen under the microscope – **(A)** Human **(B)** Dog **(C)** Goat **(D)** Sheep.

d. How much violence was used?

(1) **Force used** - Considerable force is required to pluck out a thick bunch of healthy growing hair from the scalp

(2) **Tearing of scalp** – An adult may even be lifted or dragged by the hair and yet they may not come off. So strongly are they attached that sometimes even scalp would be torn from the skull, but not the hair.

e. Sex

(1) **Male hair** –Coarser, Darker, Thicker

(2) **If hair follicles present:**

(i) Barr bodies [in females]

(ii) From DNA profile [Amelogenin]

(3) **Hair diameter** - Significant differences between sexes. Please also see **Table 6** above.

f. Age

(1) **Scalp hair** – With -ing age, there is (i) graying and thinning; (ii) growth is slower. (iii) -ed loss.

(2) **Facial hair** – -in women at about menopause

(3) **Body hair:**

(i) Human fetus and newly born child - show lanugo hair [fine, non medullated, non pigmented (colorless), soft, scale pattern simple]

(ii) Adults – coarse, medullated, pigmented, tougher, more complex scale pattern

(4) **Axillary and pubic hair:**

(i) Initially soft and fine.

(ii) Later become coarse, pigmented and curly.

(iii) In women, there is loss of axillary hair at menopause. Please also see "secondary sexual characters" above for ages of appearance.

(5) **Pigmentation** – Initially -es with age; afterwards hair start losing pigmentation.

(6) **Number of hair follicles** - .with age. There is an associated -in the proportion of telogen hair follicles.

(7) **Hair diameter** – has been measured with Atomic Force Microscopy [AFM] and has been found to -for the first 40 y of life, after which it begins to .. Thickness of scalp hair varies with age.

(8) **Scalp hair density** –-es up to 40 y. Then .es with age.

(9) **Cuticles** - of older people

(i) Show more damage. Damage is similar to that of chemo-mechanically damaged hair fibers.

(ii) Show many cracks and miscellaneous cuticular debris. Because of this, hair become brittle with age.

(iii) Scale edges - show uneven saw-tooth appearances.

(iv) Scale edge ghosts and endocuticles - more frequently seen

(v) Surface roughness -significantly -ed.

(vi) Stiffness - between the hair and the tip altered.

10. Hair roots – from children dissolve rapidly in KOH sol^{n}, but not in older people.

g. Has the hair been altered by bleaching, dyeing or disease?

(1) **Bleached hair:** (i) brittle (ii) dry and (iii) straw yellow

(2) **Dyed hair:** (i) Color is not uniform (ii) roots are of different color (iii) hair is brittle, lusterless and rough (iv) scalp skin is also colored (v) Color of head hair different from hair on other parts.

h. Blood groups from hair

Please see **ch 29**.

i. Is the hair identical with the hair of suspect or victim?

Comparison of (1) color (2) length (3) scale pattern (4) **DNA analysis** [**ch 29**] if root is available.

j. Dit it fall naturally or was it forcibly removed?

Examine base – see if root is visible

(1) If forcibly pulled out – hair bulb is larger and irregular. Sheath is ruptured [**Fig 3.29**].

(2) If fallen naturally – hair bulb is smaller, atrophied and distorted. Root sheath is absent.

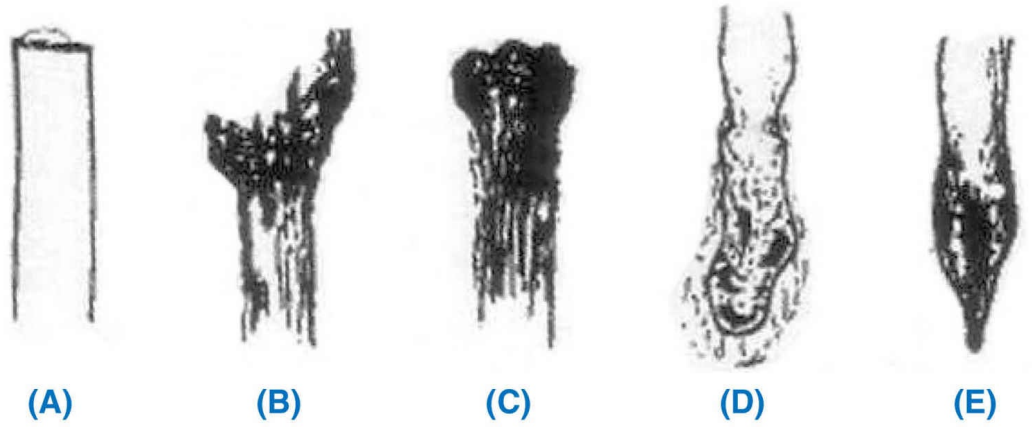


Fig. 3.29: Examination of hair can reveal weapon etc. End of human hair **(A)** cut by a sharp weapon; **(B)** Severed by a blunt weapon; **(C)** Singed; **(D)** Root of pulled out human hair **(E)** Root of naturally fallen human hair

k. Trace elements in hair

Trace elements in hair [eg P,S] are in different conc in different individuals, depending on their race, age, sex, environment, diet etc. These can be estimated by Neutron Activation Analysis [NAA] to establish identity [please see Gaetane Bouchard murder case below under “case studies”].

l. Hair and DNA

(1) Both nDNA and mtDNA analyses [**ch 29**] can be applied to hair samples.
(2) **Plucked hairs** - usually have live follicle cells that can be used for nDNA sequence analysis. Cut hair or hair lost naturally during the telogen [quiescent] phase do not have live cells associated with the sample.
(3) **Cut or shed hair** - mtDNA sequencing methods can be used to associate or distinguish hair samples from different individuals.

m. What is the cause of injury?

(1) **Injury can be:**
(i) Mechanical - (a) Broken (b) Crushed (c) Frayed (d) Glass cut (e) Tangled (f) Twisted
(ii) Burnt
(iii) Chemicals [corrosives etc]
(2) **Examine tip:**
(i) Tip pointed and non-medullated"normal uncut hair
(ii) Shows sharp ends"(a) indicates cut by a sharp weapon [**Fig 3.29**] (b) Time of inj - (I) Recently cut – sharply cut edge with a projecting cuticle (II) After a week – End becomes square, smooth (III) After 1-2 months – Round, blunt (IV) After 3-4 months – end becomes elongated, but medulla is absent.
(iii) Crushed ends"Heavy blunt weapon. Unprotected cortex splits and frays
(iv) Ragged ends"indicate frequently brushed hair (v) singed"Death by fire.

3. Medicolegal importance of hair

Each of the above questions answered has an MLI. In addition following is the MLI of hair

(1) **Age** – of a person can be determined by distribution of hair on his body.
(2) **Sex** – can be determined by
(i) Barr bodies
(ii) Body distribution
(iii) Texture
(3) **Identification** – especially when some known hair peculiarity is known, eg wavy hair, bleached, colored and dyed hair etc, very long hair.
(4) **Injuries on hair, hair tip and hair bulb** – can help in determination of nature of weapon.
(i) Blunt and sharp weapons produce different kinds of injuries.

(ii) Fire and firearms can produce singeing of hair tips.

(iii) Scalds – hair are not singed in scalds; thus burning and scalding can be differentiated.

(5) **Trace evidence** [**ch 30**] – Remains identifiable on body, clothes, weapons etc for a long time; thus identifiable even in crimes committed long before. It may be the only link between the accused and his crime and weapon.

(i) Sexual offences – bestiality, rape, sodomy [hair of either partner found on the other]

(ii) Vehicular accidents – vehicles causing injuries on victims may be identifiable long time after by hair adhering to them [**ch 18**].

(6) **Time of death** – length of hair on face.

(7) **Toxicology from hair** - Please see ch 31"Diagnosis of poisoning"Hair analysis for poisons. Also ch 36"s Arsenic.

F. Deformities, Diseases, Moles, Birthmarks

[A] **Deformities** and **diseases** - eg amputations, arthritic changes, asbestosis, bilobed ear, bowed legs, cleft palate, calcified leiomyomata, club foot (talipes), dental implants, dentures, hare lip, horseshoe kidneys, nail deformities, old fractures, poliomyelitic leg, polydactyly, prosthesis (implanted valves, orthopedic nails, pins and screws, pacemaker, plates in skull), silicosis, skull deformities [please see above under “skull”], souvenir bullets, stones in gall bladder and kidneys, spine curvature, surgically removed organs (appendix, gallbladder, kidneys, ovaries, prostate, tonsils and uterus), surgical scars, undescended testicles and web fingers or toes etc can help identification in highly putrefied bodies. [B] ***Mole***, also known as a ***nevus***, is a pigmented pin-point spot over the skin. (i) **Color -** varies from gray to brown or jet black. (ii) **Frequency** - Very common. An average person has over 20 pigmented moles (iii) **Situation** - usually situated on the face, neck or back, but they may occur anywhere (iv) **Hairs** - may be hairy or non-hairy (v) **congenital** condition, but many pigmented moles do not appear until adult life. [C] ***Birthmarks*** are blemishes on the skin present in a baby since birth or formed soon after. Birthmarks may be (i) **vascular** (eg Port-wine stains) or (ii) **pigmented** (eg Café-au-lait spots). **Port-wine stains** are flat, pink, red or purple marks that appear at birth, often on the face, arms, and legs, and continue to grow as the child grows. ***Café-au-lait* spots** are flat spots having the color of coffee with

milk. Many birthmarks disappear as baby grows, and are thus not useful as identification features [eg macular stains, strawberry marks (capillary hemangioma) and Mongolian spots].

Fig. 3.30: A large café-au-lait spot. Please note its brown color.

G. Scars

A ***scar*** is a fibrous tissue covered by epithelium devoid of hair follicles, elastic tissue, sebaceous and sweat glands and pigment; they are usually produced after the healing of a wound involving the dermis.

Salient features:

(1) If the wound involves only the epidermis [as in abrasions], it will regenerate without the formation of a scar.

(2) **Permanency** - Scars once formed are **permanent**. That is why they serve as good identification features.

1. Characteristics

(1) All scars are usually depressed, but may be raised above the surface if keloid formation has occurred (occurs especially in Negroes)
(2) **An incised wound** - leaves a linear scar
(3) **A lacerated wound** and a suppurating wound leave a prominent, irregular, firm scar.
(4) A **bullet wound** causes a circular depressed scar.
(5) **Burns** leave an irregular scar.
(6) **Scars from scalds** are spotted in appearance. They show splashing marks as well as signs of dribbling (flowing down).
(7) **Stab wounds** may leave different shaped scars, depending upon the shape of the cross-section of the knife. May be oval, elliptical, triangular or sometimes irregular.
(8) **Corrosive acids** leave an irregular scar. There may be signs of splashing and dribbling.
(9) **Vaccination scars** are circular or oval, flat or slightly depressed. They are not good identification features because they are likely to be present on several people in almost the same location.
(10) **Diseases like smallpox and syphilis** may leave characteristic irregular scar marks over the body.

2. Classification

(1) Accidental scars – eg from burns, cuts, injury to eyes, loss of limbs, scalds etc
(2) due to disease – eg smallpox
(3) surgical scars – eg incisional scars [**Fig 3.31**].

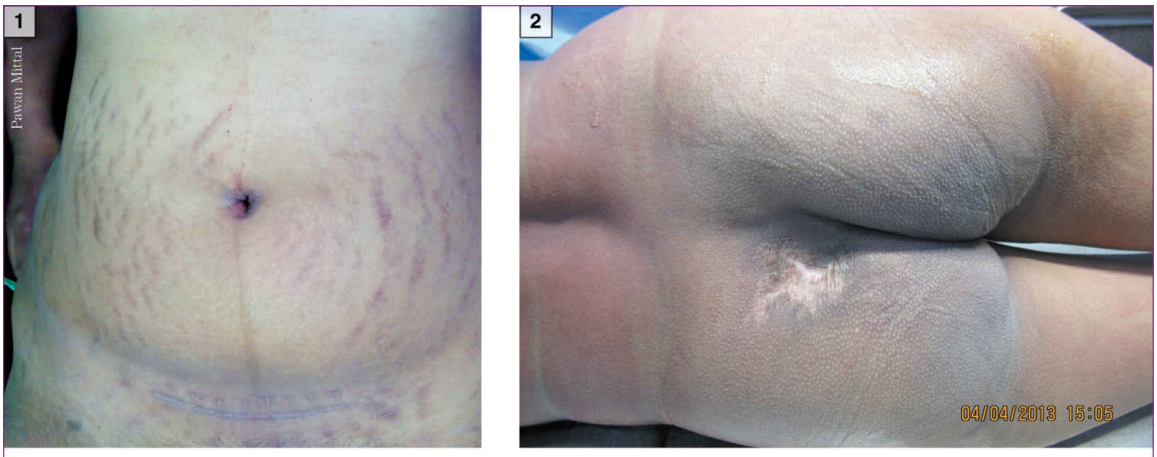


Fig. 3.31: **(1)** LSCS scar due to a previous cesarian section. It makes a good identification feature. Striae gravidarum and linear nigra are also visible, which act as additional identification features. **(2)** Scars at unusual positions, such as here on left buttock form a very good identification feature in the dead. In the living, scars for identification should only be recorded on exposed areas, as the person may have to be identified in an open court.

3. Growth

Scars produced in childhood grow in size as child grows, especially if situated on chest or limbs.

4. Age of scars

Scar forms on by secondary intention in about 2 weeks. After this period, the age of scar is calculated as follows

(1) **Up to 2 months** – Vascular; angry red

(2) **2-6 months** – Soft, sensitive and tender

(3) **After 6 months** – White, firm, tough, hard, glistening, corrugated.

Memory Aid 35: Age of scars

VAST

VAscular : up to 2 m

Soft, **S**ensitive, tender : 2-6 m

Tough : >6m

5. Examination

(1) **Good lighting** and **magnifying glass** – Essential (because old scars may often be very faint and unrecognizable).

(2) **Description** - should include, their number, size, shape, location, nature of surface (whether smooth or irregular), whether fixed or free, its level in relation to the body surface (whether raised above the surface or depressed),

whether glistening or not, and its tenderness.

(3) **Ends** - rounded or tapering (can often help determine the nature of the weapon used to inflict the wounds)

(4) **Probable direction** – of the original wound (note tailing).

(5) **If the scar is faint** - can be made more visible by the application of heat, UV light or surface friction.

(6) **Demonstration of scar in a dead body** – can be done by microscopy (scars lack elastic tissue). Elastic tissue is however present in striae gravidarum.

6. Erasure

Scars can be erased by

(1) Skin grafting and

(2) Excision with suturing of edges of excised area. This results in another scar, but which is less prominent than the earlier one.

7. MLI

(1) Identification

(2) Shape of scar – Can reveal nature of offending weapon

(3) Age of scar – if it corresponds to the date of attack, it is a good corroboratory evidence

(4) Linea albicantes – indicate previous pregnancy.

H. Tattoos

Tattoos (Tahitian tatau, a mark) are special marks, designs, pictorial diagrams (e.g. of Gods and Goddesses), or alphabetical messages (e.g. name of self, husband, or a friend etc) made or written permanently on the skin of the body. Tattooing has been practiced since ancient times. The body of "Ötzi the Iceman", dated 3300 BC, bears 57 tattoos. Several Egyptian mummies have been found to have tattoos.

Salient features:

(1) **Site** - Tattoos can be found on just about any part of the body, though the commonest site is front of forearm. Other common sites are upper and lower limb, back of neck, abdomen, breasts and vulva (prostitutes), penis (homosexuals), buttocks. Even facial tattoos are known.

(2) **Method of examination** – Record exact size, shape, design, color, site.

Photography is better.

Fig. 3.32 **(1)** Common tattoo used in India, with names of deities. Pl note that it can often reveal religion. **(2)** Tattoo on forehead, a rather uncommon site.

1. Technique

Coloring matter (dye) is injected deep in the dermis with sharp needles, or an electric vibrator, so that the mark becomes indelible. An inaccurate technique would deposit the pigment in superficial layers of dermis only, which would cause obliteration of the tattoo mark eventually.

2. Dyes

(1) Henna and Mehndi in India.

(2) **Salts of heavy metals** - Aluminum (green, violet); barium (white); cadmium (red, orange, yellow); chromium (green); cobalt (blue); copper (blue, green); iron (brown, red, black); lead (yellow, green, white); mercury (red); nickel (black); titanium (white), ultramarine [double silicate of aluminum and sodium; blue] and zinc (yellow, white). **Metal oxides** used are ferrocyanide and ferricyanide (yellow, red, green, blue).

(3) **Organic chemicals** - Azo-chemicals (orange, brown, yellow, green, violet) and naphtha-derived chemicals (red).

(4) **Homemade or traditional tattoo inks** - Made from pen ink, soot, dirt or blood.

(5) **UV (Blacklight) Reactive Tattoo Ink** - Blacklight tattoos will not glow in the dark but glow under UV light. These work on the principle of fluorescence; the inks are made of florescent material.

3. Permanency

(1) Once imprinted, tattoos remain on the body **almost indefinitely**, until and unless they are removed by specialized procedures.

(2) **Rate** at which the tattoos fade depends upon (a)**composition** of dye (b) **depth** up to which the dye is inserted - Dye should be injected to the right depth. Dye injected superficially gradually fades in some years. Dye injected too deep is removed by phagocytes (c) **Site:**

(i) Parts protected by clothing retain tattoos much longer than those which are exposed to sunlight

(ii) Tattoos on hand disappear early due to constant friction

4. Faded tattoos

Faded tattoos may be made visible by

(1) UV lamp

(2) Infrared photography (Can also reveal old tattoos superimposed by new tattoos) [please see ch 30, under the heading photography]

(3) Removal of epidermis:

(i) makes the tattoo very clearly visible on dermis.

(ii) In **decomposed** or **burnt bodies** – can be done easily.

(iii) In **fresh bodies** – Apply heat on suspected tattooed area (a) By placing a spirit soaked burning cotton over the area. (i) Examination of lymph nodes nearest to faded tattoo [eg in case of forearm tattoos, axillary LN] – Incise LN and note color"If an abnormal color [eg black, red] is noted, tattoo presence confirmed. Helpful also if the person had intentionally removed tattoos during life. The color should be mentioned in the pm report [please correlate with **ch 5**"description of an organ].

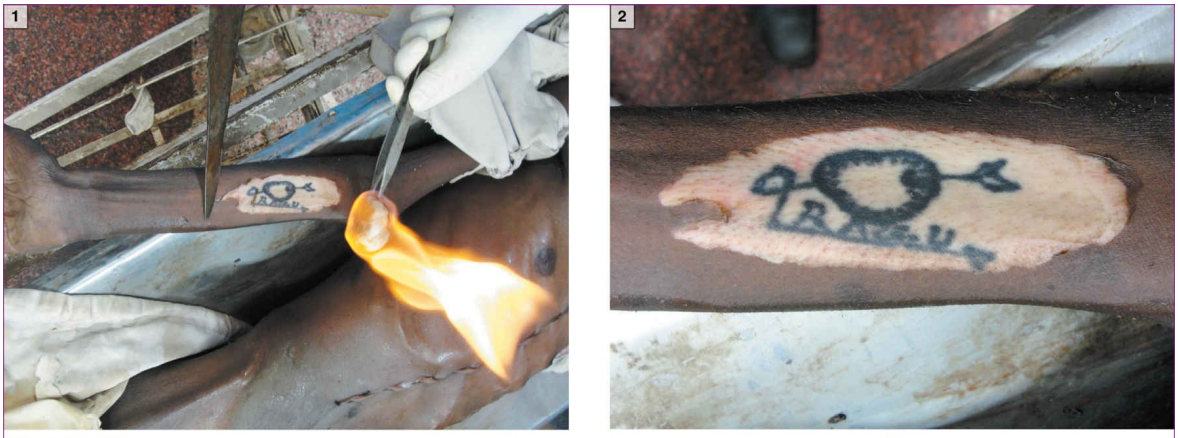


Fig. 3.33: (1) Heat applied by placing a spirit soaked burning cotton over the area can make the tattoo visible (2) Close up of tattoo made visible by this technique.

5. Complications

Abscess, AIDS, allergic reactions to tattoo inks, bruise (if bv is punctured), erysipelas, fungal infections, hepatitis, gangrene, leprosy, septic inflammation, syphilis, tetanus, tuberculosis may occur. A few cases of burns on black tattoos caused by MRI scans have been documented. Black ink commonly contains iron oxide; the MRI scanner causes the iron to heat up causing burns.

6. Removal

(1) A criminal often removes tattoos, lest he may be identified.
(2) Examination by UV, Infrared [or other methods described above] is helpful.
(3) Following methods are used.

a. Surgical removal

(1) **Excision** and **skin grafting**
(2) **Scarification** - scars are formed by cutting, branding or burning the skin.
(3) **Dermabrasion** - Surgical procedure involving controlled abrasion of the upper layers of the skin with mechanical means (eg sandpaper) or LASER (CO_2 or Erbium:YAG).

b. LASER

LASER treatment causes tattoo pigment particles to heat up and fragment into smaller pieces. These smaller pieces are then removed by phagocytosis [normally tattoo dye particles are too big to be removed by phagocytosis. That is the reason of their permanency].

c. Corrosives

Corrosives produce a scar causing disappearance of tattoo. Substances used are Formic acid, papain in glycerine, tannic acid and zinc chloride.

d. Cover-up

This is not true removal, but covering of an old tattoo with a new tattoo. Necessitates darker inks to effectively hide the older tattoo.

e. Miscellaneous

(1) Carbon dioxide snow
(2) Diseases (natural removal). Confluent Smallpox (in the smallpox era), chronic eczema
(3) Electrolysis.

7. ML importance [of tattoos]

a. Drug addicts, HIV transmission

(1) Drug addicts get the front of their arms tattooed to hide the needle puncture marks to mask their addiction habit from policemen [**ch 40**].
(2) If drug addict is HIV+ve, there is risk of HIV transmission if same needle is used.

b. Identification

Tattoos are an important aid to identification. If the number of tattoos are large, or if the shape of tattoo is very peculiar, identity can be made from tattoo alone. Other information that can be derived from tattoos is identity numerals [as in prisoners], initials and dates, name (own, spouse, friend) and regimental or nautical details.

Case studies:

Shark Arm Case – James Smith (resident of Sydney, Australia), former boxer and a small time criminal went missing on April 7, 1935. On 25 April, a human arm was regurgitated by a captive tiger shark kept in an aquarium; it had been captured shortly after April 7, the arm bore a tattoo mark showing “two men boxing”. His wife and brother identified it as the arm of James Smith as it had

exactly the same tattoo. Investigations led to the arrest of Patrick Brady, who had killed Smith and thrown the body in sea.

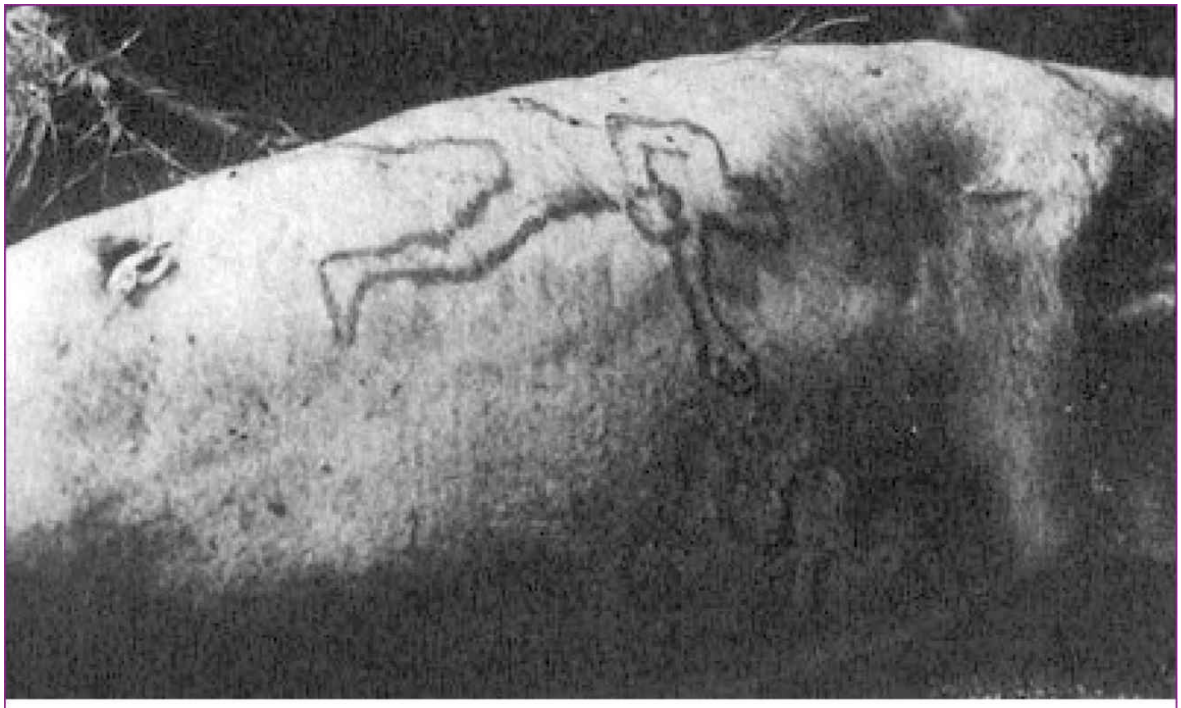


Fig. 3.34: Smith's arm regurgitated by Shark. Pl note tattoo with two men boxing. The second man is not clearly seen as it is hidden on the dark side.

c. Medical Tattoos

A ***medical tattoo*** is one used for medical reasons. It may be made

(1) to show illnesses or allergic reactions

(2) As a warning that a patient suffers from a chronic disease that can exacerbate suddenly and that will require immediate specialist treatment (eg congenital adrenal hyperplasia; patient may need steroid replacement therapy during ordinary illness).

(3) **As an aid in radiotherapy**. In order to minimize damage to surrounding tissues, the radiotherapist seeks to keep the irradiated field as small as possible. Marking a number of points on the body with tattoos can aid radiotherapists in adjusting the beam properly.

(4) During **breast reconstruction** after mastectomy or breast reduction surgery. Tattooing is sometimes used to replace the areola which has been removed during mastectomy.

d. Mental state

The presence of tattoos of various types tell about the mental state of the person, as well as his likes and dislikes. They can also tell about the past experiences of the person.

e. Occupation

Prostitutes, homosexuals, sailors, boxers etc may have peculiar tattoos at peculiar sites.

f. Protective tattoos

In many cultures tattoos are regarded as the protective amulets. Can reveal country of origin.

(1) **Burma:**

(i) Soldiers believe that tattoos on their thighs make them invincible in war.

(ii) The snake tribes of Pakokku believe these tattoos protect them from the vipers and cobras. Each member of this community goes under weekly tattooing. Black Ink mixed with snake venom is used for tattooing The entire body - arms, legs, chest, back, face and even the scalp - is tattooed [**venom tattoos**]. May be scientifically sound

(2) **India** – There is a popular belief among Hindus that a tattoo of a God like Lord Hanuman can protect them from all kinds of evils and pains.

(3) **Japan** - Women of Ainu group believe that if they get tattooed like a goddess, the evil spirits of diseases would run way from them.

g. Religion

The presence of tattoos of a particular God or Goddess tells about the religion of the person.

h. Miscellaneous

(1) May be an indicator of **social status**. Generally persons of high social status avoid tattoos.

(2) **Country** of origin

(3) **Perversions** – drawing of indecent figures

(4) May reflect **travel history**, **involvement in wars**, **sex interests** etc.

(5) May cause hypersensitivity reactions.

I. Occupation Marks

Occupational marks or ***stigmata*** are special marks on the body of a person, produced as a result of his profession or habits. These may sometimes be helpful in identifying a person. Sherlock Holmes often impressed his colleague Watson by telling the occupation of his clients simply by observing occupational marks. Occupational marks may be temporary or permanent.

J. Anthropometry (Bertillon system, Bertillonage)

Anthropometry (Gk anthropos, man; metron, measure" literally "measurement of man") refers to the measurement of ***eleven key dimensions*** of the individual for the purposes of identification.

Salient features:

(1) **Earliest** system of criminal identification.

(2) Started by **Alphonse Bertillon** (1853 –1914) in the year **1883**.

(3) **Principle** - Based on the two facts (i) Dimensions of the skeleton do not change after the age of 21 years. (ii) Quetelet's law [please see above under "Stature"]. According to **Quetelet's law**, there is one in four chance of two persons having exactly same height. Bertillon reasoned that if one more measurement was added - for example the length of the trunk - the chance of finding the both measurements exactly the same decreased to one in sixteen. By extending the number of measurements to eleven, the cumulative probability of two criminals having exactly the same measurements would be 4^{11} or over 4 million to one.

(4) The system included (i) **Descriptive data** – e.g. (a) color - of hair, eye and skin [complexion], (b) shape - of ears, nose and chin (ii) **Body marks** – e.g. tattoos, scars, moles (iii) **Body measurements** - 11 parameters were used by Bertillon. No individual has all 11 parameters exactly the same. The parameters used were: (1) Height: Standing (2) Height: Sitting (3) Reach: Arms outstretched (middle finger-tip of one hand to middle finger tip of other) (4) Length of head (5) Width of head (6) Length of right ear (7) Width of right ear (8) Length of left foot (9) Length of left forearm (10) Length of left middle finger (11) Length of left little finger (iv) **Front and side photographs**. Also called ***portrait parlé*** (Fr. The Speaking Portrait), because it was like making a

speaking portrait of a person.

(5) **Disadvantages** - (a) Measurement requires use of instruments and trained operators. Otherwise two operators may give different values for same person (b) applicable only to adults (c) Calculation errors are there (d) photographs are not reliable (e) Like fingerprinting, anthropometric data is not left at the scene of crime. Presence of criminal at the scene cannot be established by anthropometry

(6) **Current status** - This system has now been replaced by dactylography.

K. Dactylography

Dactylography **(Gk *daktylos*, finger; *graphein*, to write)** [**syn.** Fingerprinting, dermatoglyphics, Galton system] is a method of identification, based on unique epidermal ridge pattern on the tips of fingers.

Salient features:

(1) It is the ***most important, accurate*** and ***unfailing*** method of positive identification.

(2) It is a better method of identification than even DNA profiling. DNA profiling would be same in identical twins, but fingerprints are different even in identical twins.

(3) **Paternity by fingerprints** – Patterns are not inherited and paternity cannot be proved through fingerprint patterns.

(4) ***Epidermal ridges*** (also called ***friction ridges***) are raised portions of the epidermis on the fingers (also seen on toes, palms, soles). Their function is to amplify vibrations and to assist in gripping rough surfaces and wet surfaces. Sweat gland pores open on their top"Responsible for fingerprints. A subsidiary ridge [syn incipient, nascent or rudimentary ridge] is an incompletely developed ridge [thinner]. Sweat pores do not open over them; they do not produce fingerprints.

(5) **Embryology:** (i) 6-8 weeks after conception - Volar pads form. These are ball like structures that make up the contour of the fetal hand. (ii) 10-12 wks - volar pads begin to recede (iii) 13^{th} wk - Skin ridges appear (iv) Between 10-16 wks - local factors like amniotic fluid pressure influence fingerprint pattern. That is why fingerprints are different even in identical twins. (v) 21st wk – fingerprint pattern is complete.

(6) **Permanence:** (i) Fingerprints remain permanent throughout life and even

after death. (ii) Even in advanced decomposition, fingerprints can be identified.

(7) **Transmission of fingerprints** – Patterns can be easily emailed and printed, enabling intercontinental criminals to be caught easily.

1. Types of Fingerprints

a. According to fingerprint pattern

Four types are known [**Fig 3.35**]. Depend on presence of **core** and **delta**. A ridge recurving on itself is called the **core**. Convergence of ridges from three directions is called a **delta** (or **triradius**). **Rule**: If a pattern contains neither core nor delta, it is an arch; if it contains 1 core and 1 delta, it is a loop; if it contains no core and 2 delta, it is a whorl. Composites will have mixed patterns. **Types:**

(1) **Loops** [most common, about **67%**]. Ridges enter from one side, recurve and exit from the same side they entered [Show one core and one delta]. These are further divided into (a) **radial** – ridges enter and exit from radial side (b) **ulnar** – from ulnar side.

(2) **Whorls** [next most common, about **25%**]. Ridges make a complete 360° circuit around the center of the print [show 2 deltas].

(3) **Arches** [**7%**] – Show neither core, nor deltas. Further divided into two types. (i) **plain arch** - Ridges enter from one side, rise to a slight bump and exit out from the opposite side. (ii) **tented arch**. Similar to arch, but ridges stand at an angle of 45° or more.

(4) **Composite** [**1%**] - A mixture of above types in a single print.

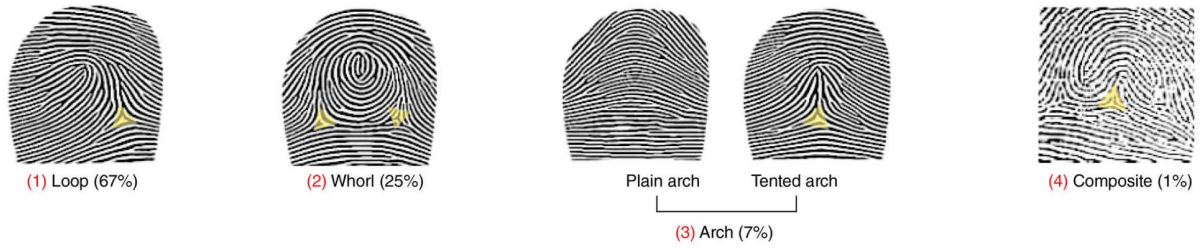


Fig. 3.35: Major fingerprint patterns. Yellow triangles show position of deltas. Please correlate nos (given in red) with text.

Memory Aid 36: Fingerprint patterns

[**Most common patterns to least common patterns**]

Mnemonic (1) **L**ily **W**orks **A**t **C**afe

Loops [67%], **W**horls [25%], **A**rches [7%], **C**omposite [1%]

Mnemonic (2) **L**ove **W**ife **A**nd **C**hildren

2. Comparison of fingerprints

(1) **Identification** - is done by comparing many details of characteristics [**minutiae**] which occur throughout the ridge areas, eg ridge termination, bifurcation etc [**Fig 3.36**].

(2) **ACE-V:**

An acronym for 4 stages of the examination process: **A**nalysis, **C**omparison, **E**valuation, and **V**erification.

(3) **Points to be compared:**

(i) There is no fixed rule.

(ii) Different countries adopt different number of points to be compared.

(iii) Generally 16-20 points of comparison are accepted as proof of identity.

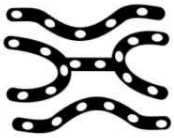
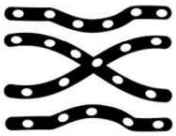
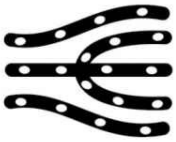
Bifurcation [fork]	
Crossover [bridge]	
Dot	
Double bifurcation	
Independent ridge	
Island	
Lake [enclosure]	
Opposed bifurcations	
Ridge crossing	
Ridge termination	
Spur [hook]	
Trifurcation	

Fig. 3.36: Ridge minutiae characteristics which are compared in fingerprint patterns. White dots are the opening of sweat pores, which are compared in poroscopy

3. Fingerprints on human skin

(1) Can be done to detect fingerprints of murderer, rapist etc. Especially useful

where hands have been used, eg throttling.

(2) **Body at crime scene** - should be processed immediately, and before its touching by anyone else.

(3) **Recommended technique** - Superglue fuming of deceased's body, followed by the application of magnetic powders

(4) **Fingerprints from refrigerated bodies** - cannot be recovered because condensation washes away prints, and interferes with fingerprinting techniques. Yet an attempt may be made after taking out the body and allowing the moisture to evaporate.

4. Alteration of fingerprints

Alteration of fingerprints may be (a) Natural (b) Occupational (c) Intentional [mutilation].

a. Natural

Fingerprints are better protected than other parts of the body, and are thus not easy to change [eg in burns, the fingers reflexly bend inwards against the palm protecting fingerprint patterns]. Following changes are most important

(1) Overall pattern retained, but there is change of distance between ridges – in

(i) **A**cromegaly

(ii) **I**nfantile paralysis

(iii) **R**ickets.

Memory Aid 37: Natural causes of alteration of fingerprints

AIR – Gives 3 most common natural causes when overall fingerprint pattern is retained, but there is change of distance between ridges

(2) Complete loss of pattern – in (i) Burns (ii) Coeliac disease

(3) Epidermal ridge atrophy - in (i) Coeliac disease

(4) Incomplete atrophy of ridges - in (i) Dermatitis

(5) Permanent impairment of fingerprint pattern - in (i) Electrical injury (ii) Leprosy (iii) Radiation exposure

(6) Ridge alteration - in (i) Acanthosis nigricans (ii) Eczema (iii) Scleroderma (iv) Skin atrophy.

b. Occupational

In manual laborers working with cement, gravel, lime, sand etc, fingerprint

ridges may get damaged.

c. Intentional

(1) **Used by:** (i) Criminals to avoid detection by police (ii) Illegal immigrants (iii) Refugees

(2) **Methods used** are

(i) Abrading with a rasping device [a tool similar to a file, used for shaping wood or other material]

(ii) Burns

(iii) Cauterization

(iv) Corrosives

(v) Rubbing against a hard surface

(vi) Self inflicted wounds [by knifes, needles, biting or chewing skin off]

(vii) Skin grafting [plastic surgery] from other portions of body, eg chest.

(viii) Transmogrification - changing into a different pattern, by removing two or more portions of fingerprints and exchanging their places. By doing so, finger ridge patterns become so unusual that they at once arouse suspicion

(3) Except in skin grafting, some part of the skin is generally left undamaged, which can lead to identification.

(4) Attempted mutilation also produces **additional characteristics**, making identification easier.

(5) In most cases [eg burns], fingers on healing begin showing original pattern.

5. Poroscopy

Poroscopy is the examination of impressions left by the sweat openings on fingerprint ridges [**Fig 3.36**].

Salient features:

(1) First described by Edmond Locard

(2) Each mm of fingerprint ridge contains 9-18 pores

(3) **Uniqueness** - They are unique in size, shape, extent, position and number in a given individual

(4) **Permanence** - Pores are permanent and do not change during life

(5) **Medicolegal Importance** – Useful when only fragments of fingerprints are available.

6. MLI [of fingerprints]

(1) **Identification** of (i) criminal (ii) deserters (iii) persons suffering from amnesia (iv) Unknown persons found dead (v) unconscious persons involved in accidents etc (vi) decomposed bodies (vii) accidental or intentional **exchange of babies.**

(2) Fingerprinting in burnt bodies [**ch 14**].

(3) To **maintain identity records** [eg in army, police].

(4) Authentication of sensitive documents [eg driver's license, migration records, passport].

(5) Prevention of **impersonation.**

(6) Documents are often signed by thumb impressions by **illiterate persons.**

(7) **Cheques**, bank notes and other legal documents sometimes may be found to bear a latent fingerprint

(8) **Reveal drug use** - Secretions, skin oils and dead cells in a human fingerprint contain residues of chemicals and their metabolites present in the body. Drugs eg cannabis, cocaine, marijuana, methadone and nicotine can be identified from fingerprints.

(9) **Time of fingerprints** – Can be calculated roughly. Salts in fingerprint residues are stable relative to the esters and proteins. Thus fresh fingerprints will reveal all; older fingerprints would reveal salts, but not the other components.

(10) **Sex from fingerprints:**

(i) From DNA profile [Amelogenin]

(ii) Females deposit more sebaceous residue than males.

(11) **DNA profile from fingerprints** – An average fingerprint leaves between 0.04 to 0.2 ng of DNA from which a DNA profile can be prepared.

(12) **Association with blood groups:**

(i) Blood group A+ and O+ have highest incidence of loops [52% & 54.3% respectively] followed by whorls (33.4% & 30.6% respectively)

(ii) Incidence of whorls is highest in blood groups A- and O- [53.3% & 50% respectively] followed by loops [46.7% & 30% respectively).

(iii) Blood group AB shows highest loops [55% in Rh+ve & 52.5% in Rh-ve], followed by whorls [32.5% in Rh+ve & 27.5% in Rh-ve] and arches [12.5% in Rh+ and 20% in Rh-].

(iv) Frequency of whorls is highest in blood group B in both Rh+ and Rh-

subjects, followed by loops and arches.

L. Footprints

Footprints are prints left in the soil or any other surface by either a bare foot (bare footprint) or shoe (shoeprint). These can be used for identification.

M. Other Prints (Palatoprints, Lip Prints)

1. Ear prints

[A] When a human ear is pressed against a surface, materials present on the ear's surface (waxes, skin oils, etc.) are left behind, forming a two-dimensional "***ear print***" [**Fig 3.37**].

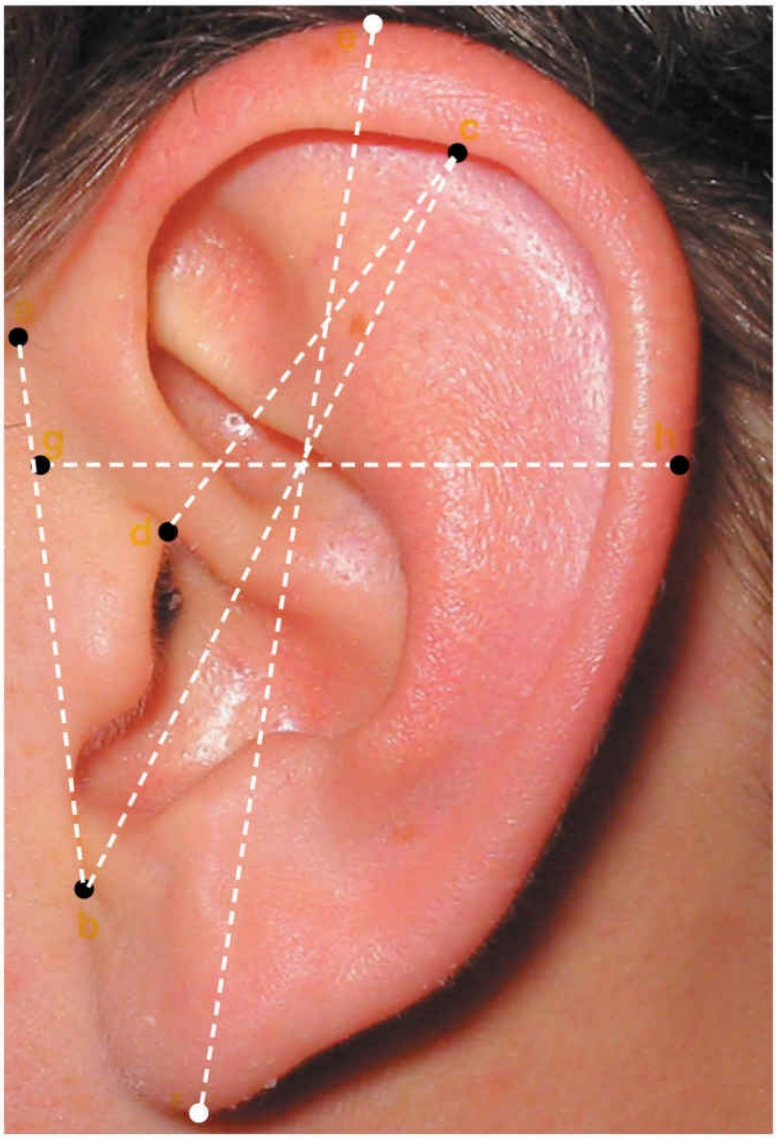


Fig. 3.37: Comparison of ear prints. 5 lines of comparison are formed by 8 points shown here from "a" to "h". The 8 points are: a=upper point of attachment of pinna to face; b=lower point of attachment of pinna to face; c=Darwin's tubercle; d=upper point of attachment of tragus; e=highest point of pinna; f=lowest point of pinna; h=outermost point of pinna; g=the point of intersection of "ab" with a horizontal line drawn from h. The 5 lines of comparison are: ab - Base of auricle; cd - Real length; ef - Largest overall length; gh - Largest overall width; bc - distance between lower insertion to Darwin's tubercle.

2. Lip prints (Cheiloscopy)

The study lip prints is called ***cheiloscopy*****. Forensic cheiloscopy** is the study of

lip prints for the purposes of law and administration of justice.

Salient features:

(1) Like fingerprints, lip prints have various patterns which are different in any two individuals. These patterns are identified and then matched to obtain at a positive identification.

(2) There may be differences in patterns of upper and lower lips and right and left halves.

3. Nose prints

Used to identify pet animals [eg dogs], but not yet used in humans.

4. Palate prints

Palatoscopy, palatoprints or ***rugoscopy***, is the study of palatal rugae to establish identity.

Salient features:

(1) The anterior part of the palate has rough raised ridges (palatal rugae), which are different in every individual, much like fingerprints.

(2) Prints of palatal rugae [**Fig 3.38**] are known as ***palatoprints***. They can be used and matched for positive identification.

Fig. 3.38: Palatal Rugae

5. Tongue prints

Tongue prints include its ***shape*** and the unique ***surface textural features*** found on its surface.

N. Superimposition

Superimposition is the technique applied to identify a dead person when both his skull and antemortem photograph are available [**Fig 3.39**]. Two techniques are used (1) Photographic superimposition (2) Video superimposition.

1. Photographic superimposition

Technique –

(1) **Processing of Photograph** - (a) **Position** - The photograph need not be the frontal view of the person. It can be a lateral, semi-lateral view or any other view. Essentially all major bony prominences namely cheek bones, chin, nasal prominence, outline of top of skull etc must be clearly visible, because these would ultimately be matched with the photograph. (b) **Recent** - In case of a growing child, most recent photograph is better. More recent grown skull may not match with the protuberances of an old photograph.

(c) **Size** - If the photograph is small, it must be enlarged to exact life size. [i] **From an object in picture** - This can be done by noting any physical object in the picture (ear ring, nose ring, hair pin, picture in background, a design on worn cloth, tie etc) and obtaining it from the family of deceased. Photograph is enlarged till the picture of object (in picture) attains a size exactly equal to the size of object obtained from family. [ii] **From interpupillary distance** – If no object is present in the picture, or if the object present in picture is not available, enlargement to life size can be done from interpupillary distance. This is constant in a given person irrespective of where his gaze is. Considering the orbits to be two rectangles, diagonals are made; the pupil lies at the point where the diagonals meet. Interpupillary distance is thus calculated. The picture is enlarged till the interpupillary distance of the picture becomes exactly equal to that calculated from skull. The picture is then exactly life size. (d) A **transparency** of this picture is then prepared.

(2) **Preparation of skull** – (a) Ideally entire skull should be available, but if

mandible is not there, one can still perform the technique (b) If skull had attached soft parts, they must be removed (c) **Mounting** – Skull is mounted on a moveable skull rest which can be rotated on all three axes. Position of the skull can be suitably adjusted (d) Skull is so adjusted that its outline corresponds that of the photograph on transparency (e) A photograph of skull is taken and a transparency prepared (f) The transparency of the life size photograph is now **superimposed** on the transparency of the skull [**Fig 3.39**].

(3) **Comparison** – Several points are then compared.

(4) **Photograph** – The superimposed transparencies are then photographed for court display. Lay judges can be shown this photograph and points of comparison explained.

(5) **Presumptive test only** – Superimposition is a presumptive test.

Fig. 3.39: Technique of superimposition

O. Reconstruction of Features from Skull

Facial reconstruction [syn, facial approximation] is based on the fact that face is influenced by the underlying bone tissue.

(1) **Traditional Method:**

(i) Average thickness of soft parts of the face is measured at several predetermined points.

(ii) Using this data, small wooden or plastic sticks of appropriate thickness are

pasted on the skull
[**Fig 3.40**].

(iii) Clay is then applied over the skull in such a way, that it just comes up to the level of sticks.

(iv) Soft features of mouth, nose, ears etc are applied using the racial data extrapolated from skull [eg negroid etc].

(v) Color of eyes is similarly applied.

(2) **Computer method** – skull is scanned and data fed in computer. From the tissue thickness data, the computer "clothes" the skull with soft tissues. Advantage is that the tissue thickness or other facial features can be altered easily on the computer screen if a new fact comes to light later.

(3) **Advantages** – reconstructed features when published in a newspaper etc is likely to draw much greater public response.

Fig. 3.40: Reconstruction of features from skull

P. Teeth, Bitemarks, Oral Structures [Forensic Odontology]

Forensic odontology or ***forensic dentistry*** is the application of the ***knowledge of dentistry*** for the purposes of law and administration of justice. **Forensic**

odontostomatology is a wider discipline, which uses knowledge of all oral structures [lips, palate] and secretions [saliva] for the purposes of law and administration of justice.

1. Identification from teeth

[A] No two persons have identical dentition in all respects. Dental identification can thus be done by comparison of records between missing person and dead body. Comparison is done by gross examination and radiography. [B] Why teeth are especially suited for identification - because they are protected from fire and trauma etc by external oral structures. Highly resistant to putrefaction also. ***Disaster dentistry*** is use of teeth in identifications in mass disasters [**ch 4**].

a. Gross examination

Following things are compared

(1) **Restorative work** – Note especially (i) Bridge work (ii) Conservative dental work (iii) Crowns (iv) Dental fillings (v) Dentures - (a) Full or Partial (b) If partial, upper or lower (c) Type (d) Shape (e) Look for patient's name or code number included in them (f) Restorative materials used (vi) inlays (vii) Prosthesis if any.

(2) **Special and unusual features:**

(i) Present teeth - Number, spacing and situation. Note especially (a) whether deciduous or permanent (b) caries (c) decayed teeth (d) undersized or oversized teeth (e) loosened teeth (f) chipped teeth (g) broken teeth (h) Fractures (i) Surface (j) configuration (k) Incisal edges
(l) ridges (m) Accessory cusps.

(ii) Absent teeth – Number and situation. Note especially if they are (a) unerupted (b) erupted and extracted by medical intervention. Time period of extraction [there may be evidence of old or recent healing] (c) erupted and fallen (d) If fallen whether due to natural causes or trauma (e) Any evidence of fresh loss.

(iii) General condition of teeth - (a) Age related changes [eg Attrition, periodontosis etc] (b) Cavities (c) Cleanliness (d) Color and Staining [please see below under MLI].

(iv) Peculiarities of arrangement - (a) Crowded teeth (b) Deformities (c) Ectopic teeth (d) Malposition (e) Overlapping (f) Rotation.

(v) Supernumerary teeth.
(vi) Mesiodistal width of teeth.
(vii) Peculiarity of jaws, eg prognathism [prominence of the lower jaw, overbite].
(viii) Oral pathology [eg disease, gingival hyperplasia, old injury].
(ix) Unusual findings – eg Diastema [space between two teeth], Palatal tori [Bony protrusions on the palate].
(3) **Diseases** – Syphilis [Hutchison's teeth].

b. Radiography

(1) **Useful in** – Identification, especially in badly burnt, putrefied or mutilated bodies; in mass disasters etc [please also see "X-rays in identification" below].
(2) **Antemortem X-rays** - In all cases, antemortem X-rays are necessary for comparison. More recent the antemortem X-rays, the more reliable the comparison

2. Age from teeth

Please see above under the heading "Age".

3. Sexing from the teeth

Please see **Table 31**.

Table 31: Sex differences in Teeth

S.No	Criteria	Male	Female
1.	Size and shape of mandibular teeth	Bigger and less pointed	Smaller and more pointed
2.	Size of upper central and lateral incisors	Different	Same
3.	Mandibular first molar	Has a 5th cusp	5th cusp is absent
4.	Detection of sex chromosome from pulp tissue	Y-chromosome	X-chromosome

4. Stature from teeth

Take combined mesiodistal width [CMDW] of 6 maxillary anterior teeth [i.e. all 4 upper incisors + both upper canines]. The formula is as below.

Stature in mm = CMDW [in mm] x 13.65 + 982.421

5. Race from teeth

(1) **Size:**

(i) Large – in Amerinds, Eskimos, Melanesians

(ii) Small – in Lapps and Bushmen

(2) **Mutilation of teeth** by filing and inlaying with precious metals and stones – indicates tribals [tribal custom, cultural peculiarity]

(3) **Cusp of Carabelli** - [syn Carabelli's tubercle, tuberculus anomalus of Georg Carabelli] is a small additional cusp at the **mesiopalatal angle of maxillary first molars** [Table 2]. It is most common among **Caucasoids** (75-85%) and rarest in **Pacific Islanders** (35-45%).

(4) **Shovel shaped teeth** [Fig 3.4]– in Mongoloids

(5) **Taurodontism** – "Bull like" teeth [L, taurus, bull; Gk, odous, tooth]. Found

in molars only. Seen in Eskimos.

(6) **Eruption cyst** - Benign cyst in the gums associated with a deciduous or permanent tooth after it has erupted through the bone. Most prevalent in the Caucasian race.

(7) **Hypodontia and double teeth –** More common in Taiwanese children. Hypodontia - Patient is missing 6 teeth or less, excluding the 3rd molars.

6. Personal habits

(1) **Smoking:**

(i) Cigarette smokers - smoke marks mainly the lingual (inner) surface of upper anterior teeth

(ii) Pipe smokers – develop oval-shaped notches on the occlusal (biting) surfaces from clenching the pipe stem. Similar pattern also in people who chew cigarette holders

(2) **Loss of tooth substance:**

(i) Serrated occlusal surfaces - carpenters, cobblers, seamstresses and women who open bobby pins with their teeth [because they use their teeth to hold nails and pins]

(ii) Anterior tooth loss – trauma caused by wrestling or fighting or using the teeth in certain professions and sports [eg power grasping in holding sled reins or fish lines]

(3) **Abrasions** on labial and occlusal surfaces of teeth – sandblasters and stonemasons

(4) **Dissolved enamel structure** – persons who consume excessive amounts of citrus fruit juices and carbonated drinks

(5) **Alteration of positions of teeth** – Musicians who play mouth instruments

7. Pink teeth

[A] Pink teeth can occur during life or postmortem. During life they occur mainly in CO poisonings. After death they develop in about 1-2 weeks especially near the gumline. [B] **Mechanism of development** - Seepage of hemoglobin or it's derivates into the dentinal tubules. **Prerequisites** are (i) hyperemia and congestion (ii) extravasation of erythrocytes into pulp capillaries (iii) autolysis (iv) damp and humid environment. Therefore, most often seen in ***drowning***. In victims of drowning the head usually lies in a head-down position.

From this it can be assumed that pink teeth could be because of PM lividity. Although seen in drowning most often, they are not pathognomonic of any condition. They have been described in victims of drowning, hanging, knifing, barbiturate poisoning, carbon monoxide and CO_2 poisoning. In addition, pink teeth have also been observed in many cases where the cause of death was not known.

8. Bitemarks

An ***odontoglyphic*** or a ***bitemark*** is a patterned injury produced by the voluntary action of teeth being forced into a softer object.

Salient features:

Bitemarks can be left by humans, animals [both land and sea] and insects.

Human bites

Human bitemarks can be offensive, defensive, self-inflicted, or consensual.

9. Dental charting

Dental charting is a means to provide a quick graphic description of a person's teeth.

Salient features:

(1) There are **>150 different methods** of dental charting in use around the world. The most common ones are described below.

(2) **Advantages:**

(i) Provide a quick overview of patient's teeth

(ii) Records can be stored easily

(iii) Since only mathematical symbols are used, can be understood by professionals knowing different languages

(iv) can be transmitted easily over internet, phone and telegrams.

a. Zsigmondy system (1861)

Earliest system was proposed by Austrian dentist **Adolph Zsigmondy**. The entire mouth was divided in **4 quadrants**, and teeth in each quadrant were represented by **Roman numerals** in children, and **Arabic numerals** in adults.

	Deciduous		
Right	V IV III II I	I II III IV V	Left
	V IV III II I	I II III IV V	

	Permanent		
Right	8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	Left
	8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	

b. Palmer system (1870) [Fig 3.41]

Zsigmondy system modified by Ohio dentist **Corydon Palmer**, because Arabic numbering system (1,2,3 etc) could be confused with Roman numbering system (I, II, III etc). Used very widely in UK and several other countries.

	Deciduous		
Right	E D C B A	A B C D E	Left
	E D C B A	A B C D E	

	Permanent		
Right	8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	Left
	8 7 6 5 4 3 2 1	1 2 3 4 5 6 7 8	

In both Zsigmondy and Palmer notations, if a tooth has to be represented, use is made of a symbol

(

) which designates the quadrant in which the tooth is located. A number indicates the position from the midline. Thus

represents upper right permanent canine. Similarly

represents lower left first deciduous incisor.

Disadvantages – Since letters of English alphabet are used, only English speaking nationals can understand it.

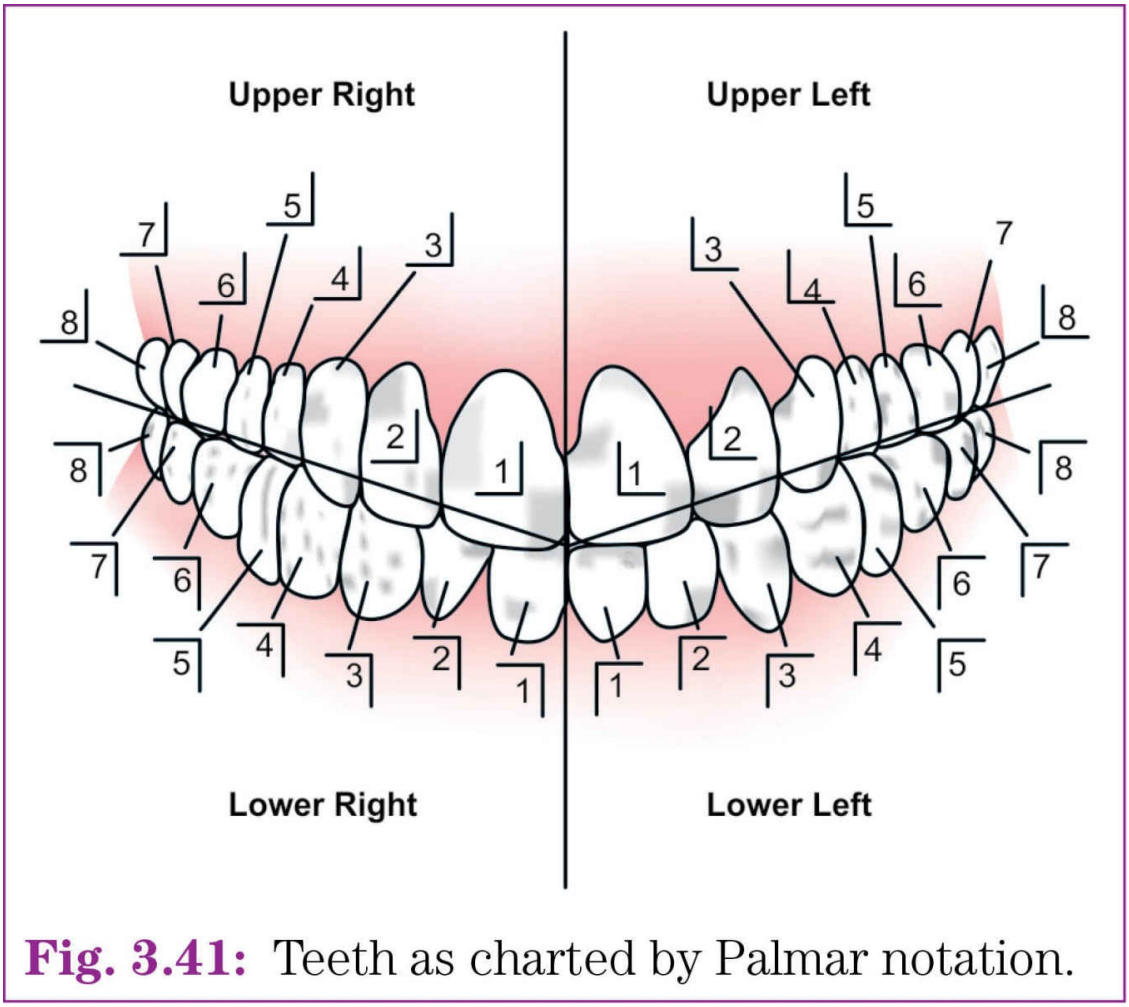


Fig. 3.41: Teeth as charted by Palmar notation.

c. FDI (Theilman) notation (1971)

Two digit notation. Quadrants are 1,2,3 and 4 in adults and 5,6,7 and 8 in children.

Right	1	2	Left
	4	3	

Right	5	6	Left
	8	7	

Charting - Quadrant number comes first. Teeth number comes after this.

	Permanent		
Right	18 17 16 15 14 13 12 11	21 22 23 24 25 26 27 28	Left
	46 77 46 45 44 43 42 41	31 32 33 34 35 36 37 38	

	Deciduous		
Right	55 54 53 52 51	61 62 63 64 65	Left
	85 84 83 82 81	71 72 73 74 75	

Both universal system and FDI system use several two digit numbers which refer to different teeth. This is resolved by insertion of a hyphen between the two digits of the FDI notation [Thus 18 is more appropriately written as 1-8]. They are spoken also differently. **Example**: 16 in universal notation is "upper left third molar" – pronounced as "sixteen" 16 in FDI notation is "upper right first molar" – pronounced as "one six"

d. Modified FDI notation

Same as FDI, except that numbers of the lower quadrant start on a new line.

Right	1	2	Left
	3	4	
Right	5	6	Left
	7	8	

Memory Aid 38: Modified FDI notation

M"N

In **M**odified, numbers start on a **N**ew line

10. MLI [of teeth, bitemarks, oral structures]

(1) **Teeth:**

(i) Identification, age, sex, race. [please see above]. Especially useful in mass disasters

(ii) Blood group from dental tissues [**ch 29**]

(iii) Loss of tooth – is grievous hurt [S.320, IPC; **ch 11**]

(iv) Poisons – As, Hg etc may be detected

(2) **Dentures** – useful in identification [please see "Dental prostheses" above]

(3) **Information from color changes in teeth:**

(i) Intrinsic – Coloration is due to changes in the composition or thickness of the dental tissues (a) Alkaptonuria – brown (b) Congenital erythropoietic porphyria – Color Red-brown. Red fluorescence under UV light. (c) Congenital hyperbilirubinemia - yellow-green (d) Amelogenesis imperfecta

- Varies from ‘snow-capped’ enamel to a yellow to yellow-brown (e) Dentinogenesis imperfecta - bluish or brown. Demonstrate opalescence on transillumination. (f) Tetracycline staining - yellowish or brown-grey (g) Fluorosis – chalky white to a dark brown/black appearance (h) Enamel hypoplasia - pitting and grooving which predisposes to extrinsic staining of the enamel. Later it becomes internalized (i) Pulpal hemorrhagic products – Black (j) Root resorption – pink appearance at the amelo-cemental junction (k) Ageing – gradual darkening of teeth occurs with age.

(ii) Extrinsic - Discoloration is outside the tooth substance; lies on the tooth surface. It can be metallic and non-metallic

(4) **Bite marks:**

(i) Identification [bitemarks left in human tissues or on foodstuffs]

(ii) In children may indicate child abuse [**ch 27**]

(iii) On cheeks, breasts etc of female may suggest sexual assault

(5) **Oral structures** – lead line in plumbism and some other metallic poisons [**ch 36**].

(6) **Age determination** - of antemortem tooth loss or extraction [**ch 17**].

4. Mass Disaster

INTRODUCTION

A. Definition

Mass disaster (also known as ***mass fatality incident***) is any event that produces more fatalities than can be handled using local resources.

B. Classification

Mass disasters can be (1) Natural and (2) Man made (due to acts of omission or commission).

(1) **Natural:** (a) Biological - Disease epidemic [eg cholera, plague] (b) Chemical - Mass poisoning [consumption from naturally contaminated wells, eg arsenic

contaminated wells in some parts of the country, Mass CO_2 poisoning] (c) Other - (i) avalanches (ii) blizzards (iii) cold wave (iv) cyclone (v) drought (vi) earthquakes (vii) famines (viii) floods (ix) heat wave (x) hurricanes (xi) landslide (xii) limnic eruptions [please see ch 44 for details] (xiii) shipwrecks (xiv) storms (xv) tornados (xvi) tsunami (xvii) volcanic eruption (xviii) wildfires and bushfires

(2) **Man made:** (a) Accidental - (i) Airplane crashes (ii) Building collapse (iii) Dam bursts (iv) Fires [Uphaar film hall tragedy](v) Adulterated food [causing food poisoning] (vi) Adulterated liquor [eg hooch tragedy (methyl alcohol poisoning)] (vii) Mining accidents (viii) stampedes in unruly crowds [during sporting events, football tragedies eg Ibrox park, Heysel, Hillsborough, places of worship eg Mecca, temples etc] (ix) Transportation [air, rail, river, road, sea, waterways] (b) Civil disturbances - riots and demonstrations (c) Industrial – (i) Explosions (ii) Fires (iii) Leakage of toxic substances [Bhopal MIC disaster of 1984] (d) Terroristic - (i) bombings (ii) special terroristic incidents like 9/11 WTC incident, 26/11 Mumbai incident (e) Warfare – CBRN - chemical, biological, radiological, and nuclear warfare.

Fig. 4.1: From 14-17 June, 2013, Uttrakhand and adjoining areas received heavy rainfall, and the resulting floods caused widespread devastation including collapse of buildings. **(1)** and **(2)** The first task for the investigating team is to look for any survivors below such debris, rescue them, and provide medical aid immediately. **(3)** A human body recovered after removal of debris **(4)** Recovered bodies are packed for transportation to base camp, where identification procedures etc can be carried out. Help of relatives may be taken to identify victims from clothes, tattoos etc.

5. Medicolegal Autopsy

I. INTRODUCTION

Autopsy, [syn; necropsy, post mortem examination, sectio cadaveris, thanatopsy] is the examination of a person's body after his death. It is an essential part of **death investigation**. An autopsy is of the two types – medicolegal autopsy and hospital autopsy.

A. Medicolegal Autopsy

Done when some ***legal issue*** is involved (homicide, accident, suicide, any other suspicious death). In India, any registered medical practitioner can conduct an medicolegal autopsy on the request of the investigating officer. Permission of

relatives not required.

B. Hospital Autopsy

Done for ***academic purposes***, ***research*** etc. ***No legal issue*** is involved (also called clinical autopsy or pathological autopsy). Permission of ***relatives required***.

II. OBJECTIVES OF A MEDICOLEGAL AUTOPSY

Among the objectives are **6Ws** and **1H** [often jokingly referred to as 6 **w**ives and 1 **h**usband]:

(1) **What** injuries are present? [description of injuries]
(2) **When** did the injuries occur? **When** did the victim die? [time of injuries and of death]
(3) **Where** did the death occur?
(4) **Which** injury caused death? [cause of death]
(5) **Who** is the victim? [identification in unknown bodies]
(6) **Why** were the injuries produced [Manner of death - suicide, homicide or accident]
(7) **How** were the injuries produced [**self-inflicted**, by others, which **kind of weapon** was involved]
(8) **Collection of evidence** (a) **External** - To collect **incriminating evidence** from the dead body (e.g. semen in case of rape, grease and dirt in case of automobile accidents, rope fibers in case of strangulation) (b) **Internal** - To retain relevant organs and tissues as evidence
(9) **Fetus** - In case of fetus, to find its age and thus its viability
(10) In **newborn infants** to determine live birth or dead birth
(11) **Co-relation** - To correlate with ante mortem diagnosis.

III. POSTMORTEM INSTRUMENTS

Common postmortem instruments and their common usages are given in [**Fig 5.1**].

(1) **Forceps** – Various forceps used in PM facilitate manipulation of anatomical

structures. They are also used to stabilize tissues and assist in dissection. All forceps perform essentially the same function but differ in the design of their tips and their intrinsic delicacy of form.

(i) **Toothed forceps** are useful for **stabilizing** and **moving** tissues, but they are traumatic and may lacerate structures.

(ii) **Smooth atraumatic forceps** are more appropriate when trauma must be avoided, as in exploring traumatized structures, or for delicate vascular manipulation.

(iii) **DeBakey forceps** are good general-use instruments with atraumatic **flat tips** and **tiny fine serrations**.

(iv) **Fine-toothed Adson forceps** and

(v) **stout-toothed Bonney forceps** are more commonly used in surgery but may be used for PM also. **Adson** is ideal for skin closure, while **Bonney** is excellent for facial closure [after facial dissection].

(2) **Knife blades** – In **India, Bard-Parker knife blades** are commonly used. These are made functional by attachment to a **standard no. 3 Bard-Parker knife handle**. Choose the size and shape of the blade based on the intended indication. **In surgical practice, abdominal** or **thoracic skin incisions** are typically made with no. **10, 20, or 22 blades**, whereas more delicate incisions require the **smaller no. 15 blade**. However such distinction in PM is more or less of an academic nature. In surgery, the **sharp-tipped no. 11 blade** is ideal for entering and draining an abscess or for making an arteriotomy by incising a blood vessel in preparation for vascular procedures.

(3) **Needles** - come either straight or curved.

(i) **Straight needles** are rarely used in PM, but may be used if curved are not available

(ii) **Curved needles** -are usually **half circle** or **three-eighths circle**. Sewing in a deep hole may require a **five-eighths circle needle**, whereas sewing up of smaller structures often requires **quarter circle needles**. Needles can have an eye for threading the suture (**French eye**) or an already attached suture (**swaged**). Surgeons mostly use **swaged needles** [needle-suture combination], but they are rarely used in PM. Etymologically, a swage is a blacksmithing tool used to shape metal. Thus a **swaged needle** is manufactured by placing the suture into the hollow shank of a needle and then compressing the needle around the suture, holding it firm. Some

sutures are swaged to needles in such a manner that they pop off if excess tension is applied between suture and needle.

(iii) **Large needle** [with twine]– This is a general purpose needle most commonly used for sewing up bodies after PM.

(iv) **Needle tips** - are either tapered or cutting. **Tapered needles** are **circumferentially smooth** and slide between the elements of tissues, whereas **cutting needles** are **triangular** in cross section and **cut through tissues like a tiny knife**. More tougher tissues, eg **mummified bodies** may require cutting needles.

(4) **Dissecting scissors** – cutting of tissues, general purpose cutting

(i) **Iris scissors** - are fine and are used for delicate dissection and cutting.

(ii) **Mayo scissors** -are sturdier and are used for cutting thick or dense tissues, such as fascia, scar, or tendons.

(iii) **Metzenbaum scissors** - are versatile, general-use instruments.

(iv) **Richter Dissecting scissors** - comes with angular blades, one sharp and one blunt point. Useful for reaching inaccessible areas eg below pubis to cut out prostrate.

(v) **Scissors with one pointed and one blunt end** – used for cutting tough tissues, fibrotic scars etc.

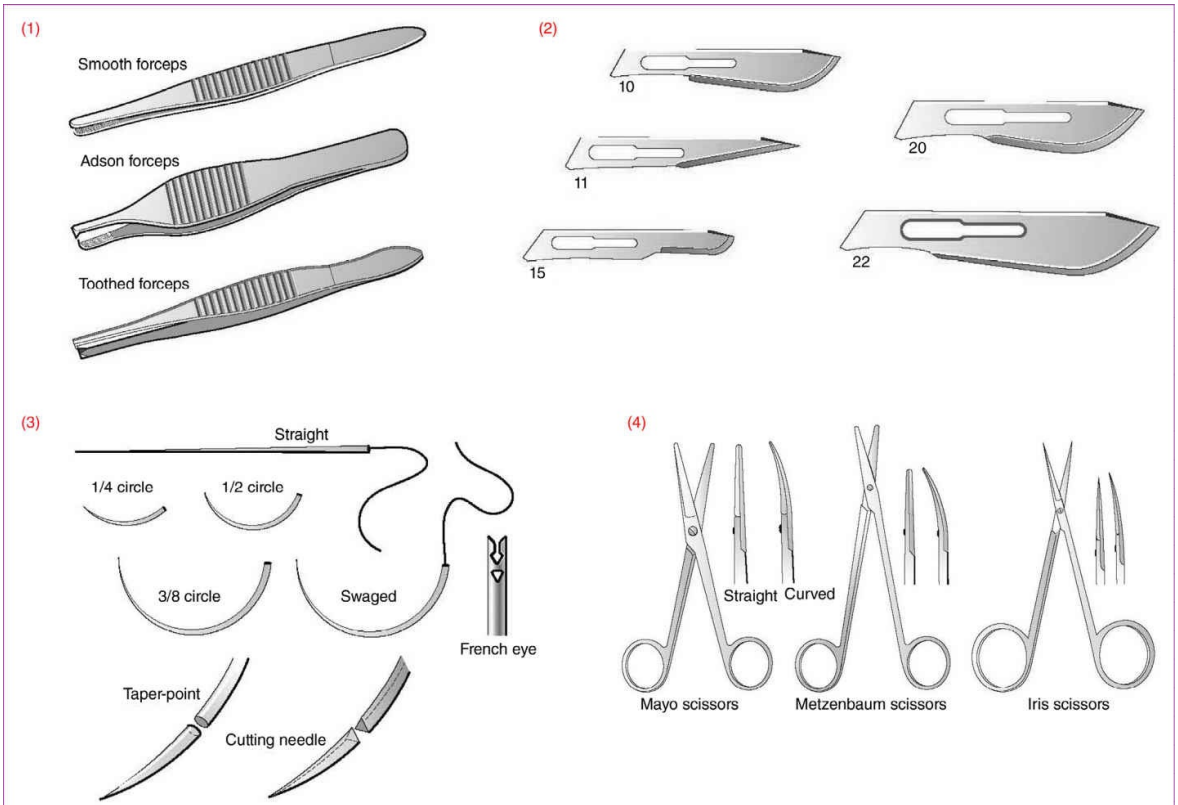


Fig. 5.1: (1) Smooth, Adson, and toothed forceps. (2) Knife blades used for PM in India. Shown here are Bard-Parker knife blades nos. 10, 11, 15, 20, and 22 (3) Various needles used for sewing up dead body (4) Mayo, Metzenbaum, and iris scissors. (4) **Scissors** - mainly used for dissecting and cutting tissues. All scissors are designed for right-handed use. Most scissors have either **straight** or **curved tips**. Please correlate nos (given in red). with text.

IV. EXAMINATION OF CLOTHINGS AND ORNAMENTS

A. Clothings

(1) List - all clothings

(2) Describe -

(i) Burns [if present]

(ii) Buttons [loss of]

(iii) Color

(iv) Creases [lack of]

(v) Design

(vi) Disarrangement [indicates struggle]

(vii) Staining [blood, feces, grease, mud, poison, saliva, semen, urine, vitriolage], and their relative distribution. Are the stains in several layers, eg a layer of semen over blood or vice-versa. Can help reconstruct events

(viii) Tears [indicates struggle]
(ix) Type of garment
(x) wetness [as in drowning]
(xi) wrinkles [presence of].

V. EXTERNAL EXAMINATION OF THE BODY

External examination is very important, especially in cases of trauma, where a number of injuries are visible externally. Following points must be noted

(1) Record height, weight, age, sex
(2) **General features**
(3) **Hands** - (a) Trace evidence
(4) **Skin**
(5) **Eyes**
(6) **Palpate** (a) Abdomen (b) breasts [In females] (c) testes [In males].

VI. INTERNAL EXAMINATION

(1) **First cavity to be opened** –

(i) General rule - As a rule of the thumb, skull should be opened first. Reasons (a) **smell** of poisons is best appreciated before odorous gases from abdomen are released. Brain can give out smell of poisons like alcohol, phenol, organophosphorus etc. (b) **Blood drained out** – through vessels going to the brain. Creates a bloodless field in the neck, which is ideal for appreciating antemortem neck injuries [**ch 10, 19**].

(ii) If a cavity shows obvious signs of injury – It may be profitable to start examination with the affected cavity [eg abdomen in case of stab abdomen]. Reasons – (a) Pathologist is less exhausted initially and his best energies are spent on the main cause of death (b) Findings in other cavities may be co-related appropriately with those in the chiefly affected first [eg bleeding in abdomen will help understand better a pale brain]

(iii) In infanticide - after skull, abdomen should be opened next [**ch 27**].

(iv) For other precautions, please see **ch 10**"Artifacts introduced during autopsy"extravasation of blood.

(2) **Incisions** – Following incisions are standard, but they may have to be adapted to special situations [eg in stab wounds in the midline of chest and abdomen, the midline incision may have to be diverted sideways].

A. Skin Incisions

1. I-shaped incision

(1) Most common

(2) Starts from just under the chin in midline and goes up to the pubic symphysis, sparing the umbilicus (**Fig 5.2**). The umbilicus is spared as it consists of dense fibrous tissue which is difficult to stitch.

2. Y-shaped incision

(1) **Incision** -

(i) Starts from the two mastoid processes, which form the two limbs of the Y [**Fig 5.2**].

(ii) The two limbs move medially to meet at the level of xiphisternum, from where it goes downwards exactly as I-shaped incision

(2) **Used** -

in cases of neck trauma, where layer dissection of muscles is needed

(3) **Precautions** -

Air embolism - Neck dissection should be performed particularly carefully, being alert not to injure the large neck veins.

3. Modified Y-shaped incision

(1) Starts from below both anterior axillary folds, goes below the breasts, meets at xiphoid process and proceeds down to symphysis pubis [**Fig 5.2**].

(2) **Used** - in females to preserve appearance [incision line is not visible on exposed face and neck].

4. T-shaped (subclavicular) incision

(1) Straight incision from suprasternal notch till the pubic symphysis

(2) From the suprasternal notch, the incision is extended horizontally on both sides to the acromion processes [**Fig 5.2**]. Gives a wide exposure of base of neck

(3) **Used** – when exploration of base of neck is necessary as in dissection of

vertebral arteries.

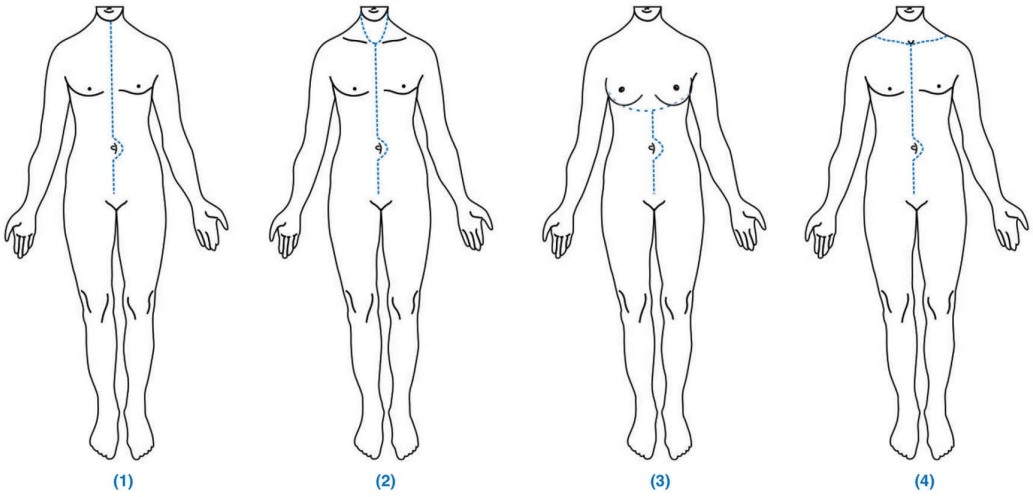


Fig. 5.2: **(1)** I-shaped, **(2)** Y-shaped, **(3)** modified Y-shaped, **(4)** T-shaped (subclavicular)

B. Method of Removal of Organs

1. Virchow's method

(1) Advocated by **Rudolph Virchow** (1821–1902), German pathologist.
(2) Organs are removed separately one by one and then studied individually.
(3) **Adv** - Quick
(4) **Disadv** - causes loss of continuity; difficult to evaluate interrelationships.

2. Rokitansky's method

(1) Advocated by **Carl Rokitansky** (1804–78), German pathologist.
(2) in situ dissection
(3) **Adv** – good in children; infected bodies
(4) **Disadv** – Difficult in adults.

3. Letulle's method

(1) Advocated by **Maurice Letulle** (1853-1929), French pathologist.
(2) All organs from tongue till prostate [oral, cervical, thoracic, abdominal, pelvic] are remove en masse in a single block
(3) **Adv** -
(i) excellent preservation of organ inter-relationships, their regional lymphatic drainage, and their vasculature

(ii) body can be handed over to relatives quickly
(4) **Disadv** – Organs difficult to handle.

4. Ghon's method

(1) Advocated by **Anton Ghon** (1866-1936), Austrian pathologist.
(2) Compromise between Virchow's and Letulle's. Cervico-thoracic, abdominal and pelvic organs are removed as 3 separate blocks [en bloc method]
(3) **Adv** -
(i) excellent preservation [as in Letulle's]
(ii) Handling of organs easier than in Letulle's
(4) **Disadv** – If disease process extends across the three blocks, interrelationships may be difficult to study.

C. Examination of Organs

1. Head

a. Skull

(1) A wooden block is placed under the shoulders. It extends the neck.
(2) Head is stabilized by a head rest which has a semicircular groove to hold the back of neck.
(3) The hair is parted with a comb in a line joining the mastoid processes, passing over the vertex, and the scalp incised down to the bone along it. The incision should be deep enough to penetrate the periosteum.
(4) The two scalp flaps are then reflected down to the supraciliary ridges in front and to the external occipital protuberance behind [**Fig 5.3**]. If the reflection is difficult, a scalpel blade can be used to cut the loose connective tissue as the other hand continues to peel the scalp. The knife edge should be directed toward the skull and not toward the scalp [otherwise it would create "buttonholes" in the scalp]. The anterior flap is reflected to a level approx 2 fingerbreadths above the supraorbital ridge [**Fig 5.3**]. The posterior flap is reflected down to a level approx 2 fingerbreadths above the occipital protuberance.

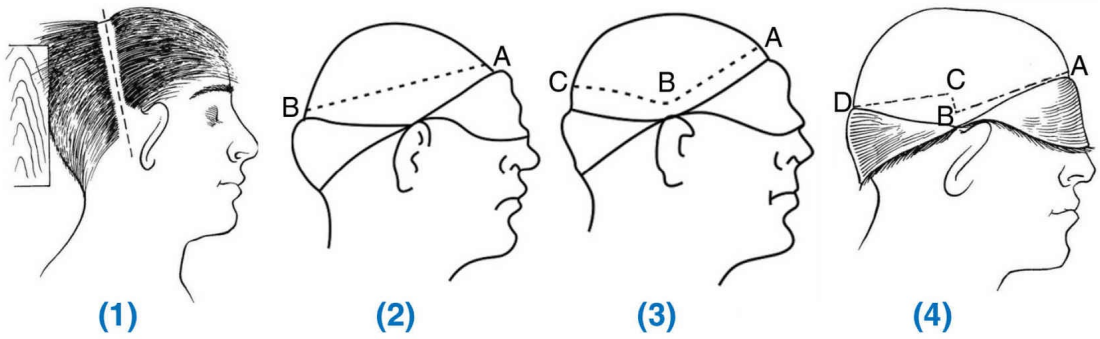


Fig. 5.3: (1) Scalp incision. (2), (3) and (4) depict lines of saw cut for skull cap removal. (2) shows straight line AB; (3) V shaped line ABC; and (4) zigzag line ABCD. Please see text for details.

i. Dissection of Head in infants

(1) Child's brain attains mature size and weight at about 5 y

(2) In newborns and infants, different approaches for removal of brain are adopted for two reasons

(i) firm adherence of dura to the skull [**ch 17**]. It tends to dip into sutures

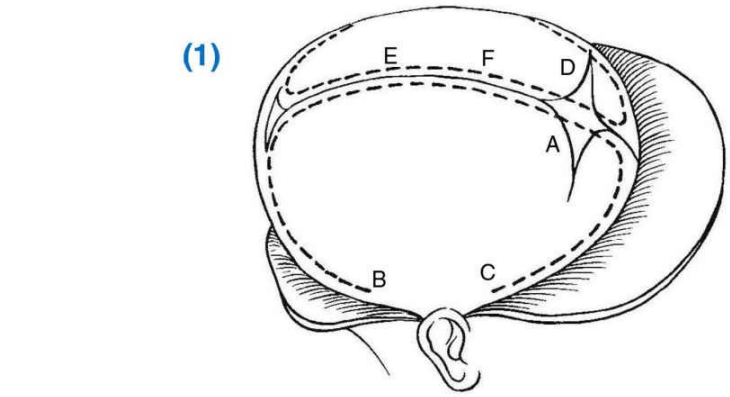
(ii) Importance of keeping the falx and tentorium intact [their injuries are common in difficult births].

(3) **Two approaches are used** -

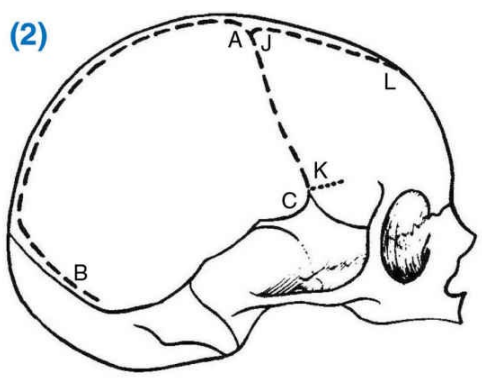
(i) Beneke's technique - Opens skull in 2 flaps. (a) The scalp is reflected as in adults. (b) Points to be noted at this stage - (I) Any caput formation or hemorrhage (II) width of the sutures, size of fontanelles, degree of calcification [to assess hydrocephalus] (III) Any separation of the occipital plates caused by birth trauma (IV) Incision of the atlanto-occipital membrane will reveal tonsillar herniation and posterior fossa hemorrhage. (V) A sample of CSF from the cisterna magna can be removed if necessary, using a sterile needle. (c) Removal of skull - (I) cutting along the suture lines will destroy the falx and the superior sagittal venous sinus. Therefore cut is made about 0.5 cm on either side of the sagittal suture (II) A scalpel is used to make an incision starting from anterior fontanelle at its posterior margin, about 0.5 cm [5mm] on the right side of midline [point A in **Fig 5.4**]. The incision will cut both skull and dura together. After an incision of about 2 cm or so, introduce one end of a heavy duty scissors inside skull, and cut posteriorly up to point B and anteriorly up to point C. This will

allow the right side of skull to come out like a flap [B-A-C]. (III) Similarly dissect the left side of skull making similar incisions and reflect the flap E-D-F [**Fig 5.4**]. This leaves an intact area in the temporal squama on either side [BC on right side and EF on left], which serves as a hinge as the bone flaps are reflected. (IV) A midline strip approx 1 cm wide [5mm either side of midline], is now left at the top, containing the superior sagittal sinus and the falx. The older the infant, the narrower the sagittal strip will be because ossification advances toward the midline, as age advances. (d) Now vertex of the brain can be seen [barring for a 1 cm wide strip at the top]. Make sure that gloves are wet before touching the brain. Now Examine (I) the terminations of pial veins entering into superior sagittal sinus (II) Any hemorrhages around (III) Gently push each cerebral hemisphere to the side turn by turn and examine falx cerebri for any tears (IV) medial parts of each cerebral hemisphere (V) space between two dural layers of the falx (e) Now open superior sagittal sinus and look for any thrombi (f) Separate falx at its antero-inferior insertion (g) From the sides examine tentorium for tears and for hematoma between its layers (h) Remove brain as in adult (i) Open dural sinuses at base of skull and examine.

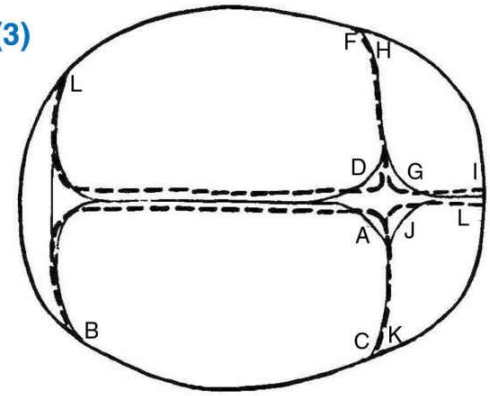
(ii) Baar's technique – Opens skull in 4 flaps ["butterfly" manner of removal] – Procedure is same except that in this case, 4 flaps [**Fig 5.4**, B-A-C, E-D-F, H-G-I and K-J-L] are removed like the 4 wings of a butterfly. This technique is preferred by most pathologists.



Beneke's technique



Baar's technique (Side view)



Baar's technique (Top view)

Fig. 5.4: Two methods of opening the calvarium in fetus and neonate. **(1)** Beneke's technique **(2)** and **(3)** Baar's technique. Please see text for details

b. Brain

Examination of brain

(1) **Surface and base of brain** - examine for any obvious signs of disease, eg encephalitis.

(2) **Cerebral vessels** – note atherosclerosis, aneurysms, or evidence of other disease, especially in those forming the circle of Willis.

(3) **Cortical contusions and hemorrhages** – are detected best in fixed brains; conversely small hemangiomas and ruptured Berry aneurysms are best detected in unfixed brains.

(4) **Ruptured berry aneurysms** – put the brain under a gentle flow of running

water. Ruptured walls of berry aneurysms “fan out” under the stream, much like those of a ruptured balloon. Size of Berry aneurysms vary from few mm – few cm, and are usually present at the junction of vessels.

(5) **Note presence of cerebral infarctions** – These can be due to atheroma, thrombus for due to -ed ICP causing obstruction to venous outflow.

2. Chest

a. Heart

i. External examination

(1) Hold in anatomical position - Assess its size and shape.

(2) Examine epicardial surface - for evidence of

(i) adhesions

(ii) aneurysms

(iii) flaccidity [may indicate underlying infarction]

(iv) focal hyperemia

(v) pericarditis

(vi) rupture

(vii) Small white “soldier’s” patches are often seen, particularly anteriorly over the right ventricle. They represent either previous trauma or episodes of subclinical pericarditis.

(3) **Weight** - Heart should not be weighed at this point, as it will inevitably contain blood clot which would falsely elevate the weight obtained, leading to mistaken impressions of hypertrophy.

ii. Examination of the coronary arteries

Dissection of the Coronary Arteries – Two methods are mainly used – transverse sectioning [more common] and longitudinal opening. In both the origin of the arteries must be identified and their course within the epicardium followed. This is variable, and in difficult cases it may be necessary to first identify the ostia by inspecting the aortic sinuses from above. Look for any evidence of thrombus or loose atheroma; its extent and severity should be assessed, and the affected areas noted. If the arteries are sectioned transversely, an estimation of the percentage of luminal stenosis should be made, and must be represented by diagrammatic charts

(1) Transverse Sectioning Technique - (a) A sharp scalpel blade is used to slice completely through the arteries at intervals of about 2-3mm. (b) Proximally, where atheroma and thrombus are more likely, the transections should be even closer together. (c) **Note** – Calcification, Plaque hemorrhage, Plaque rupture, Stenosis, Thrombi etc (d) If vessels heavily calcified, it will be necessary to cut the vessel with a sharp pair of artery scissors; it will give a gritty feel as if one is cutting through chalk. (e) Advantages – (I) Percentage block can easily be estimated (II) Antemortem thrombi can be demonstrated without risk of dislodging them. In longitudinal opening method neither is possible. Thrombi can easily be dislodged and pushed further along.

(2) Longitudinal Opening Technique - (a) A sharp pair of very fine artery scissors is used to open the arteries from the ostia, extending as far down their course as possible. (b) Advantages - (I) Gives a good idea of the length of the plaque. Helps to assess the length of the vessel occupied by a thrombus or by complicated atheroma or both (II) allows to track any sizeable small branches, and to look at narrowing of their openings (III) gives a view of the intimal tears (IV) If the vessel wall is markedly calcified, less damage and distortion occurs than by transverse sectioning.

(3) **Clinical Correlation** - Significant or severe disease is indicated by a stenosis of 75% or more. In the absence of occlusive thrombus, death should be ascribed to atherosclerosis only if the stenosis is severe.

iii. Dissection of the heart

(a) Along the path of blood flow

(1) Several methods have been described, but the best is opening along the path of blood flow [**Fig 5.5**]. An enterotome is used for dissection.

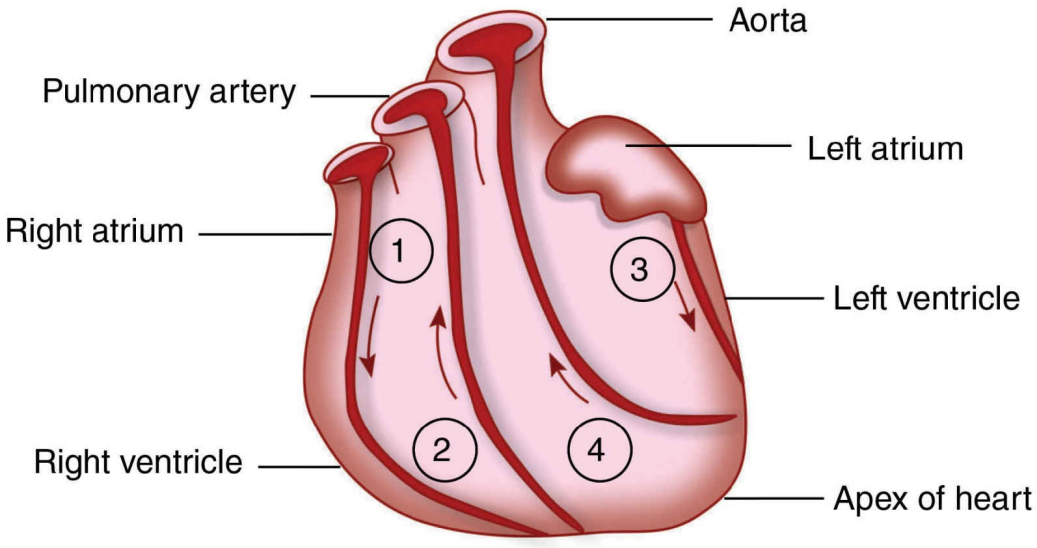


Fig. 5.5: Opening heart along line of blood flow.

iv. Internal examination of the heart

(1) **Weight** - Whichever method of examination has been used, all post mortem blood clots should now be removed and the heart weighed.

(2) **Morphology of the chambers** - examined closely and any septal defects noted.

(3) **Endocardium** - examined for evidence of fibrosis, attached antemortem thrombi, and subendocardial hemorrhages.

(4) **Subendocardial hemorrhages** [syn, Sheehan's hemorrhages]–First described in the **1930s** by the English pathologist **Harold Sheehan** (1900-1988) in cases of abortion and acute hemorrhage associated with pregnancy.

(i) Mostly seen in (a) left ventricle (b) left side of IV septum, opposing papillary muscles and adjacent columnae carnae.

(ii) Shape - flame shaped, confluent, tend to occur in one continuous sheet rather than patches.

(iii) When bleeding is severe, endocardium may be raised like a flat blister

(iv) **Seen in the following conditions** - (a) Acute heavy metal poisoning, especially arsenic (b) Deaths due to obstetric complications (ante-partum or post-partum hemorrhage, ruptured tubal pregnancy, ruptured uterus, abortions) (c) Deaths due to shock especially hemorrhagic shock (d)

Intracranial damage [cerebral edema, head injuries, surgical craniotomy, tmours]

(v) **Mechanism of production** - obscure but may be due to sudden and profound hypotension within the ventricles"BP within the coronary system for some moments remain normal"causes a pressure gradient across the endocardium (at least of 80 mm Hg, if we assume the pressure within the ventricle at 0 mm Hg and in the coronary system at a diastolic level of 80 mm Hg)"superficial vessels rupture"subendocardial hemorrhages. The theory however fails to explain these hemorrhages in cases of head injury. If the vagus nerves of goats are severed and then they are killed by head injury, Sheehan’s hemorrhages fail to appear. This suggests that these hemorrhages may be mediated by autonomic nervous system.

(5) **Valves** - Examine number of cusps and leaflets, fusion between them and evidence of vegetations, fibrosis, or calcification. The valve circumference is then be measured with a ruler or a length of string. Average measurements are given in appendix at the end of book.

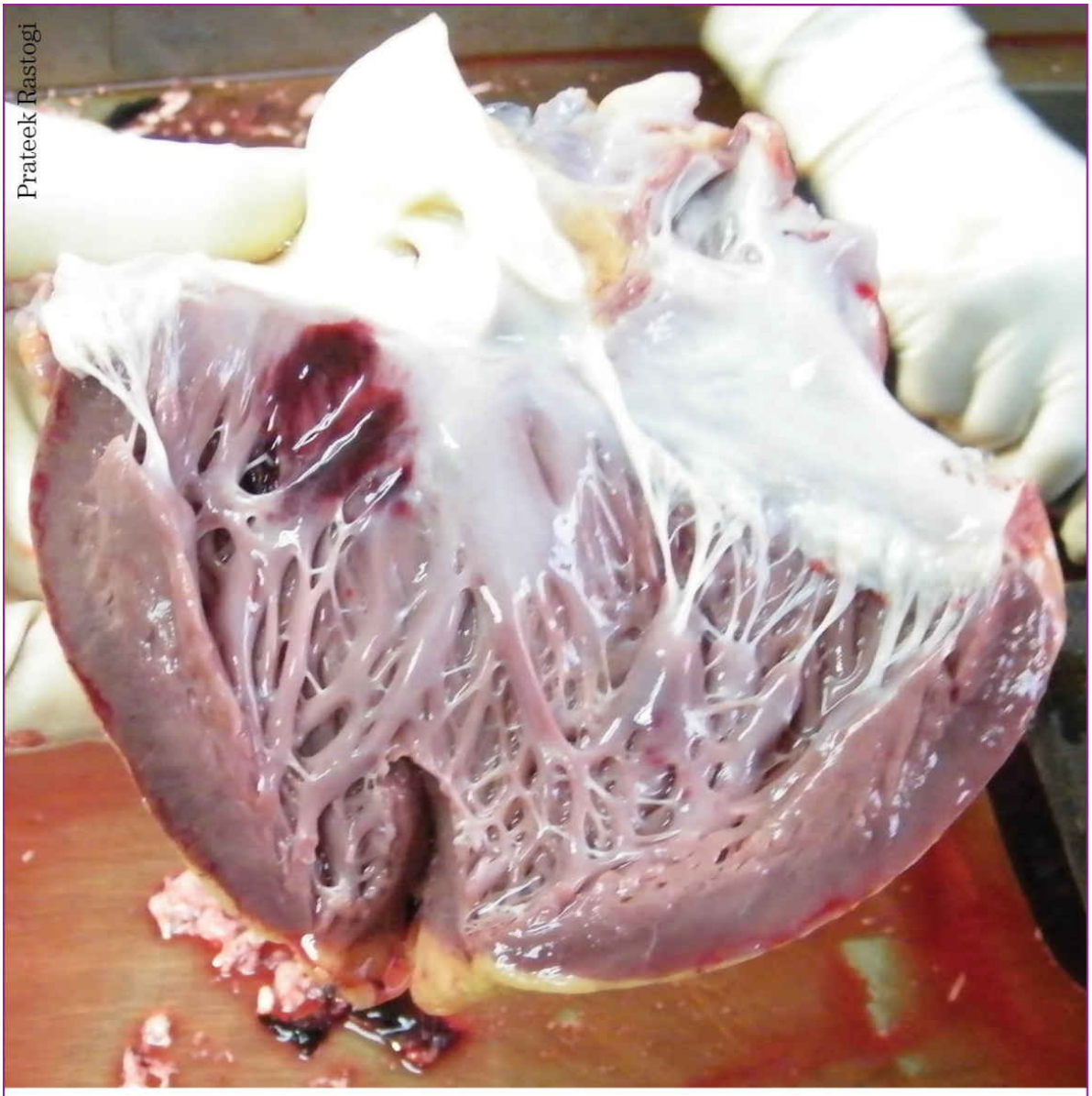


Fig. 5.6: A large patch of subendocardial hemorrhage just below the mitral valve in a case of head injury.

b. Lungs

Removal - by cutting through each of the main bronchial stems distal to the carina with a large pair of scissors or PM40. A large bronchial stump should remain attached to the lung by making the cut as away from lung as possible.

3. Gastrointestinal system

a. Stomach

(1) **Removal** - Stomach is removed after applying ligatures at each end"Placed on a clean dish"Open along greater curvature [reasons **ch 31**].

(2) **Contents** -

(i) Examine for nature of food, its state of digestion, smell, color, character, presence of foreign and suspicious matter.

(ii) Clear contents in a clean jar.

b. Liver

Removal and examination of accessory structures

(1) Removed by itself or when still attached to stomach and duodenum.

(2) The latter is done when patency of bile ducts are to be examined. Duodenum is exposed where ampulla of Vater opens and gall bladder pressed. Greenish bile is seen to ooze from the ampulla if bile ducts are patent.

(3) Open the bile duct with a fine pair of scissors and look for calculi, strictures and tumors

(4) Open hepatic artery and portal vein

(5) Examine lymph nodes in the neighborhood.

4. Genitourinary system

Kidneys

(1) **Renal ostia** - As the abdominal aorta is opened along its anterior midline, the renal artery ostia are examined for thrombi, emboli and atherosclerosis.

(2) **Renal vessels** – examine for thrombi

(3) **Kidneys** – note

(i) Congenital malformations – agenesis, aplasia, hypoplasia, horseshoe kidney, pancake kidney [both kidneys are fused into a single mass in the pelvis]

(ii) size and shape - (a) Normal size 11 x 6 x 3cm, in adults. (b) Normal Wt –150 g [males, each kidney], somewhat less in females. (c) Causes of atrophic kidneys - (I) arteriosclerosis of renal arteries, (II) chronic obstruction, (III) glomerulonephritis

(iii) Note smoothness or granularity of surface.

5. Spinal cord

(1) **Methods of removal** -

(i) anterior approach

(ii) posterior approach

(2) **Anterior approach** –

(i) Advantages – body need not to be turned over (a messy procedure if evisceration has already taken place) allowing the nerve roots and dorsal ganglia to be dissected.

(ii) Disadvantages - more difficult

(3) **Posterior approach** -

(i) Advantages – (a) quicker and easier, but should be performed before the rest of the PM, to avoid mess. (b) Allows for removal with less tension on the cord (c) allows the SC and brain to be removed in continuity

(ii) Disadvantages- (a) does not allow easy examination of the nerve roots or dorsal ganglia (b) When body is returned to relatives, the sheet may become blood soaked through oozing of blood from posterior incision. To reduce this oozing, it is advisable to put a plastic sheet inside before sewing up the incision.

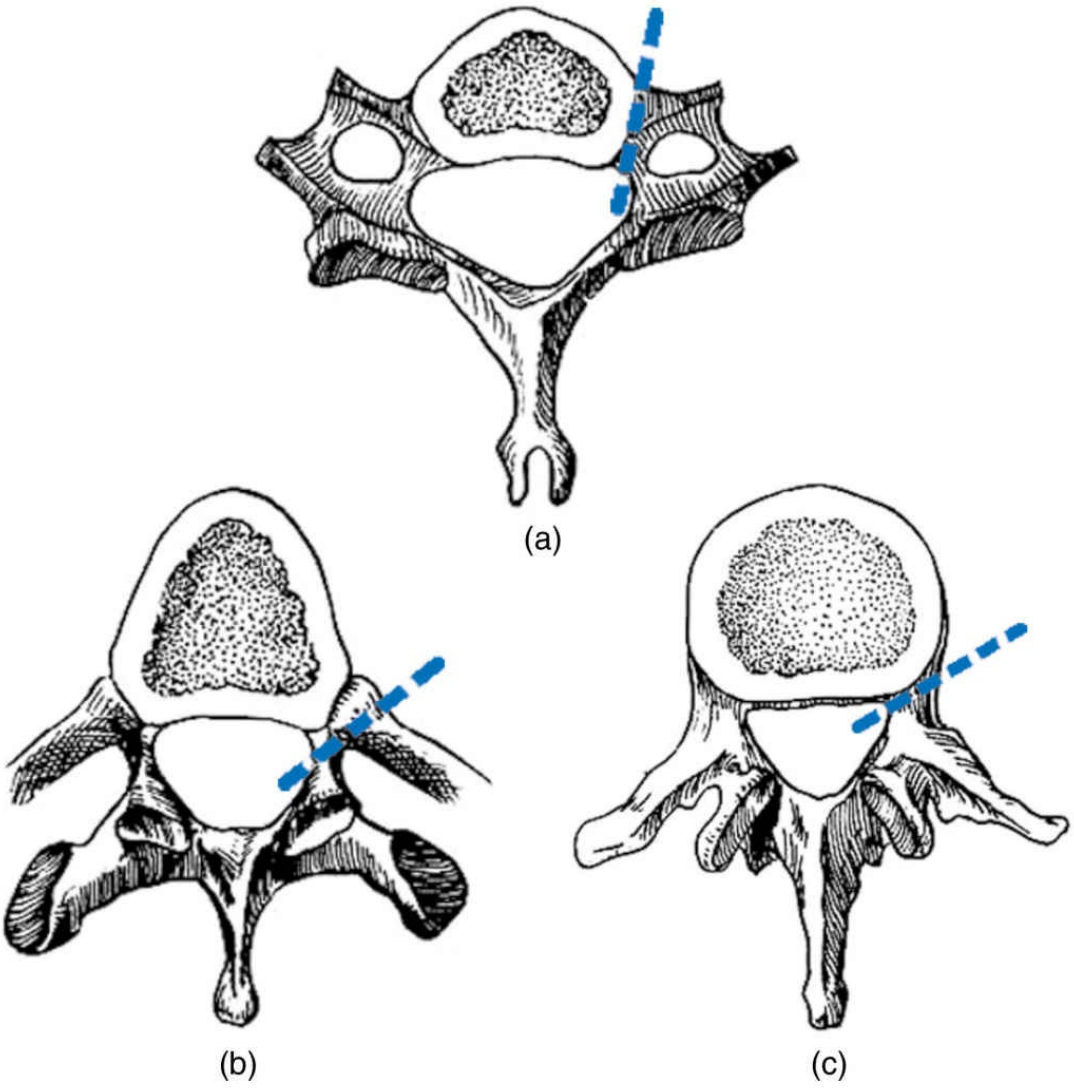


Fig. 5.7: Removal of the spinal cord via **Anterior approach** - Dotted lines between vertebral body and arch indicate planes of saw cut adjusted to shapes of different levels of vertebral column. (a) Cervical. (b)Thoracic. (c) Lumbar.

6. Blood

(1) **Postmortem clotting -**

(i) Initiates within **1 hour** of death.

(ii) Simultaneously, clot lysis also initiates.

(iii) Since the two processes occur simultaneously, the presence of absence of clots in a dead body depends on which process prevails. It may be completely clotted, completely fluid or partly clotted and partly fluid.

(iv) Generally in small capillaries, blood is completely clotted by **8 h** [time of fixation of pm staining - **ch 9**]. But at the same time blood may be partly fluid in large vessels.

(v) Amount of blood clots vary from body to body, and from site to site within the same body.

(2) **Clotted blood for toxicologic analysis:**

(i) When fibrin clot is present it always entraps large numbers of RBCs, so that the clot is relatively RBC-rich.

(ii) For drugs with an unequal distribution between RBCs and serum [eg (a) alcohol - distributed only in the water portion of blood (b) chloroquine -has an RBC: serum ratio of 32:1], examination of clots may give spurious results

(3) **Clotted blood for DNA analysis - ch 29**.

(4) **Hb levels and poison concentrations -** no proven correlation exists.

a. Agonal thrombi

Agonal [Gk agon, struggle] ***thrombi*** are firm, stringy, tough, pale yellow thrombi which form during terminal phase of life in the cavities, usually on the right side of heart, especially in persons dying slowly with circulatory failure, and diseases such as lobar pneumonia, in which the coagulability of the blood is increased.

Salient features:

(1) **Beginning of process** – occurs in (i) atrial appendage (ii) apex of ventricles or (iii) in the angles of the ventricular surfaces of tricuspid valve

(2) **Right heart** – Extends and fills both right auricle and ventricle, pulmonary artery and even its finest branches to give an appearance of tree like cast

(3) **Left heart:** (i) usually less extensive (ii) When it passes through aorta it bears projections, corresponding in shape, though not in size, to the sinuses of Valsalva.

b. Postmortem clots

(1) Postmortem clots form after death. They should be differentiated from antemortem clots [**Table 1**].

(2) **Types** – Two types

(i) Black currant jelly type – forms when blood clots rapidly. Heavier RBCs do

not have time to settle down; they are found distributed throughout the clot. The clot is lumpy, moist, slippery, soft and uniformly dark red.

(ii) Chicken fat type – forms when blood clots slowly. Heavier RBCs have time enough to settle down. Two layers are seen in the clot; a lower redder layer, which is quite similar to black currant jelly type clot, and an upper pale or bright yellow layer of serum and fibrin

(3) **Cardiac polyps** - These are pm fibrinous, pedunculated clots in the heart.

Table 1: Differences between antemortem and postmortem clots

S.No	Criteria	Antemortem clots	Postmortem clots
1.	Color	More or less uniformly colored [grayish red]. There are no contrasting layers.	Two distinct layers are seen (in chicken fat type) with a dark red dependent portion where red cells have settled by gravity and a yellow "chicken fat" upper portion.
2.	Consistency	Firmer, tougher	Gelatinous, homogenous, moist, rubbery, shiny, smooth, softer
3.	Lines [striae] of Zahn [fine white lines of fibrin]	Present. Little coral-shaped masses of platelets [coralline thrombi] form within the turbulent bloodstream, as the framework on which fibrinous clot continues to form.	Absent
4.	Attempts at stripping in layers	Shreddy, and often can be stripped or peeled in layers	Each part of the clot is homogenous in construction, like jelly and it cannot be thus stripped into layers
5.	Adherence to bv	Clot firmly adherent to blood vessels	Not firmly adherent. PM clot may interlace amongst the chordae tendinae, which may cause difficulty in its removal. It should not be mistaken for true adhesion
6.	Surface of bv after blood clot is stripped off from it	Roughened	Quite smooth and healthy looking
7.	When lung is sliced	Emboli project [or pour out] slightly above the surface of cut vessels	Do not project

c. Unclotted blood

Blood remains fluid after death in certain conditions [ch 9"pm lividity]. PM clots are greatly reduced in number in these conditions.

D. Procedure after Completion of Autopsy

(1) Clean body cavities – of blood, fluids

(2) Sew all cuts, clean body, hand over to police.

VII. EXAMINATION OF WEAPON

If weapon is submitted along with body, it must be examined after the autopsy is over. Co-relations of injuries must be made. The weapon may be blunt, sharp or

firearm.

VIII. THE AUTOPSY REPORT

(1) ***Autopsy report*** [syn, postmortem report] is a medicolegal report. Like all MLRs it consists of preliminaries, body and opinion. For details, please see **ch 1.**

(2) **Headings** – An ideal autopsy report should have following headings

(i) Preliminaries – name, age, sex etc
(ii) History
(iii) Date, Time, Place, Assistants, Attendees
(iv) Presentation, Clothing, Personal Effects, Associated Items
(v) Evidence of Medical Intervention
(vi) Postmortem Changes
(vii) Postmortem Imaging Studies
(viii) Identification
(ix) Evidence of Injury
(x) External Examination
(xi) Internal Examination
(xii) Histology samples Listing
(xiii) Microscopic Descriptions
(xiv) Toxicology Results, Laboratory Results, Ancillary Procedure Results
(xv) Pathologic Diagnoses
(xvi) Summary and Comments
(xvii) Cause of Death Statement.

IX. AUTOPSY ROOM HAZARDS

A. Commonly Acquired Infections

Micro-organisms have been classified into 4 hazard groups by the ACDP (Advisory Committee on Dangerous Pathogens) on the basis of pathogenicity to humans, risk to laboratory and autopsy workers, transmissibility to the community, and whether effective prophylaxis is available.

(1) **Group 1** [**category 1**]- An organism that is (i) most unlikely to cause human

disease

(2) **Group 2** [**category 2**]:

(i) may cause human disease

(ii) may be a hazard to laboratory workers, but lab exposure rarely produces infection

(iii) effective prophylaxis or treatment is usually available

(iv) unlikely to spread to the community.

(v) **Ex** – (a) adenoviruses, (b) herpesviruses (c) ortho and paramyxoviruses (d) picornaviruses (e) unconventional slow viruses.

(3) **Group 3** [**category 3**]:

(i) may cause severe human disease

(ii) serious hazard to laboratory workers

(iii) usually effective prophylaxis or treatment available

(iv) presents a risk of spread to the community.

(v) **Ex** – (a) Hantaviruses (b) HBV (c) **HIV** (d) Japanese B encephalitis (e) rabies (f) Rift Valley fever (g) **Tuberculosis** - Anatomists and autopsy pathologists usually got skin TB through cuts when doing autopsies on infected corpses [anatomist's wart, prosector's wart] (h) Yellow Fever.

(4) **Group 4** [**category 4**]:

(i) causes severe human disease

(ii) serious hazard to laboratory workers

(iii) usually no effective prophylaxis or treatment

(iv) high risk of spread to the community.

(v) **Ex** – (a) Viral hemorrhagic fevers - Hantavirus, Ebola virus (b) Russian spring-summer encephalitis (c) smallpox [if released for biowarfare]

B. High Risk Autopsy

The ***high risk autopsy*** [syn, hazardous autopsy] is defined as postmortem examination of a deceased person who has had, or is likely to have had, a serious infectious disease that can be transmitted to those present at the autopsy, thereby causing them serious illness and/or premature death.

Salient features:

(1) Bodies infected with group 3 and 4 organisms are high risk autopsies [eg HBV, HIV etc].

(2) Pathogens may be acquired by inhalation (of aerosols), ingestion, direct inoculation, entry though pre-existing breaks in the skin, and through the mucous membranes of the eyes, nose, and mouth. Any procedure that may result in infection through one of these routes constitutes a hazard.

(3) **HIV:**

(i) HIV remains viable in high concentrations in blood up to 2 months.

(ii) may be transmitted through cuts while doing autopsy, needle pricks during collection of blood and other body fluids, mucosal splashes, skin contact with superficial injuries.

(iii) HIV is present mostly in brain, colon, lungs and lymphoid tissue.

(4) **Airborne transmission** - does not occur with HIV and HBV.

1. Universal Work Precautions

(1) **Tagging** - All infected bodies should be wrapped and tied in double layer tough plastic bags. Both body and bag should have a red color tag mentioning "Biologically Hazardous"

(2) **Handling of bodies** - Workers who have external injuries, exudative lesions or weeping dermatitis should not handle bodies with infectious diseases [eg AIDS]

(3) **Handling sharp instruments:**

(i) Use either chain mail or Kevlar gloves.

(ii) If not available, wear double gloves and over the gloves, wrap leucoplast over fingers of the left hand [for a right handed person].

(iii) Most common digits pricked during autopsy in a right handed person are left index finger, left middle finger and left thumb in that order. These three digits should certainly be protected. (iv) If despite all precautions, there is a cut, the finger should immediately be dipped in bleach solution [sodium hypochlorite] and then washed with soap and water. No effort should be made to "milk" blood towards the cut.

(4) **Protective clothing** - should be worn. Consists of face mask, full sleeve overall [instead of simple surgical gowns], goggles, head cap, plastic visor, protective gloves [double gloves or chain mail], waterproof plastic apron and rubber gum boots of knee length with shoe covers

(5) **Handling specimens for lab examination** - Should be handled with gloved hands only

(6) **Treatment of used instruments** - Should be washed in glutaraldehyde solution

(i) Cidex Plus - is a high-level disinfectant [3.4% glutaraldehyde]. Instruments dipped for 20 min.

(ii) Cidex OPA [ortho-Phthalaldehyde] - 15 min. After treatment with glutaraldehyde, the instruments should be washed with soap, detergent and water, dried and then rinsed in methylated spirit and then air dried.

(7) **Soiled cotton and gauze** - collect in a double plastic bag"Send for incineration

(8) **Laundry material** - eg towels etc should be soaked in 1% bleach for 30 min"washed with detergent and hot water"autoclaved

(9) **Completion of autopsy** - take a complete bath with soap and water.

Fig. 5.8: Special cut resistant Kevlar gloves

X. COLLECTION OF SPECIMENS

A. Fresh Bodies [Undecomposed]

1. Anal swabs

For spermatozoa if anal intercourse is suspected

2. Bile

(1) **When** - For determining presence of poisons, particularly those excreted through the biliary system.

(2) **How** -

(i) **Method 1** - Passing a needle, attached to a syringe, into the lumen through gall bladder wall. Bile aspirated and collected into a container

(ii) **Method 2** - The gallbladder is dissected from the adjacent liver and bile is squeezed into a sterile container.

(iii) **Method 3** - Expression through cystic duct after evisceration.

3. Bite marks

Bite marks should be swabbed with a saline moistened cotton swab. Dry and place in a test tube and plug with cotton. Saliva of the assailant can reveal his blood group [in secretors]. DNA analysis can be done from mucosal cells in saliva.

4. Blood

Collection for toxicology is described here. Blood for microbiology"please see below. Collection of blood for toxicology in the living"**ch 31**.

a. General Guidelines

(1) **Site** -

(i) Best site to collect blood for toxicological analysis is a peripheral vessel like iliac, femoral or subclavian [being at periphery least affected by postmortem diffusion]

(2) **Quantity taken**"10-20 ml.

(3) **Preservatives** – please see below under the section “preservation of viscera in suspected poisoning”.

(4) **For grouping** - a piece of filter paper or clean cotton gauze is used to soak up some blood, dried and sent to lab.

b. Before autopsy

(1) **From femoral v** – withdraw 10-20 ml by a syringe.
(2) **From jugular or subclavian v** – use similar procedures.
(3) **From heart** – preferable not to take for toxicology because of postmortem diffusion [please see below], but is highly desirable for microbiological studies. For procedure, please see below.

c. During autopsy

Blood can be collected after removal of viscera. Sites to sample in .ing order of preference are

(1) **Femoral v**
(2) **Iliac v** – elevate leg to ease flow
(3) **Subclavian v** – Hold a small container under the cut end of subclavian v. and elevate arm.

i. Precautions

(1) **Scooping up:**
(i) Never take directly from heart or scoop out of thoracic or abdominal cavities [**please also see ch 31**].
(ii) Reasons -(a) Contamination - with gastric or intestinal contents, lymph, mucus, pus, serous fluid and urine [**ch 10**"Toxicological artifacts]. (b) PM glycogenolysis in the liver "-ed glucose in right atrial blood (c) Postmortem diffusion – please see below.
(2) If blood is being collected for volatile substances [chloroform, ether], use small glass bottles lined with aluminum foil. Fill up to top to avoid loss of volatile substances into headspace. Store at 4°C.
(3) Erroneous results can be obtained if
(i) clotted or
(ii) hemolyzed blood is taken.
(4) Massaging or milking limbs to - flow – Should not ideally be done as blood is being taken for toxicology. It can produce significant alterations in drug concentrations in the expressed blood [drug moving from muscles to blood

stream].

(5) Samples taken for alcohol estimations – Must contain and anticoagulant and preservative [prevents alcohol formation by microorganisms]- please see below, and also **ch 40.**

ii. Postmortem diffusion

Postmortem diffusion [syn PMD, postmortem redistribution, PMR] is the passive diffusion of poisons from stomach and intestines to adjacent organs ***after death*** giving spuriously high levels.

Salient features:

(1) Diffusion mainly occurs from stomach to the liver (especially the left lobe). For this reason, whenever a liver sample is taken, it must be taken from the middle of the right lobe, which is a relatively sequestered region of the liver. Also occurs in base of left lung, spleen, heart, pericardial fluid, aorta and IVC.

(2) **Causes** – (i) Barriers formed by living cell membranes breakdown after death, enabling molecules to move freely through tissues (ii) Severe trauma"gastric rupture"gastric contents may spill over in thoracic cavity"chances of PM diffusion are still more

(3) **MLI** – It is an important medicolegal considerations in all poison deaths [eg alcohol (**ch 40**)].

d. Blood stains

Very rarely only blood stains may be available as body has been disposed of. Toxicology is possible from stains [Please see **ch 29** for collection of blood stains].

5. Bone marrow

For diatoms [please see **ch 19**].

6. CSF

(1) by lumbar puncture on intact body before autopsy

(2) withdrawing fluid using a needle and syringe from cisterna magna. Pass needle through the atlanto-occipital membrane, just below the occiput, into the cistern.

(3) From lateral ventricles directly [after removal of skull].

7. Cyst fluid

Collect from hydatid cysts. Examine for echinococcus hooklets.

8. Feces

Collect 5-10 g from sigmoid colon or rectum. Examine for protozoa and helminths.

9. Lungs

(1) Mobilize lung

(2) Tie off main bronchus with a strong ligature

(3) Divide hilum

(4) Immediate put in a nylon bag, which should be heat sealed. Plastic (polythene) not suitable as it is permeable to volatile poisons.

10. Saliva

For spermatozoa if oral intercourse is suspected

11. Tissue

Small pieces of tissues collected into a thermos flask containing liquid nitrogen [for enzymes]

12. Urethral discharge

For microscopy and culture in case of STD

13. Urine

Urine can be obtained at two stages

(1) before the dissection - (a) catheterizing the urethra [usually for microbiological analysis] (b) syringing through the anterior abdominal wall directly above the pubic symphysis

(2) after the abdomen is opened -(a) incising bladder and collecting urine directly with a ladle. Such specimens can be useful for toxicology, but not for microbiology. [**Ch 31**"method of collection in the living].

14. Vaginal swabs

For spermatozoa

15. Vitreous humor

(1) Take 10 cc syringe and 20 gauge needle
(2) Insert in eye ball
(3) apply gentle suction. Aggressive removal or employment of vacuum tubes results in retinal tissue getting dislodged, causing contamination.
(4) All vitreous [About 1-5 mL] from each eye is removed, as conc near the retina are different as compared from centre of the globe. Volume is somewhat . in infants.
(5) If samples are too viscous"Centrifuge"use supernatant.

XI. PRESERVATION OF VISCERA IN CASES OF SUSPECTED POISONING

A. When Viscera should be Preserved?

Viscera should be preserved in following cases:

(1) **Advanced decomposition**
(2) **Cause of death not found** - after complete autopsy
(3) **Criminal abortions**
(4) **Death due to burns** [CN can be found in such cases. Please see chapter on burns]
(5) **Death of a machine operator** in a factory [may be under the influence of alcohol or drug]
(6) **Drug induced deaths** [penicillin etc]
(7) **History of addiction, alcoholism etc**
(8) **History of intoxication**
(9) **Road side vehicular accidents** [often alcohol or addiction drugs are found in the driver and occasionally in the victim]
(10) **Surgical and anesthetic deaths** [to rule out deaths due to anesthetic gases]
(11) **Suspicion of poisoning** - Death is suspected to be due to poisoning either by (i) police [eg when open drug sachets etc are found at scene of death. Request comes from IO] or (ii) doctor [peculiar smell from viscera]. For technique of collection, please see above.

B. What Viscera should be Preserved?

Following viscera must be preserved (**Fig. 5.9**)

(1) **Stomach and contents** – because most poisons are taken orally [of primary value for estimating the quantity ingested in acute poisonings]

(2) **Small intestine and contents** – same reason as above.

(3) **Liver** (200-300 g) – Because

(i) it is the major detoxifying poisons

(4) **Kidney** – One half of each as one kidney may be dysfunctional. Because it is the major excretory organ [Poison detected in liver and kidney is proof of absorption. Therefore it is important to keep the contents of stomach and intestines in separate bottles].

(5) **Blood** – 30 ml. Poison in blood is **proof of absorption**.

(6) **Urine** – 30 mL. Poison in urine is **proof of absorption and excretion**. [Poison found in urine – unless added with an evil intention - is proof of absorption and excretion]

(7) **Food or medicine** – Found at the scene of crime. Presence of poison in food or medicine is a very strong evidence of poisoning. **Table 2** gives a list of body fluids/viscera to be preserved in special cases.

Fig. 5.9: **(1)** Stomach and intestines tied at both ends, next to bottle in which they will be sent [Bottle no. 1] **(2)** One half of each kidney (top), one half of spleen (lower left) and 200-300 g of liver (lower right) are sealed in a different bottle [Bottle no. 2] **(3)** 30 mL of blood is sealed in a 3rd bottle **(4)** Sample of preservative [common salt] is sent in a 4th bottle. This picture shows all 4 bottles together with their contents **(5)** Seals and labels are put on all bottles **(6)** All bottles are then packed in a wooden container **(7 & 8)** Container is then sealed and handed over to police

Table 2: Viscera to be preserved according to poisoning

S.No	Body fluid/viscera to be preserved	Poisoning by
1.	Aborted fetus	Abortifacients
2.	Adipose tissue	Pesticides, thiopental, glutethimide, ethchlorvynol
3.	Alveolar air	Anesthetic deaths, inhalant abuse
4.	Amniotic fluid	Intra-amniotic drugs used in criminal abortion
5.	Bile	Barbiturates, cocaine, morphine, methadone, narcotics, glutethimide [please also see **ch 31**"Drugs excreted in human bile]
6.	Blood (collect 30 ml in screw capped bottles)	**In all cases. But especially in** Alcohol, cyanide, carbon monoxide, barbiturates and other depressants, tranquilizers.
7.	Blood clots	May be submitted if fluid blood not available. But blood clots have a high RBC:serum ratio, which may affect determinations [please see above under "blood"]
8.	Bone	Pesticides, heavy metals, radioactive poisons (radium)
9.	Brain	Anaesthetics, alcohol, barbiturates, CO, cyanides, opiates, strychnine, volatile poisons
10.	Breast milk	Poisons excreted in milk [opioids, thallium]
11.	CSF	Alcohol
12.	Decomposition fluid [from pleural and other cavities]	In all cases of putrefaction
13.	Extradural hematoma	Please see SDH below
14.	Fat (10 g from abdominal wall, mesentery or perinephric region)	Pesticides, insecticides, especially organochlorine
15.	Feces	GIT poisons
16.	Fingernails	Chronic metal poisoning
17.	Hair	Chronic metal poisoning
18.	Heart	Strychnine, digitalis
19.	Injection site (take a specimen of skin around the injection site, and include s/c fat and muscle)	Injectable drugs, snake bite, scorpion bite, bites by other venomous animals
20.	Insects [eg maggots]	Antidepressants, Barbiturates, Benzodiazepines, Cocaine, Opiates [Method of collection – **ch 9**"How to collect maggots]
21.	Intestines and contents	**In all cases. But especially in** drug packers or "mules" [Smugglers of packets of drugs hidden in intestines]
22.	Intracranial hemorrhages	Please see SDH below
23.	Kidney	**In all cases. But especially in** metals, sulfonamides
24.	Liver	**In all cases. But especially in** high levels of toxic material or biometabolites
25.	Lung	Alcohol, chloroform, HCN, heroin (IV) , inhalant abuse,
26.	Meconium	Fetal exposure to poisons, neonatal drug addiction **[ch 46]**
27.	Milk	Please see "Breast milk" above
28.	Muscle, skeletal (especially from	In all cases of putrefaction, embalmed bodies

30.	Nasal swabs	Cocaine (it is often snorted), inhalant abuse
31.	Pericardial fluid	In all cases in which blood is usually taken, but not possible because of putrefaction or otherwise
32.	Placenta	Abortifacients
33.	Scalp hair	Heavy metals
34.	Skin with underlying tissues	Corrosives, injected poisons, snake bite, bites by animals
35.	Small intestine (with contents)	**In all cases**
36.	Spinal cord	Gelsemine, strychnine
37.	Spleen	CO, CN (these bind to Hb, and may be detected in spleen as it is a good storage place for blood)
38.	Stomach and contents	**In all cases. But especially in** Tablets, capsules.
39.	Subdural Hemorrhage (SDH)	Alcohol or any other poison, especially if survival has been for days or weeks. Intracranial hemorrhages [EDH, SDH etc] are produced at the time of trauma [vehicular accident, violent fight, fall from height etc], and contain poison, if it is present in blood at that time. In delayed deaths, poison may have disappeared from blood (due to metabolism), but in ICH, it remains unmetabolized due to its sequestration from mainstream blood. Analysis is however subject to same weaknesses as those of blood clots [please see above].
40.	Sweat [collected by wiping the skin surface with cotton]	Drugs of abuse
41.	Synovial fluid (from joints such as the knee)	Alcohol. Useful in advanced decomposition
42.	Urine	**In all cases. But especially in** diabetes
43.	Uterus, appendages, upper part of vagina	Criminal abortion. If sticks and foreign bodies are found, preserve them separately.
44.	Vitreous humor	Alcohol, chloroform, embalmed bodies

C. Preservatives

(1) **Saturated solution of common salt** – Most common preservative used in India. Not to be used in corrosive acids (use rectified spirit instead)

(2) **Rectified spirit** – except in cases of (i) acetic acid (ii) alcohol (iii) chloral hydrate (iv) chloroform (v) ether (vi) formaldehyde (vii) formic acid (viii) kerosene (ix) paraldehyde (x) phenol and (xi) phosphorus. These are soluble in rectified spirit and may be leeched away from tissues.

(3) **Preservatives for blood** – **(a)** For **10 mL** of blood, use 100 mg **sodium fluoride** (enzyme inhibitor) + 30 mg **potassium oxalate** (anticoagulant)"shake thoroughly (e) **Alternative preservatives for alcohol –** For 10 mL of blood [or urine], 50 mg of **phenyl mercuric nitrate** or **sodium azide.**

(4) **Preservatives for Urine** – Urine should be taken in addition to blood. Preservatives used (a) an equal quantity of saturated solution of common salt (b) equal quantity of rectified spirit (c) some fine grains of thymol (d) one gm of sodium benzoate (e) 5 mL concentrated HCl (the last two for about 200-500

ml of urine). **Some important pointers:** (1) ***Formalin*** should never be used as a preservative" Makes the extraction of poisons difficult, especially non-volatile organic compounds (2) **Samples of preservative** - should be sent separately to rule out contamination [**ch 10**" Toxicological artifacts] (3) **Preservatives for virological analysis** - (a) **Frozen at -90°C** in liquid nitrogen flasks (b) If this facility not available - **50% glycerol saline** [glycerol in buffered saline] (4) ***Heparin and EDTA*** should never be used as anticoagulants. They interfere with the detection of some poisons (eg methanol).

Conditions when preservative is not necessary

(1) When viscera can be analyzed **within 24 hours**
(2) If sample can be kept in a **refrigerator** or **ice box**
(3) For **hair, nails, bones**
(4) **lungs** for detecting inhaled poisons.

6. Examination of Decomposed Bodies, Mutilated Bodies, Fragmentary Remains and Bones

I. AUTOPSY ON DECOMPOSED BODIES

All human remains must be examined, because even the most highly decomposed bodies provide some information.

II. AUTOPSY OF MUTILATED BODIES AND FRAGMENTARY REMAINS

Forensic anthropology is the application of physical anthropology for the

purposes of law and administration of justice.

A. Questions to be Answered

Following questions are asked by police in relation to mutilated bodies or fragmentary remains.

1. Human or animal

(1) If head, trunk, limbs available – very easy

(2) If only muscles etc are available – perform precipitin test with **antihuman globulin**.

2. One or more bodies

(i) Anatomically fit together all parts and count bodies (ii) If only parts are available, apply **logical count** eg (a) two limbs of the same side"two persons (b) two opposite limbs of different lengths"2 persons (c) two opposite limbs of grossly different weights"2 persons (d) two limbs of different colors"2 persons (e) two limbs of different DNA profiles"2 persons (f) Three ears"definitely more than one person, but may be 2 or even 3. Confirm sides. Perform DNA (g) multiple fingers"sort according to side and count. If unsure, perform DNA.

3. Sex

(1) **If head, trunk, limbs available** – very easy. Look for distribution of hair, development of breasts, striae gravidarum

(2) **If not available, look for non gravid uterus and prostate** – they resist putrefaction for long.

(3) DNA based methods – **ch 3**.

4. Age

Can be determined from (1) General development (2) eruption of teeth (3) ossification centers (4) obliterations of the sutures of skull [**ch 3**].

5. Stature

In mutilated bodies, following pointers may be helpful

(1) If only torso available - (a) Arms outstretched"Distance between tips of middle fingers of both hands is equal to height (b) Length from vertex to

symphysis pubis is roughly half of total height [if the age is >14 years. If age <14 y, trunk is longer than the lower limbs] (c) Length from sternal notch to symphysis pubis # 3.3 = Height

(2) If only lower half of body available – As above. Measure height from symphysis pubis to heel and multiply by 2

(3) If only vertebral column available – Length of vertebral column # 100/35 = Height

(4) If only one arm available – Multiply length of arm by 2. Add 30 cm for two clavicles. Add 4 cm for sternum. Gives height

(5) If only part of one arm available – Length of forearm from tip of olecranon process to tip of middle finger # 19/5 = Height

(6) If only head available – Height of head from vertex till tip of chin # 7 = Height

(7) If only palm available – 34.5 + (5.77 # Palm Length in cm) + (2.7 x Palm Breadth in cm) = Height in cm

(8) If only middle finger (of hand) available – Length of middle finger # 21.5 = Height

(9) If only index and ring fingers are available

(10) If only foot available – Foot length # 100/15 = Height

(11) Can be measured from long bones too (please see below)

6. Identity

Identity can be determined from (1) clothing (2) deformities (3) fingerprints (4) hair (5) moles (6) scars (7) superimposition (8) tattoos (9) teeth etc. Please refer to ch 3 for details.

7. Manner of separation of parts

Margins of the separated parts must be examined to determine if they are (1) cleanly cut (2) disarticulated at the joints (3) gnawed by animals (4) hacked (5) lacerated or (6) sawn.

8. Time since death

From condition of parts. For details, please see ch 9

9. Cause of death

Look for signs incompatible with life, eg

(1) cut or rupture of a large blood vessel, [eg jugular vein]

(2) evidence of fatal injury such as cuts and stabs to a vital organ [eg heart or brain]

(3) fractures [esp of skull, ribs, cervical vertebrae, hyoid etc)

(4) marks of burning

(5) If body recovered from a body of water, look for diatoms

(6) Perform toxicology from available tissues. Metallic poisons can be detected for years in hair, nails and bones

(7) Signs of natural disease [eg blocked coronaries]

(8) Emaciated body might point to death by starvation. For details, please see ch. 20.

10. Injuries antemortem or postmortem

Examine margins of parts for evidence of vital reaction. Gross examination, histopathological, enzyme histochemistry and immunohistochemistry is useful. [For details please. see ch 11 on injuries].

11. Miscellaneous info

Handedness of the individual – can be determined by measuring muscle mass in both upper and lower limbs on each side. Muscle mass is -er on the dominant side. Right dominance in upper limb may not necessarily correspond with right dominance in left limb and vice-versa.

III. EXAMINATION OF BONES

Forensic osteology is the application of knowledge of bones for the purposes of law and administration of justice.

A. Questions to be answered

Following questions are usually asked by police in relation to bones

1. Whether these are bones or only rubble

(1) **Naked eye inspection** - shows peculiarities of bones, eg trochanters,

tuberosities, condyles etc, which would not be visible in rubble

(2) **Density of bone** - is different from that of rubble [sp gr of bone = 2; rubble lesser or greater, but rarely 2]

(3) **Microscopy**

(4) **Precipitin test** - possible when bones are within 15 y; plasma proteins are extractable up to this period [details in **ch 29**]. Not possible with burnt or cremated bones.

2. Human or animal

(1) **Comparative anatomy:**

(i) differentiation is done by studying details which are different in man and animals.

(ii) Easy when entire bones are available, but difficult when bones are burnt, fragmented or otherwise mutilated.

(iii) Also difficult with very small bones eg metacarpals, metatarsals, ossicles, phalanges etc.

(iv) Hand and foot bones of bear resemble human bones greatly.

(v) Virtually impossible with sesamoid bones.

(2) **Precipitin test**

(3) **Microscopy** – but bones of great apes cannot be distinguished

(4) **Chemical analysis** - of bone ash.

Fig. 6.1: **(1)** Some animal bones may look remarkably like human bones. For instance human hand bones [L] look remarkably similar to bear paw [R] **(2)** Animal bone submitted to the author by police. It was among several human bones found lying below a tree in an isolated park. The anatomy is not consistent with any human bone. Species can be confirmed by precipitin test.

3. One or more individuals

Look for incompatibilities, which will indicate more than one individual (i) Age – bones belonging to persons of different ages, indicate more than one person. (ii) Sex– eg if only two bones are available, say skull and pelvis, they are compatible with one person. But one bone of male and another of female

indicate two persons. (iii) Race – bones of two different races, indicate at least 2 different persons (iv) Stature - determined from two bones (v) Side – eg two femurs are normally compatible with one person. But if they are of same side"indicate 2 persons. (vi) Number – 2 skulls indicate two persons. 3 femurs indicate at least two persons, but if all are of same side"3 persons and so on. (vii) Articulation – normally one femur and tibia of same side indicate one person, but if they cannot be articulated together, they indicate different persons. (viii) State of putrefaction – some fresh and some very old indicate different individuals (ix) Other studies - (a) bone weight (b) density studies (c) neutron activation (d) radiography (e) serological testing (f) trace element analysis.

4. Stature

a. If complete skeleton is found

(1) **By articulation** - Articulate (skeleton. Add 2.5 cm for soft tissues of scalp, vertebral bodies and heels.

(2) **By Weight** – **Matiegka** has given a formula [**Matiegka's formula**] for calculation of skeletal weight from stature and vice-versa. **Skeletal weight (g) = 1.2 # D^2 # H**. Here "H" is standing height in centimeters [this can be calculated by articulating skeleton], and "D^2" is the average of the squares of four measurements–transverse dimension of the lower end of the (i) humerus (ii) forearm (iii) Femur, and (iv) ankle.

b. If incomplete skeleton is found, but individual bones not fragmented

(1) Individual bones are measured by osteometric board [**Fig 6.2**] and multiplication factors applied.

(2) **Osteometric Board:**

(i) Hepburn board modified by Trevor is most commonly used

(ii) Has a rectangular base [1]

(iii) A ruler [2] is fixed along one of its long sides

(iv) Upright boards - One is fixed at one side [3]; second slides along the board [4]

(v) Procedure – one end of the bone is placed against the fixed board; the moveable board is brought up to the other end; Ruler then shows the length of the bone.

(3) **Long bones** - Multiplication factors for all six long bones [femur, tibia,

fibula, humerus, ulna and radius]

(i) provided by different workers (a) Breitinger [1937] (b) Dupertuis and Hadden's [1951] (c) Muñoz [2001] (d) Pearson - Oldest. Developed in **1899** by the British anthropologist **Karl Pearson** [1857 – 1936] (e) Telkkä [1950] (f) Trotter and Glesser [1952].

(ii) A simple rule of thumb is that **humerus** is **20%**, **tibia 22%**, **femur 27%** and the **spine 35%** of individual's height in life.

(iii) If long bones are not available - Articular length of metacarpals may be used.

c. If fragmented bones are found

Steele and McKern's formula is used.

d. If only skull available

Height of skull # 8.

e. Stature from teeth

Please see ch 3 [forensic odontology].

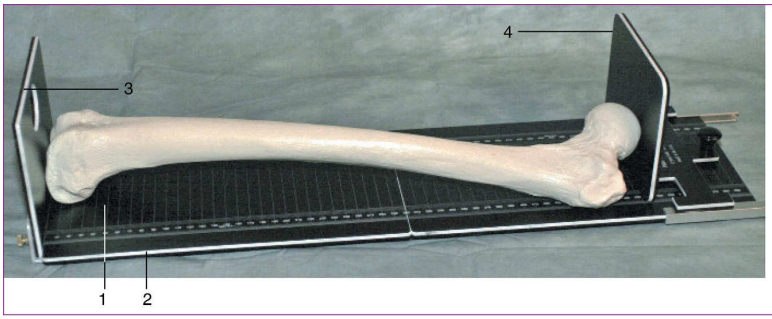


Fig. 6.2: Osteometric Board.
(1) Rectangular base
(2) Ruler
(3) Fixed upright board
(4) Moveable upright board. It can be moved in either direction.

5. Time since death

(1) **Skeletal changes depend upon**

(i) Accessibility to scavengers [carnivorous animals and insects]

(ii) Burial or non burial

(iii) Climatic conditions [temp, humidity]

(iv) Soil type.

(2) **Determination of Time since death:**

(i) up to 6 months - (a) Heavy [because of the preservation of the organic stroma] (b) Greasy to touch. Greasiness persists for several years (c) bad odor (d) color – pale ivory, if directly placed under sunlight [bleaching by sun] (e) Sawing – (I) Bone would be hard to saw (especially the shafts of limb bones such as the femur) (II) uniform through the whole thickness. (III) Smell of burning organic tissue if vigorous sawing produces heat. In an older bone (**>5 y**), the loss of the collagenous stroma will lighten the bone and make it easier to cut. (f) Nitrogen content - 4.5%. It progressively .es with time

(ii) 6 m – 1 y - (a) Soft tissue remnants [tendon, ligament tags] adherent, especially around the joint ends. (b) Periosteum visible as fibrous material closely adherent to the shaft surface. (c) Cartilage present on articular surfaces.

(iii) 1y - 5y - Microradiography and electron microscopy reveal vacuoles in compact bone left in soil for 5 y.

(iv) 50-100 y - (a) Nitrogen content – between 4-4.5%

(v) 200 y – UV fluorescence of cut surface is useful up to 200 y [**Fig 6.3**].

(vi) >350 y - (a) Nitrogen content ≤2.5%.

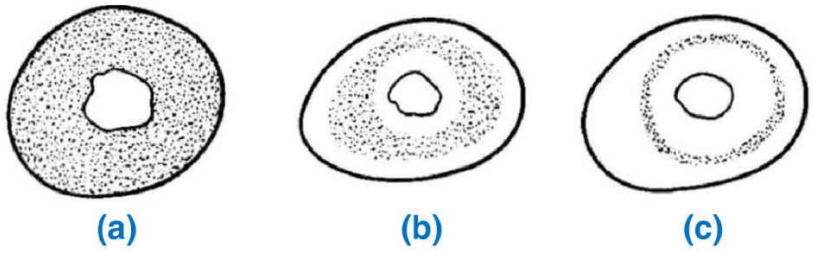


Fig. 6.3: Fluorescence of bone in UV light to determine TSD. A freshly sawn cross-section of a dense bone, eg femoral shaft is preferable. Up to several decades after death, the entire cut surface fluoresces a bluish color **(a)**. From 3-80 years, greatly depending upon environmental conditions, the outer zone and the zone around the marrow cavity progressively lose fluorescence **(b)**. After a century or more, the residual fluorescence contracts to a narrow central band **(c)**. It vanishes completely at about 200 y.

6. Signs of pathology

a. Gross examination

(1) **Anemia** - Cribra orbitalia**,** a fine porosity visible on the ceiling of the orbits

is a sign of severe anemia.

(2) **Syphilis:**

(i) Characteristic tibial lesions (“sabre shins”) are indicative of treponemal infection.

(ii) purify IgG from powdered bone"use ELISA" reactivity with T. pallidum antigen.

(3) Stones – in kidneys, gall bladder

(4) **Tuberculosis** – signs of Pott’s disease, Other bone anomalies.

b. Biochemical analysis

Heavy metal poisoning, eg lead

7. Signs of Trauma

Examine ends of long bones - Note if bones are (i) Cut by sharp instruments (ii) Gnawed by animals – (a) Crabs, fish, turtle – leave shallow marks (b) Deer and sheep – hold bones with back teeth"fork shaped or double pronged fragments (c) Dogs, fox and wolves - cause splintering of bone (d) Rodents – produce parallel “chisel marks” due to gnawing (iii) Hacked (iv) Sawn.

Memory Aid 1: Remembering multiplication formulae

Start with the number 36. Go on adding 8 and generate 5 numbers as shown below

36
Add 8
44
Add 8
52
Add 8
60
Add 8
68

Now we have 5 numbers: 36, 44, 52, 60, 64. Write them in a straight column. Modify third and fifth numbers as shown and then put a decimal point in between the two numerals.

Original number	Modified number	Number after putting decimal point
36		3.6
44		4.4
52	53	5.3
60		6.0
68	64	6.4

We get five numbers: 3.6, 4.4, 5.3, 6.0, and 6.4. These are the multiplication factors. Now we have to assign bones to them. Femur is the largest bone, so it must have the smallest multiplication factor→3.6 is assigned to it. Arrange smaller bones in order of their ↓ing length; and assign larger numbers.

Bone	Multiplication factor	Humerus	5.3
Femur	3.6	Ulna	6.0
Tibia and fibula	4.4	Radius	6.4

8. Cause of death

Can be ascertained in some selected cases only

(1) **Bullets** - found lodged in crucial bones, eg skull, vertebrae. Even in bullets are found in non-vital bones eg femur, one can be fairly certain that the victim was exposed to a volley of bullets.

(2) **Burns** - If bones are burnt, possibly the victim was burnt to death. Very difficult to know if body was burnt after killing.

(3) **Deep cuts** - on crucial bones eg ribs on left side overlying heart indicating the victim was stabbed. Cuts on lumbar vertebrae indicate abdominal stabbing. Type of weapon can often be made out by studying cuts.

(4) **Diatoms** - can indicate drowning

(5) **Diseases of bones** - eg malignancies

(6) **Fractures** - of bones containing vital structures eg skull, vertebrae, multiple ribs, hyoid bone

(7) **Poisons** - Metallic poisons can be detected in bones, even after burning.

9. Miscellaneous info

(1) Bones discarded after anatomical dissection – Some stains are seen in the nutrient canals.

(2) **Harris lines** - [syn, growth arrest lines, HLs Lines of Arrested Growth (LAGs)] – First described by **Harris** in **1933** are transverse radiopaque lines of -ed bone density that are only visible by radiographs or in cross-sections.

(i) Seen in most bones, but most frequently in distal half of tibia.

(ii) Cause - Episodes of temporary arrest of longitudinal growth caused by stress [eg disease, illness, malnutrition, psychogenic stress, trauma]. Can be used to predict these stresses in the individual.

(3) **Natural disintegration of body** – articular surfaces are smooth.

(4) **Stride length** - Femur length has a positive relationship with stride length. Opinion can be formulated regarding person's gait, biomechanics, movement, and posture. If video or closed-circuit television footages from video surveillance systems are available, these can be linked to the deceased whose femur is being examined.

B. MLI (of mutilated bodies and bones)

(1) **Cremation litigation** – Mishandling of cremated remains may result in doubts and litigation. There are reports of families receiving two sets of cremains each purported to be of their relative.

(2) Human rights investigations
(3) Mass disasters
(4) separation of recent from ancient remains
(5) All other questions answered above may be raised in one or other civil or criminal case.

7. Exhumation

Exhumation (Latin ex, out of; humus, ground) [syn ***disinterment***] is authorized digging out of the coffin of a dead person from his grave, in order to establish his cause of death, or to decide upon some other relevant fact, such as the person's identity.

Salient features:

(1) Incidence - Exhumation is rarely done in India, because the bodies are mainly disposed off by cremation. Only certain communities bury their dead.
(2) **Time limit** – No time limit in India. Can be done any number of years after burial.

I. REASONS FOR EXHUMATION

A. Criminal

1. Determination of cause of death

(i) Homicide (ii) suspected homicide (iii) homicide disguised as natural, accidental or suicidal death (iv) suspicious poisoning (v) death as a result of criminal abortion (vi) criminal negligence.

2. Retrieving evidence

(i) some vital object which may throw more light on the case, such as bullet from the dead body (ii) poison from body.

B. Civil

1. Identification

Identification of the deceased for

(1) burial of the wrong body inadvertently or by fraud,
(2) settling of inheritance
(3) survivorship claims [disputed identity].

2. Others

(1) Accident death claim
(2) Civil negligence
(3) Insurance
(4) Workmen's compensation claim [**ch 2**].
(5) When the **site of a graveyard is moved** e.g. for redevelopment.
(6) When the relatives wish to **relocate the grave** for some reason.

II. AUTHORIZATION

(1) **Magistrate** – In India, the body can be exhumed only upon the written order from an executive or judicial magistrate **[S.176(3), CrPC]**. Police ***CAN NOT*** order exhumation.
(2) **Coroner** - In countries with coroner system [eg UK].
(3) **Medical examiner** – in USA.

III. PROCEDURE

(1) **Information to relatives** – Wherever practicable, the magistrate should inform the relatives of the deceased whose names and addresses are known, and shall allow them to remain present at the inquiry **[S.176(4), CrPC] [ch 1]**.
(2) **Officials present at the time of exhumation:** (i) magistrate (ii) medical officer (iii) police officer
(3) **Identification of grave:** (i) Done in association with local priest. Identifying features are (a) Burial plot location (b) Grave marker (c) Headstone (ii) Note distance of grave from some permanent objects eg a prominent grave, fence,

gate, road, rocks or trees.

(4) **Time** – Early in the morning. Advantages (i) Exhumation, postmortem and reburial can be conducted on the same day (ii) Less curious onlookers, and less police presence is necessary. However if still onlookers are there, areas should be screened off (iii) Presence of natural light

(5) **Removal of earth:** (i) Earth should be removed in layers [10-15 cm at a time]. Note should be made about the condition of soil, water content and vegetable growth. (ii) Burial pit should be opened up to **30 cm** on all sides of the body. This enables the investigator to stand at side of grave and clear the body [please see below].

(6) **Measurements** – Measure the depth of grave from the surface to the skull as well as surface to feet [gives inclination of body]

(7) **Before recovery of body:** (i) Photography - With the body in situ photograph it"Clear the body surfaces with the use of a soft brush or whisk broom"Photograph the body in situ again (ii) Drawing – of the grave as well as body or skeleton should be made now. This enables various measurements to be recorded on the drawing, eg height of body, extent of flexion or extension of limbs etc, grave's distance from notable landmarks etc.

(8) **Recovery of body:** (i) If body is with a coffin – Note condition of coffin; whether eaten up by worms, sodden, soggy, damaged, worn out, peeling on the outside etc. Whether dry or filled up with water. If filled up with water, remove as much as possible, preserve, label and send for toxicology. Now lower a plank or strong PVC sheet to the level of earth on which coffin rests"Gently lift coffin on to the plank or PVC sheet"Remove from grave"Transport entire coffin to mortuary (ii) If body is without a coffin - (a) If decomposition is not advanced – Use same procedure to remove body as above. Body is gently lifted and placed on to the plank or PVC sheet (b) If decomposition is advanced – There is possibility of body disintegrating and breaking away as soon as it is attempted to be lifted. So an alternative procedure is followed. Dig down beside and beneath the body"Insert a wooden plank under the body"Lift up the wooden plank. (c) If body is skeletonized – Use same procedure as above [advanced decomposition]. In addition, after removal of skeleton, remove earth from below and sides of the body and sift it in a finely-meshed screen. This can often recover **smaller bones** [carpals and tarsals , epicondyles, epiphyses, phalanges, sesamoid

bones, teeth, tuberosities] as well as **objects** [bullets, gall stones, kidney stones, medical prosthesis eg nails, plates, tips of knives and arrows etc].

(9) **Identification** – Body should now be identified by close relatives and friends either from features [if intact] or from clothes, objects kept within the grave, appearance of coffin etc.

(10) **Note** – condition of body, burial clothes

(11) **In suspected poisoning:** (i) Collect all personal effects, clothings, nails, hair, bones etc and send for toxicological analysis (ii) Collect samples of earth [about 1kg] that are in actual contact with body and also from above, below and each side of the body. Also from a site well away from the grave [**Fig 7.1**]. (iii) Also preserve samples of burial clothes and coffin to preclude the possibility of contamination from these sources.

(12) First autopsy or second autopsy – please see **ch 5**.

Fig. 7.1: **(1)** Digging the grave is done early in the morning in the presence of doctors, executive magistrate and police. **(2)** Measuring the depth of grave **(3)** Collection of soil from above the coffin for toxicology. Later it would be collected from below and from the sides of the coffin too, as well as from some distance away from coffin as mentioned in the text **(4)** After the grave has been dug up to the level of coffin, it is safely lifted up with the help of ropes.

8. Thanatology, Death and its Causes

I. INTRODUCTION

A. Thanatology

Thanatology (Gk ***Thanatos***, the God of Death) is that branch of science that studies death in all its aspects.

II. STAGES OF DEATH

A. Somatic Death

Somatic death is the permanent, irreversible death of an organism as a whole. Historically the concept of when somatic death occurs has kept changing. Traditionally the concept was that it occurs when there is irreversible cessation of heart, lungs and brain [**Bichat's criteria** - please see below]. The concept was gradually changed to **brain death** and eventually to **brain stem death**. With modern technology, bizarre situations can arise. Circulation and respiration of a brain stem dead individual can be maintained artificially. By classical criteria, such an individual would be somatically alive, which is an absurd situation.

Salient features:

(1) According to modern concept, somatic death coincides with the death of brain stem. It involves **complete and irreversible stoppage** of vital brain stem functions.

(2) Legally a person is dead after somatic death.

(3) It is death as a common man understands it.

B. Molecular Death

Molecular death is the death of all individual cells within the body. All biochemical (molecular) activity within the cells comes to a stop. It comes **2-3 hours after somatic death**.

1. Demonstration of supravital period in the dead

Electrical excitability of muscles - The *Orbicularis oculi* muscle is tested by inserting needle electrodes into the upper eyelid at a distance of **1.5–2 cm**, and at a depth of **0.5–0.1 cm**. The *Orbicularis oris* muscle is tested by inserting similar

electrodes at **1 cm** distance from the mouth angles, at same depth as above [**0.5–1 cm**]. Muscles are then stimulated using constant current rectangular impulses of 10 mA at 50 cycles/s. The muscular reaction is graded **according to the intensity and spread of contraction**. Different intensities can be related to different periods of death.

C. Antemortem, Postmortem and Perimortem Injuries

(1) **Antemortem injuries** - Injuries that occur during life.
(2) **Postmortem injuries -** Injuries that occur after molecular death.
(3) ***Perimortem (around death) injuries*** - injuries caused ***between somatic and molecular death*** [supravital period].
(4) **Agonal period** – is the period just before death.

III. SUSPENDED ANIMATION

Suspended animation [syn **apparent death**] is a state where the heartbeat and the respiration of a person become so weak, that they cannot be detected by routine clinical methods. The person thus appears clinically dead but he is not since the brain stem is functioning.

Salient features:

(1) The **metabolic rate** of life is so reduced that the oxygen requirement of individual cells is satisfied through oxygen dissolved in body fluids.
(2) Testing for **brain stem reflexes** [please see below] would establish that the victim is not dead.

A. Causes

1. Voluntarily induced

By yogis during samadhi.

2. Accidental

Pl see **memory aid 1**.

Memory Aid 1: Causes of suspended animation

(1) **A**nesthesia, (2) **B**arbiturates, (3) **C**oma (4) **C**holera (5) **C**oncussion, (6) **D**eep shock (7) **D**rowning, (8) **E**lectrocution, (9) **F**rozen state (Hypothermia)

(10) yo**G** is [voluntarily induced, but may be remembered by the student here as a memory aid], (11) **H**anging

(12) **H**eat stroke, (13) **H**ypothermia, (14) **H**ysteria

(15) **I**llnesses [prolonged], (16) **I**nfections [Cholera, Typhoid], JKL, (17) **M**esmeric trance, (18) **N**ewborn, (19) **O**piates.

B. ML Importance

(1) **Premature burial or cremation** - Before 19th c, when modern methods of diagnosis of death were not available, some people were undoubtedly buried as dead, when they were actually in a state of **suspended animation**. In many cases, the "dead" person awoke just before he was about to be buried, but in many others they were actually buried. (i) **Sommer's movements** - Several coffins dug up years later due to some reason, showed that the limbs of dead person were in different position, than when he was buried [In **1833**, this was attributed by **Sommer** to contraction of muscles after death due to rigor mortis. These were the so-called **Sommer's movements**].

(2) **Postmortem** – Persons in suspended animation have been brought for PM examination. This reflects the need for more careful diagnosis of death in emergency wards.

IV. THE MOMENT OF DEATH

The ***moment of death*** refers to the exact moment, when the person dies. The concept of the moment of death has radically changed over the years.

A. Classical Criteria of Death

Criteria of death refer to criteria and tests which confirm death.

Salient features:

(1) **Bichat's criteria** - Before the era of heart transplantation, the criteria of death were the so-called **tripod of life**, advanced by French physician **Marie Bichat** (1771 –1802). Also known as **Bichat's tripod of life**. Bichat believed that life could be compared to a tripod (**Fig 8.1**), with the three legs representing brain, heart and lungs. If any leg perished, very soon (i.e. within minutes) all legs would perish, and the tripod of life would fall (death).

(2) ***Atria mortis*** [Latin, Gateways of death, death's portals of entry]- Since death could "enter" only by stoppage of these organs, each was called an "atrium mortis" [please, atria mortis].

(3) The rule of the thumb at that time was: breathing stops ***within 20 seconds*** of cardiac arrest, and heart stops ***within 20 minutes*** of stoppage of breathing. Brain would die within ***3 minutes*** of the stoppage of any of the other two. Tests were developed to determine the death of each organ.

Fig. 8.1: Representation of the concept of Bichat's tripod of life

1. Tests for circulation

Stethoscope was invented in **1819**. But it was **Bochet** in **1846** who suggested cardiac auscultation for 2–3 min to diagnose death. Because in suspended animation the stethoscope could not detect heart sounds, it could not gain wide acceptance.

a. Magnus test [Syn, ligature test] [Fig 8.2]

Suggested by an Italian **Hugo Magnus** in **1872**. The test consisted of tying a string around one of the fingers of the supposedly dead man. The pressure applied had to be just above that in veins, but just below that in arteries. This would occlude the veins but not the arteries. If the circulation was still maintained the finger distal to the ligature would swell. Although stethoscope had been discovered by Laënnec in 1816, it was still very basic in design, and it could not be used reliably to listen to heart sounds, especially if it was beating very feebly. Magnus test was in vogue for some time, but is now **completely outdated**.

Fig. 8.2: Magnus test used in a live person. Note the swelling and redness of index finger indicating active blood flow. Finger would behave in a similar way in a person in suspended animation, but not in a dead person.

b. Icard's test

Suggested by **Séverin Icard** of France in **1895**. It consisted of **subcutaneous injection of a compound** [**Fluorescin** with **sodium bicarbonate**; pl co-relate with Fluorescein test (**ch 29**). **Fluorescin** is a colorless amorphous substance; oxidation by heme produces **fluorescein** which is fluorescent. **fluorescein** also causes urine to fluoresce in ethylene glycol poisoning (**ch 40**)]. If the circulation was still going on, the whole skin would turn a **vivid yellow**. If the person was already dead, no such change would be seen. This test is now completely outdated.

c. Diaphanous test (transillumination test)

Hand with closed fingers was held against a strong source of light. During life, it appears red and translucent. After death, it is yellow and opaque.

d. Finger nail test

Pressure applied on nail bed. During life normally nail bed is red; on pressure it turns pale, and on release of pressure becomes red again. After death remains pale throughout.

e. Other tests

(1) **Cutting of small artery**"Jerky flow during life. Continuous or no flow after death.

(2) **Heat test - Application of heat** [heated matchstick; hot melted wax]"Blisters appear during life, not after death.

2. Tests for respiration

a. Winslow's test

Winslow's test was suggested by the Danish physician **Jacques-Bénigne Winslow (1669-1760)**. A small pot containing water, milk or mercury was kept just below xiphisternum. Slightest motion of the chest-walls, such as occurs even with the faintest respirations, is indicated by the rippling on the surface of the water, or by the movements of a reflected image (such as a candle-flame) from the surface of the mercury. The absence of such movements indicates the absence of any motion of the chest-walls, and the probable stoppage, therefore, of respiration.

b. Mirror test

Mirror held in front of mouth and nostrils. Becomes dim due to water vapor in

breath

c. Feather test

Feather placed in front of nostrils or mouth. Movement during life. No movement after death. Sometimes soap bubbles or a candle was held to the nose to detect respiration.

d. Others

Submerging the body in water to detect bubbles produced by respiration.

3. Tests for brain function

Tests for "sensibility"

(1) Blowing a strong stimulant such as hellebore or mustard into the nose
(2) inserting a sharp instrument under the nail
(3) cauterization or incisions
(4) scalding with hot water or oil
(5) trumpeting or loud noises.

4. Miscellaneous tests

(1) **Ripault sign** - External pressure to the eye causes permanent (vs. temporary) distortion of the roundness of the pupillary aperture.
(2) **X-ray fluoroscopy** -to search for organ movement was suggested in 1910. All above tests are now completely outdated.

B. Brain Death

With the advent of heart transplant in 1967, the definition of death needed to be revised and made more logical (Table 1). Transplant surgeons needed a "live" and "beating" heart from a "dead" donor (beating heart donor). This was not possible with the **old illogical definition**, where all three vital organs needed to perish before death could be pronounced. Advent of modern resuscitative measures could also keep heart and lungs "alive" for long intervals even after the brain of the individual was dead.

Harvard criteria of Brain Death (1968)

In **1968** the **Harvard Criteria of the moment of death** [Table 1] were developed. Laid stress on determining the activity of only the brain to determine

death [**Fig 8.4**]. The criteria suggested that the activity of the heart and lung are important not by themselves, but as a sign of brain activity.

Table 1: Harvard Criteria of the moment of death (Brain Death)

1. Unreceptivity and unresponsivity
2. No movements or breathing
3. No reflexes
4. Flat EEG
5. All these criteria should be repeated after 24 hours

a. Unreceptivity and unresponsivity

The patient is totally unaware of the externally applied stimuli (**unreceptivity**). Even the most intensely painful stimuli evoke no vocal or other response, not even a groan, withdrawal of a limb, or quickening of respiration (**unresponsivity**).

b. No movements or breathing

There should be no spontaneous muscular movements, nor any breathing movements for at least one continuous hour. The breathing movements are to be observed by turning off the respirator for 3 minutes and observing closely whether there is any effort on the part of subject to breathe spontaneously.

c. No reflexes

(1) The pupil should be fixed and dilated, and unresponsive to bright light.

(2) Ocular movement [to head turning and to irrigation of the ears with ice water] should be absent.

(3) Corneal and pharyngeal reflexes are absent.

(4) Spinal reflexes [eg knee jerk] are also absent.

d. Flat electro-encephalogram

Flat or iso-electric EEG [at 5μV/mm] is of great confirmatory value.

e. Repetition of tests

(1) All tests should be repeated again after 24 hours with no change.
(2) Respirator must be put back on during the intervening period. It can only be taken off after the patient has been declared dead. This was done to offer **legal protection** to physicians, otherwise, it could be argued that they turned off respirator on a living patient.

C. Brain Stem Death (Modern Criteria of the Moment of Death)

Even the brain consists of many parts, the cerebral cortex, brain stem, cerebellum etc. If some cells of the brain were dead, and others alive, how was one going to say that the person was dead? In 1971, **Mohandas** and **Chou** (two neurosurgeons from **Minnesota**) suggested that even within the brain, the most important part was the ***brain stem*** (**Fig 8.4**). Thus one must look only for the **brain stem death**. The **reasons** for this assertion were

(1) **Resistance to anoxia** - Among all brain cells, medullary neurons are **most resistant to anoxia**. Thus if they are dead, all brains cells can reasonably be assumed to be dead
(2) **Vital centers** - Brain stem is responsible for all **vital functions of the body**
(3) **Gateway** - All **cortical pathways pass through the brain stem**. Thus if brain stem was dead, all pathways would be dead. The criteria for brain stem death (now popularly known as the **Minnesota Criteria**) suggested by them were as follows.

1. Minnesota criteria of Brain stem death (1971)

(1) **No spontaneous movement**
(2) **No spontaneous respiration** when tested for a period of 4 minutes at a time
(3) Absence of 5 major **brain stem reflexes** [**Fig 8.3**]
(4) **EEG not mandatory.** All the above findings must remain unchanged for at least 12 hours [**Table 2**].

2. Indian law on brain stem death

[A] **S.2(d)** of The Transplantation of Human Organs and Tissues Act, 1994 - please see ch 2. [B] **S.3(5)** of the same Act - “where any human organ is to be removed from the body of a deceased person, the doctor must confirm that (i) life is extinct in such body or (ii) where it appears to be a case of brain-stem death, such death has been certified under section 3(6). [C] **S.3(6)** of the same

Act - Where any human organ is to be removed from the body of a person in the event of his brain-stem death, no such removal shall be undertaken unless such death is certified by a Board of medical experts consisting of (i) the registered medical practitioner, **in charge of the hospital** in which brain-stem death has occurred (ii) an independent specialist nominated by the doctor specified in clause [i] (iii) a neurologist or a neurosurgeon to be nominated by the registered medical practitioner specified in clause [i] and (iv) the registered medical practitioner treating the person whose brain-stem death has occurred.

Table 2: Differences between 3 major criteria of death

S.No.	Trait	Harvard criteria (1968)	Minnesota criteria (1971)
1.	Basis	Brain death	Brain stem death
2.	EEG	Mandatory	Not mandatory
3.	Duration of testing respiration	3 minutes	4 minutes
4.	Spinal reflexes	Important	Not important
5.	Arterial pressure falling continuously without support by drugs or other means	No specific requirement	No specific requirement
6.	Repetition of tests	After 24 hours	After 12 hours
7.	Respirator during the intervening period of two tests	Can be put back on	Can be put back on
8.	Organ retrieval	Possible because respirator is turned on during intervening period of two tests	Possible
9.	Who can pronounce death	No specific recommendation	No specific recommendation

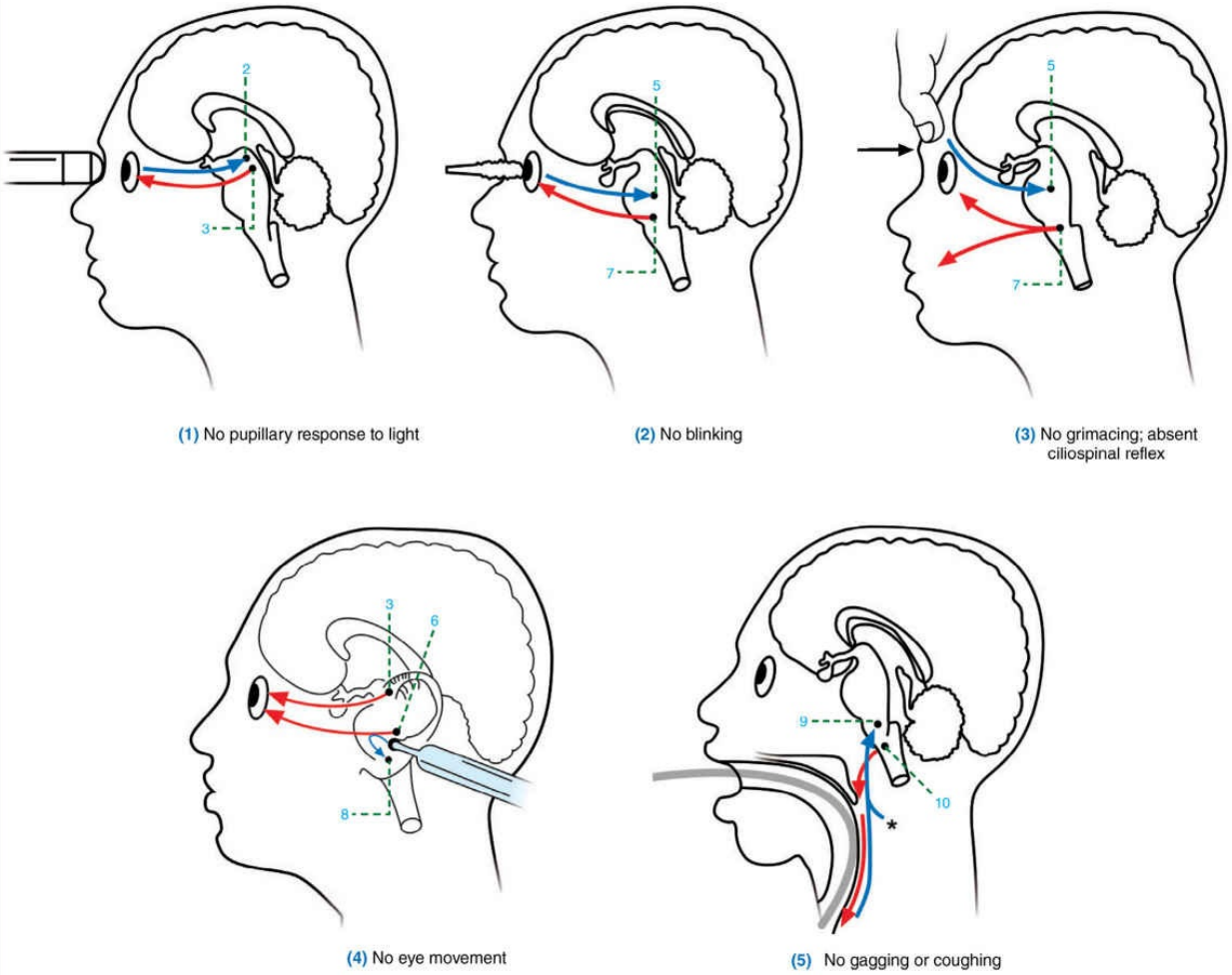


Fig. 8.3: Five major brain stem reflexes tested as Minnesota criteria. Blue lines represent afferent pathways; red lines represent efferent pathways. **(1)** No pupillary response to light [A-2;E-3] **(2)** Absent corneal reflex (touching with a wisp of cotton does not elicit blinking) [A-5;E-7] **(3)** No grimacing [no cranial nerve motor response to pain] and absent ciliospinal reflex (dilation of the bilateral pupil to a pain stimulus applied to the neck, face, and upper trunk) [A-5;E-7] **(4)** Absent vestibular response to caloric stimulation (irrigation of the tympanic membrane with 20 ml of ice cold or warm water) [A-8;E-3,6]. C old water"Eye moves O pposite side; W arm W ater"S ame side [**Memory Aid 2** - COWS] **(5)** Absent gag reflex (reflex contraction of the back of the throat, evoked by touching the soft palate) [A-9;E-10].

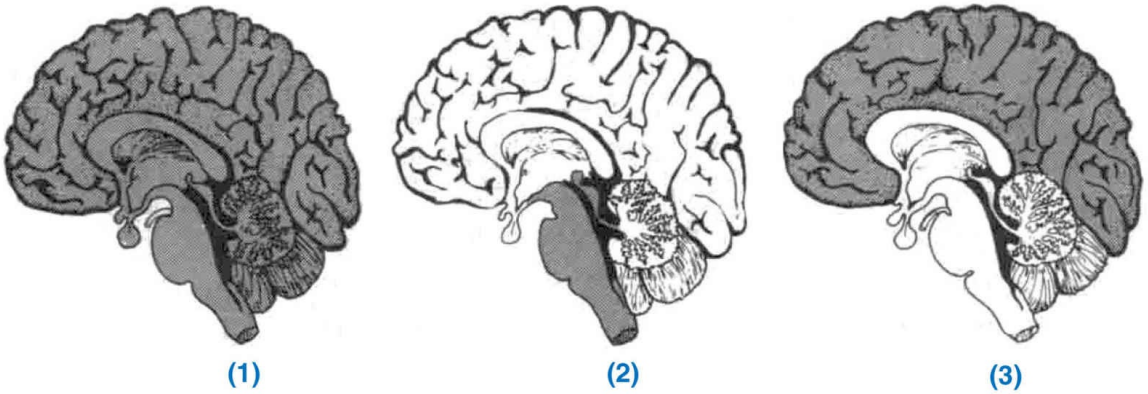


Fig. 8.4: The evolution of the concept of brain stem death. **(1)** Whole brain death (Harvard criteria) **(2)** Brain stem death (Minnesota criteria) **(3)** Vegetative state (Cortex destroyed but brain stem intact)

V. MODES OF DEATH

Mode of death in an old concept of death based on ***Bichat's concept of the tripod of life***.

Table 3: Bichat's modes of death, based on his concept of tripod of life.

Death occurs first in	Mode of death (Bichat)	Typical PM findings
Brain	Coma	Injury to head, disease of brain, **congestion** of brain and meninges
Lungs	Asphyxia	Cyanosis, edema, engorgement of the right side of the heart, petechial hemorrhages, **Tardieu's spots**, viscera **congested**, **-ed fluidity of blood [ch 19]**
Heart	Syncope (fainting)	Anemia , **excessive hemorrhage**, heart contracted, chambers empty, viscera **pale**.

VI. GORDON'S CLASSIFICATION OF DEATH

Gordon's classification of death is an old way of classifying medico-legal deaths taking ***tissue anoxia*** as the fundamental cause of all deaths. Gordon came to the conclusion, that in all cases of deaths of medico-legal importance, **tissue anoxia** must supervene in one or the other way, before death occurred. There are **four major ways** in which tissue anoxia can occur, and thus death was classified into four types.

A. Anoxic Anoxia

[syn, asphyxia, hypoxemic hypoxia, hypoxic hypoxia] O_2 content of blood is .ed. It occurs in following ways:

1. .ed Oxygen in the Ambient Air

(1) Getting trapped in a closed almirah, closet, old unused refrigerators etc
(2) Exposure to noxious gases such as sulphur dioxide, chlorine, sewer gas, etc.
(3) Hypothetical situation of trying to inhale in a vacuum.
(4) Anesthetic agents [**ch 21**]
(5) High-altitude.

2. .ed supply of oxygen

(1) Mechanical Interference with the Passage of Air - Hanging, strangulation, choking, drowning.
(2) Ventilation disorder.

3. .ed pulmonary blood supply

Perfusion disorders.

4. .ed pulmonary gas exchange

Diffusion disorders.

Last two are unimportant medicolegally.

B. Anaemic Anoxia

Oxygen carrying capacity of the blood is reduced. Eg
(1) acute massive hemorrhage
(2) poisoning by CO.

C. Stagnant Anoxia

Blood is incapable of movement. E.g.
(1) heart failure (heart unable to pump the blood),
(2) embolism [heart pumping the blood, but there is obstruction to the flow of blood] and shock.

D. Histotoxic Anoxia

Tissue loses the capacity to utilize oxygen. Histotoxic anoxia is of **4 types**.

1. Cellular histotoxic anoxia

Oxygen cannot be taken up due to failure of tissue enzyme system by poisoning, eg **CN** [**Ch 44**], **PH_3**, **H_2S**, **overdose of hypnotics** and **anesthetics**.

2. Pericellular histotoxic anoxia

Oxygen cannot be taken up due to .ed permeability of cell membrane, eg in **nitrous oxide** or lipid soluble anesthetic agents like halogenated hydrocarbons [**chloroform**, **cyclopropane**, **ether**, **halothane, trilene**].

3. Substrate histotoxic anoxia

Oxygen cannot be taken up due to failure of efficient cell metabolism, eg **hypoglycemia**.

4. Metabolite histotoxic anoxia

Oxygen cannot be taken up due to due to accumulation of end products of cell respiration, eg **CO_2 poisoning**, **uremia**.

VII. NEGATIVE OR OBSCURE AUTOPSY

Negative autopsy is one, when gross and histopathological examination, toxicological, microbiological, serological and other laboratory examinations fail to reveal a cause of death. The term "**negative autopsy**" is used in a wrong sense by several books, which tend to include so called "**defective autopsies**" within this term. Whenever a well-established PM procedure, technique or ancillary investigation is not done, be it due to ignorance, lack of training or due to non-availability of resources etc, it is a defective autopsy. There are thus **four kinds** of autopsies (i) **Normal autopsy** - cause is apparent from gross examination (ii) **Defective autopsy** – cause was ascertainable, but was not ascertained due to some constraint on the part of doctor, hospital, facilities etc (iii) **Obscure autopsy** - Gross findings are **minimal, indecisive,** or **obscure** as in adrenal insufficiency, anesthetic overdose, myxedema, rare plant poisons, thyrotoxicosis etc. Subsequent examinations such as histology, microbiology, serology or toxicology reports establish the diagnosis. (iv) **Negative autopsy** - Cause is not clear from gross as well as subsequent examinations. Some pathologists merge **obscure autopsies** with **normal autopsies** to avoid confusion.

Salient features:

(1) **Rate of negative autopsies**"5-10% of all autopsies.
(2) **Mostly seen in** – children and young adults.

A. Causes of Defective Autopsy

Lack of competence or inadequate training of doctor - (a) Inadequate crime scene examination – (i) presence of legal drugs [eg cardiac medications, asthma inhalers, rare drug prescriptions] (ii) presence of illegal drugs [eg open drug ampoules, drug paraphernalia, open drug sachets, illicit drugs] (iii) Position of the body when first found – Positional asphyxia (eg hyperflexion of the neck from collapse in the corner of a room) may leave no anatomic findings. (iv) Suffocation by the placing of a plastic bag over the head can cause asphyxia in the absence of anatomic findings. The bag may be removed by a careless or ignorant policeman, or by a relative before the body is sent for pm (b) Inadequate examination of clothings - (i) may contain drugs (ii) may show burn marks in low voltage electrocutions (c) Inadequate history. (i) Personal h/o allergies, chest pain, diabetes mellitus, dizziness, epilepsy, prior electrocardiograms [whether positive or negative], syncopal episodes (ii) family h/o sudden death/lethal heart disease (iii) activities of the deceased just before the time of collapse - Exercise, emotional stress, altercation in which the victim received a blow to the chest [Commotio cordis] may suggest sudden cardiac death (d) Inadequate or improper external examination – (i) Electrocution with hidden and/or insignificant entry mark [as in low-voltage electrocutions](ii) Insect bite (iii) Snake bite (iv) Needle marks in drug addicts [fresh and old]. Sometimes these may be hidden by an overlying tattoo (v) therapeutic transdermal patches [eg for nitroglycerin and fentanyl] (e) Inadequate or improper internal examination - (i) Air embolism (ii) anomalous coronary arteries [eg origin of the left coronary artery from the right sinus of Valsalva, the right coronary artery from the left sinus of Valsalva, origin of the coronary artery from a pulmonary artery, ostial stenosis from aortitis, congenitally narrowed coronary ostium, coronary artery bridging] (iii) blunt injuries to heart (iv) concussion (v) Pituitary tumors (vi) Pneumothorax (vii) whiplash injury [self reducing neck injuries] (f) Insufficient laboratory examinations – as in (i) Allergy and hypersensitivity and drug idiosyncrasy [IgE determination would reveal cause] (ii) Biochemical disturbances [eg porphyrias, potassium deficiency, uremia etc] (g) Insufficient toxicological analysis – rare poisons eg tetrodotoxin.

B. Causes of Obscure or Negative Autopsy

Obscure or negative autopsy may be due to: **Unsuspected or obscure conditions**, which leave minimal, indefinite or obscure signs – (a) Concealed trauma - [please see below] (b) Endocrine dysfunction [eg uncontrolled diabetes causing diabetic ketoacidosis, hyperinsulinemia causing profound hypoglycemia] (c) Cardiac conditions - (i) Brugada syndrome (ii) Coronary artery spasm (iii) long QT syndrome (iv) Wolff–Parkinson–White (WPW) syndrome (v) Early MI – No changes may be detectable up to 3 hours [please see below under the heading "sudden and unexpected death] (vi) Flutter and fibrillation [histological examination of SA node, AV node or Bundle of His may reveal diagnosis] (d) Natural conditions and diseases - (i) Epilepsy (ii) Fear, anger, extreme happiness or other emotional outburst (iii) Work stress (e) Poisoning (f) Miscellaneous - (i) Hypersensitivity reactions (ii) Laryngeal spasm [choking, dry drowning] (iii) Status epilepticus (iv) Vagal inhibition. Many of these autopsies would not be negative, if conducted by a thoroughly trained pathologist in well-equipped lab, with all tests conducted properly.

Concealed Trauma

a. Cerebral concussion

May cause death without any marks of injury – external or internal [**ch 17**].

b. Commotio cordis

Please see ch 17

c. Hidden neck injuries

Railway spine"ch 17

d. Reflex Vagal inhibition [RVI]

Vasovagal shock [***syn***, instantaneous physiological death, nervous apoplexy, reflex cardiac arrest, vagal inhibition, vagal shock, vasovagal inhibition of the heart, vasovagal syncope] is sudden death, occurring within seconds or minutes due to minor trauma or relatively simple and harmless peripheral stimulation [eg touching of neck].

Salient features:

Mechanism: (i) Pathophysiology - minor trauma to neck"- pressure on baroreceptors situated in carotid sinus, carotid sheath and carotid body [situated about the level of angle of mandible]"transient -in BP locally in the

sinus"Afferent impulses are transmitted through small **Hering's nerves** to the **glossopharyngeal nerves**"further transmitted to **tractus solitarius** in the medulla"Vagus stimulated" reflex slowing of HR, dilatation of BV, fall in BP [**Fig 8.5**]. The term "**vagal inhibition**" or "**vasovagal inhibition**" is a **misnomer**, because it gives the impression that vagus is inhibited. The correct term should be "**vagal inhibition of the heart**"; the vagus is actually **stimulated** which in turn causes **inhibition of heart**. (ii) In normal persons – this process causes minimal effects [. in HR of <6/min; . in BP of <10 mm Hg]. But in sensitive persons it may cause sudden death.

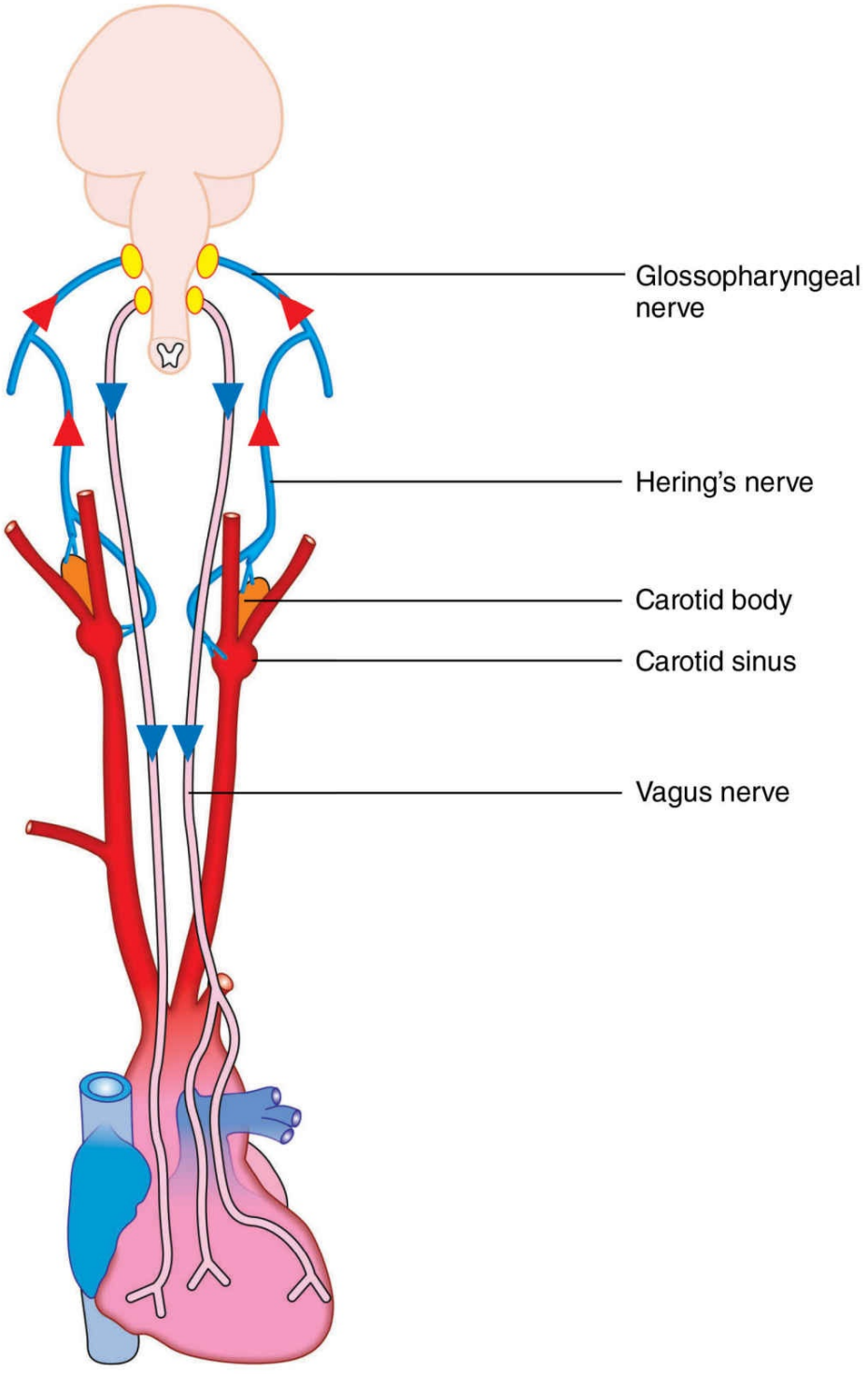


Fig. 8.5: Pathways for vasovagal inhibition

VIII. SUDDEN AND UNEXPECTED DEATH

Sudden and unexpected death is death occurring within ***24 hours*** of the onset of symptoms in an individual who was not known to be suffering from any ***disease***, ***injury*** or ***poisoning***.

A. Sudden Unexplained Death Syndrome [SUDS]

Sudden unexpected death syndrome [SUDS], or ***Sudden unexpected nocturnal death syndrome*** (SUNDS) is a specific name given only to sudden unexpected death of adolescents and adults during sleep.

1. Cardiovascular system

Diseases of the CVA are the leading cause of death in men between 20 and 65 years of age.

a. Coronary artery disease

About 80% of CVS deaths are due to coronary artery disease.

b. Other CVS causes

(1) Aortic Dissection (2) Cardiomyopathies (3) Coronary Artery Anomalies (4) Coronary Artery Spasm (5) conducting system disorders (6) Dissecting Coronary Aneurysm (7) endocarditis (acute) (8) Takotsubo Cardiomyopathy (TTC)

2. Respiratory system

(1) Acute edema of glottis (2) Acute edema of lungs (3) Air embolism

3. CNS

Most common CNS causes of sudden death are (1) epilepsy (2) nontraumatic subarachnoid hemorrhage (3) intracerebral hemorrhage (4) meningitis, and (5) tumors. Occasional rare lesions such as (6) **cysticercosis** may also be responsible.

4. GIT

Main causes are: (1) Acute hemorrhagic pancreatitis (2) Appendicitis (3) Cancer of esophagus (4) Enlarged spleen [rupture].

9. Signs of Death and Changes Following Death

I. SIGNS OF DEATH AND CHANGES FOLLOWING DEATH

Signs of death appear in the following order:

(I) Immediate:

(1) Insensibility and loss of voluntary power
(2) Cessation of respiration
(3) Cessation of circulation

(II) Early:

(1) Changes in the skin
(2) Changes in the eye
(3) Supravital reaction of the skeletal muscles
(4) Cooling of the body (Algor mortis)
(5) Postmortem lividity (Livor mortis)
(6) Changes in the muscles (Rigor mortis, RM)

(III) Late:

(1) Decomposition (combination of autolysis and putrefaction)
(2) Modifications of putrefaction (adipocere, mummification).

II. IMMEDIATE CHANGES

Bichat's concept of **tripod of life** was based on first three changes.

A. Insensibility and Loss of Movement [Cessation of CNS Function]

This sign is due to loss of brain activity. Earliest sign of death, but not an infallible sign. May be seen during life also under following conditions: (1)

Catalepsy (2) electrocution (3) Epilepsy (4) Fainting attack (5) Narcosis (6) Trance (7) Vagal inhibition. Loss of brain activity is better determined by brain stem reflexes and EEG.

B. Cessation of Respiration

Respiration ceases immediately after death. Can be checked by stethoscope (unreliable). Respiration stops for very short periods without death in following conditions: (1) Drowning (2) Drug-induced (e.g. opiate toxicity) (3) Obstructive sleep apnea [OSA] (4) Preterm infants [immaturity of CNS] (5) Voluntary act.

C. Cessation of Circulation

Circulation ceases immediately after death. Can be checked by stethoscope (unreliable) or ECG [please. see ch 8 for earlier tests for circulation].

III. EARLY CHANGES

A. Changes in the Skin

Skin becomes ashy-white, pale and loses elasticity, within a few minutes of death. Lips are dry, brownish and hard due to drying.

B. Changes in the Eye

1. Loss of corneal reflex

Loss of corneal reflex is one of the 5 Minnesota criteria of brain stem death (please see previous chapter for details).

2. Opacity of the cornea

(1) Cornea becomes opaque about 6 hours after death (this is the time period up to which cornea can be harvested for corneal transplantation).

(2) If the eyelids remain open after death, desiccation of sclera occurs giving rise to somewhat triangular shaped discolored areas [Taché noire de la sclérotique (French for “black spots of the sclera”) or simply ***Tache noire*** (black spots)].

These are yellowish in color in the beginning, but as dust settles in, they become dark red (**Fig 9.1**) or later even black. Sometimes they may be bright red, giving rise to an artifactual appearance of subconjunctival hemorrhages.

The eyes look sunken and are softer after death due to fall of Intraocular Pressure (IOP). Normal IOP during life is between 10 to 20 mm Hg [Average – 15 mm]. One hour after death, it is **12 mm** Hg; after 2 h, **10 mm Hg**; after 3 h, **8.5 mm Hg;** after 4 h**, 7.5 mm Hg** and after 8 hours, **5 mm Hg**.[1] Measurement of IOP can thus give a rough estimate of the **pm interval** [PMI].

Memory Aid 1: IOP after death [in mm Hg]

IOP figures after death are **same as figures for circumferences of heart valves**. Pl see mnemonic for heart valves in Appendix [weights and measurements of organs]

Value	IOP [in mm Hg]	Circumference of heart valve [in cm]
15	At death	-
12	After 1 hour	Tricuspid
10	After 2 hours	Mitral
8.5	After 3 hours	Pulmonary
7.5	After 4 hours	Aortic
5	After 8 hours	-

Fig. 9.1: Opaque cornea. TSD-1 day.

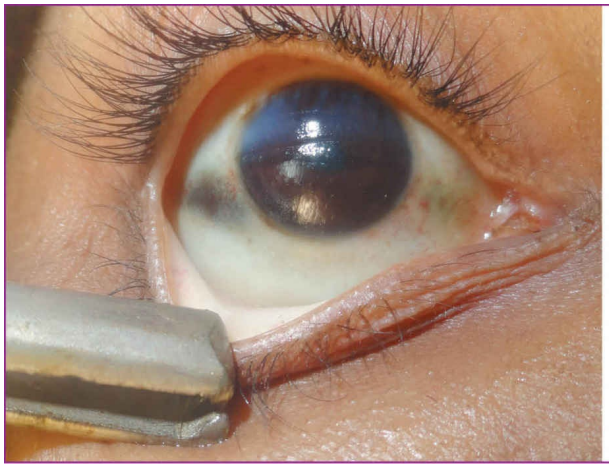


Fig. 9.2: *Taché noire de la sclérotique*. Please note black triangular area on sclera. The base sits on the outer rim of cornea and the apex touches the outer canthus. A similar triangular area on the medial side is less pronounced.

3. Pupils

Normal pupil size during life is between **1-8 mm**. No appreciable change in pupil size occurs after death. Pupils react to atropine and pilocarpine, for about 2

hours after death (till molecular death occurs). If pressure is applied by fingers on two or more sides of the eyeball, the pupil may become oval, triangular or polygonal.

4. Retinal vessels

(1) **Segmentation of the retinal blood columns** – Occurs immediately after death. On ophthalmoscopic examination:

(i) the continuous blood column in the retinal blood vessels breaks up into small segments, which then collide with each other (Kevorkian sign, railroading, trucking or cattle trucking).

(2) **Color of retina** – Retina becomes pale after death, and becomes more and more pale as time of death -es. Disk outline becomes hazy after a few hours.

Fig. 9.3: One hour after death, atropine is instilled in the left eye and pilocarpine in the right. Please note mydriasis in the left and miosis in the right eye.

5. Biochemical changes

During life, K^+ concentration is low in the vitreous humor but much higher in peripheral tissues such as retina. After death K^+ from peripheral tissues starts diffusing in the vitreous raising its concentration.

C. Cooling of the Body (Algor mortis)

Cooling of the body [Algor mortis (L. Algor, cold; mortis, death), chill of death] occurs after death since all metabolism comes to a stop.

Salient features:

(1) **Transfer** - During life heat is constantly transferred from one body part to

other by conduction as well as convection [through flow of blood]. After death this occurs only through conduction, as blood flow stops.

(2) **Heat production** - There is some production of heat during initial period after somatic death due to two mechanisms: (i) ATP molecules breaking down [up to 2 h only] (ii) Anaerobic glycolysis [much longer]. Because of this, rectal temp falls little or not at all during first 2 hours.

(3) **Cooling** – occurs from the surface of the body to the surroundings. It occurs due to temp difference between the body and surroundings [air, water etc]. If the temperature of surroundings is higher, the body temp may actually rise.

(4) **Core temp:** (i) Core temperature is the temp of the viscera. It is slightly -er as compared to surface temp of the body. (ii) Core temp is a better guide to TSD than surface temp.

(5) **Ambient temperature** – Human body rarely reaches the ambient atmospheric temperature unless the latter is at or near freezing point. This is because of the heat released due to enzymatic and bacterial actions on the body.

(6) **Site to take temp from:** (i) Rectal - Best method.

(7) **Method of recording temp:** (i) By touching – Quick, but not accurate. Can only tell if death occurred recently [body warm] or not [body cold] (ii) Lab thermometer – 25 cm long, with a range from 0-50°C, inserted 10 cm above the anus. Left there for 2 min. (iii) Thermocouple – A small electronic thermocouple with a digital readout

(8) **Things to be recorded:** (i) temp of body (ii) temp of environment (iii) Time of recording

(9) Number of recordings - at least 5 at half hour intervals, so rate of fall may be obtained.

(10) **Calculation of TSD:**

(i) TSD [in hours] =

$$\frac{\text{Rectal Temp at the time of death–Rectal Temp at the time body was found}}{\text{Rate of Temp Fall}}$$

When death is not due to hypothermia, hyperthermia etc, rectal temp at the time of death may be taken as 98.4°F. The formula thus becomes

$$\frac{\text{98.4°F Rectal Temp at the time body was found}}{\text{Rate of Temp Fall}}$$

(ii) Average rate of fall - (a) during summers - 0.75°F/h (b) during winters - 1.5°F/h.

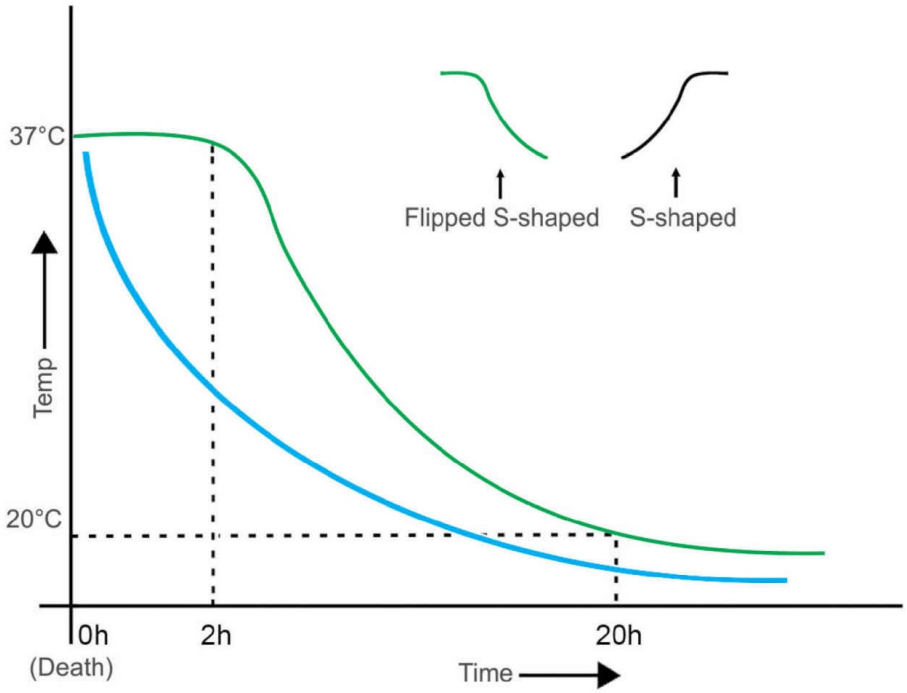


Fig. 9.4: Sigmoid or "Flipped S" shaped, double exponential cooling curve. Pl observe the initial plateau for 2 h. This happens because heat continues to generate for first 2 h after death. This is in contrast to the true Newtonian curve of cooling, which is single exponential (shown in blue).

1. Factors affecting rate of cooling

(1) **Environmental temp** – When the environmental temp is low (winters) heat loss is faster and vice-versa.

(2) **Build:**

(i) Rate of heat loss is directly proportional to the **s**urface **a**rea/**b**ody **w**t [SA/BW] ratio.

(ii) Children who have -SA/BW ratio, lose heat faster.

(iii) Lean and thin adults lost heat faster than fat adults

(3) **Physique** – Fat bodies cool slowly; lean bodies faster. Because fat is a bad conductor of heat.

(4) **Environment:**

(i) Air - (a) Well ventilated room – cooling faster than in a closed room (b) Humid atmosphere – cooling faster than in dry atmosphere

(ii) Water - (a) Body immersed in cold water - cools faster (b) Running water - Cooling faster than in stagnant water (c) Water containing sewage effluent or other putrefying organic matter - Cooling slower than in fresh water

(5) **Position of body** – Cooling faster in bodies lying spread-eagled, than in crouched positions. In the former more surface area is exposed to environment

(6) **Covering** – if body clothed, cooling is slower. Heavier the clothing, slower the cooling.

2. MLI of algor mortis

Helps to estimate TSD.

3. PM caloricity

PM caloricity is a condition when the temperature of body -es after death instead of .ing.

Salient features:

Conditions when it occurs: (i) **Dead body is lying in the open in hot summers** – Most common (ii) **Infections** - like cholera, malaria, septicemia, tetanus, typhoid etc.

D. Postmortem Lividity (Livor mortis)

Postmortem lividity (**syn**: cadaveric lividity, darkening of death, hypostasis, livor mortis (L. Livor, blueness; mortis, death), livores, postmortem hypostasis, postmortem lividity, Postmortem staining, subcutaneous hypostasis, suggillation) is the (i) bluish-purple or purplish-red discoloration (ii) which appears after death, (iii) on the most dependent parts of the skin (iv) due to collection of blood in the capillaries and small veins (rete mucosum) of the most superficial layers of the dermis (v) due to gravity [**Fig 9.5**].

Salient features:

(1) **Cause:** (i) Stoppage of circulation (ii) tendency of blood to sink by gravity to the most dependent parts; its collection in toneless BV and capillaries (iii) Backward flow – of venous blood to the capillaries from the venular end [adds to the blueness of pm staining]

(2) **Color:** (i) Hue – **Bluish purple**, but may vary in different parts of the body

(ii) Intensity – depends upon the amount of reduced Hb in the blood. Large amounts of reduced Hb produce deep purplish coloration (iii) Portions drained of blood – become correspondingly pale (iv) may depend on cause of death [Table 1].

(3) **Place:** (i) In general – well marked in lobes of ears and in tissues underneath fingernails (ii) back of chest and abdomen in supine position. Most common (iii) front of chest and abdomen in prone position; (iv) lower legs and hands in hanging (v) Face in drowning [in drowning, face is the most dependent part]. On the contrary, antigravity regions of the body are drained of blood and become **pale**.

(4) **Visibility:** (i) More clearly seen in (a) fair skinned people (b) young having good nutrition and normal hemogram (ii) Less clearly appreciated in (a) dark colored people (b) old, anemic persons

(5) **PM staining and congestion:** (i) True congestion - Table 2. (ii) Hypostatic congestion – may occur even a few hours before death in persons dying slowly with circulatory failure, eg asphyxia, CCF, cholera, coma [deep], poisonings.

Fig. 9.5: (1) This woman had been dead for 12 hours. PM staining is well developed except over pressure areas (2) PM staining in a 7 month old male child. Please note that due to lack of vertebral curvature in infants, the entire back was in contact with the ground. This has caused the PM staining to blanch out from most of the back.

Table 1: Various colors of PM Staining and associated cause of death

S.No	Color	Cause of Death
1.	Black	**Mummified bodies, Opiates [anecdotal]**
2.	Bluish Green	H_2S
3.	Bluish violet	Asphyxia
4.	**Bright pink**	**Hypothermia, refrigerated bodies, bodies taken out from cold water**
5.	**Bright red**	**HCN, Burns, Refrigerated bodies**
6.	Bronze or Grayish brown	Septic abortion caused by C. perfringens
7.	**Cherry red**	**CO**
8.	**Chocolate**	**Acetanilide, aniline, Bromates, Chlorates [eg $KClO_3$], Nitrites, Nitrobenzene, Potassium bicarbonate**
9.	Dark brown or yellow	Phosphorus

Table 2: Differences between PM Staining and true congestion

S.No	Feature	PM Staining	True Congestion
1.	Redness	Irregular. Occurs only on dependent parts	Uniform all over the organ
2.	Mucus membranes	Dull and lusterless	Congested
3.	Exudate	No inflammatory exudate	Inflammatory exudate seen
4.	Hollow viscus	Staining present only on dependent parts of intestinal coils. Stomach and intestines, when straightened, show alternate stained and unstained areas, depending upon the position of coils	Stomach and intestines show uniform staining

1. Development

a. Time period

i. 0.5-2 hours

Appears as mottled patches.

(1) These are discolored patches of about 1-2 cm diameter, which can be mistaken for bruises [please see **ch 12** for differences].

(2) These patches are initially seen on the upper surfaces of the body, especially the legs due to uneven dilatation of vascular bed. The areas then combine, enlarge and slide down to produce extensive discoloration on dependent parts.

(3) Plasma also accumulates. Good culture media for bacteria; contributes to the cutaneous blisters of early putrefaction.

(4) The time period may be more than normal in anemia and cachexia [1-4 h] and less than normal [few minutes] in persons dying of circulatory failure [because of an already developed hypostatic congestion].

ii. 4 h

Well developed like a sheet but not fixed.

iii. 8 h

Fixation occurs in 8 h [range 6-12 h].

(1) Fixation means that if the body is turned over to a different position, the pm staining would not disappear.

(2) **Causes:**

(i) Clotting of blood within capillaries [**ch 5**]

(ii) By **8 h**, capillaries become permeable"blood leaks through them "stains tissues

(iii) Plain fluid also leaks causing hemoconcentration "tends to keep blood in same location

(iv) rigor mortis of muscles occurs around capillaries "doesn’t allow the blood to move away.

iv. >8 h

Persists till putrefaction sets in.

b. Influencing factors

(1) **Vol of blood** – in circulation at the time of death

(2) Length of time blood remains fluid after death

(3) **More marked** in conditions where blood does not readily coagulate, causing more accumulation to occur. The conditions are:

(i) Amniotic fluid embolism [hypofibrinogenemia]

(ii) Anticoagulants

(iii) Asphyxia

(iv) CO poisoning

(v) Puerperal sepsis

(vi) Retained abortion

(vii) Septicemia.

2. Distribution

Depends upon following factors:

a. Pressure

Any kind of pressure prevents capillaries from filling up. This can be: **Pressure by body parts** – Pressure of one body area pressing another, eg upper arm pinned beneath the back of the body. Areas of body mutually pressing each other would be pale [**mirror image blanching**].

b. Position of body

i. Body lying on back [supine]

(1) first appears on neck, then spreads over the entire back extending up to the flanks and sides of the neck. Blotchy areas may appear on the upper surface of limbs

(2) Not seen in pressure areas [**contact pallor**]:

(i) occipital region

(ii) shoulder blades [typically “butterfly-like”; **Fig 9.5**]

(iii) buttocks

(iv) posterior aspects of thighs

(v) calves and

(vi) heels.

ii. Body lying on face [prone]

(1) **Looseness of connective tissue** - Because the connective tissues in front are relatively loose [as compared to those at back] following special appearances are seen

(i) Color of lividity - intense

(ii) Ecchymosis, petechiae, Tardieu’s spots - common, especially in areas of shoulders and chest. May be mistaken for asphyxial death

(iii) **Cutaneous blisters** - may develop on chest and abdomen containing blood

(2) **Nose** - Minute bv in nose may rupture "bleeding.

iii. Body lying on one side

Pm staining will be seen on one side, with opposite side completely pale.

iv. Suspended

Body suspended as in hanging "PM staining develops on dependent parts of

hands and legs, undersurface of chin, breasts, genitals and soles. If suspension continues for several hours, petechial hemorrhages develop in these areas.

3. PM staining in poisoning

Give in **Table 1**. Additional points

(1) **Bright pink** – in hypothermia etc [at . temperatures Hb as a -er affinity for O_2] and bodies taken out from cold water [wet skin allows oxygen to pass through]. The color is most marked over large joints.

(2) **Chocolate color** – is due to formation of **methemoglobin**, which is a form of Hb where one or more Fe atoms are in a ferric state [Fe^{3+}]. Caused by oxidizing agents [**Table 1**].

4. Internal hypostasis

(1) **In supine position** – in

(i) cerebrum and cerebellum - posterior portions

(ii) Heart – dorsal portions. Simulates myocardial infarction

(iii) Intestines – lowermost coils. Simulates strangulation

(iv) Kidneys, larynx, liver, spleen – dorsal portions. Simulates congestion

(v) Lungs – dorsal portion. Simulates pneumonia

(vi) Stomach – posterior wall. Simulates congestion. Can be differentiated as absent from anterior wall

(2) **In prone position or other abnormal positions** – depending upon the position, the part which is most dependent develops hypostasis.

5. Changes

(1) **With onset of putrefaction** - capillary endothelium shows lytic changes"blood diffuses into surrounding tissues"it undergoes secondary changes [eg sulfhemoglobin formation]"area acquires **greenish** color"Thus PM staining acquires a green color or mergers with the color of putrefaction.

(2) **Microscopic** – At this stage neither the cellular outlines nor the capillaries are identifiable. Thus it is impossible to determine if the blood present locally was intravascular [pm staining] or extravascular [contusion]. Both give the same appearance.

(3) **Advanced putrefaction** - There is destruction of blood. Entire body acquires a very dark black color. It is impossible to see any separate color due to PM

staining"PM staining disappears.

6. ML importance [of PM lividity]

(1) It is a **sign of death**

(2) It must not be confused with **bruise** [for differences, please see ch 12 – Mechanical injuries]

(3) Estimation of **time since death**

(4) Indicates the **position of body** after death

(5) Indicates **movement of the body** to another position, if it is done after pm lividity has fixed in the earlier position

(6) Color may indicate **cause of death**.

E. Changes in the Muscles

After death, all muscles of the body (voluntary, involuntary and cardiac) pass through 3 states (i) Primary relaxation [primary flaccidity] (ii) Rigor mortis [RM] and (iii) Secondary relaxation [Secondary flaccidity].

1. Primary relaxation

Primary relaxation [or flaccidity] is complete relaxation of muscles in the period immediately after somatic death [**Table 4**].

Salient features:

(1) **Relaxation of skeletal muscle** causes following changes (i) contact flattening [flattening of muscles in contact with the surface, eg gluteal and scapular muscles, when body is supine] (ii) Eyelids - lose tension (iii) Lower jaw - falls (iv) joints become flexible

(2) **Relaxation of involuntary muscle** causes following changes (i) pyloric sphincter – duodenal contents move up"bile staining of stomach walls (ii) anal sphincter – emptying of rectum. (iii) bladder sphincter – emptying of bladder

(3) **Persists** - throughout supravital period [i.e. about 3 h]. All cellular functions continue as in supravital period [**ch 8**].

(4) **Muscle protoplasm** – slightly alkaline.

Fig. 9.6: Contact flattening over gluteal region has produced a distinctive pattern. It reflects the kind of surface, the deceased had been lying upon.

2. Rigor mortis

Rigor mortis [L. rigor, stiffness; mortis, death] (***syn*** cadaveric rigidity, death stiffening, RM) is a state of stiffening of muscles, with very little shortening of fibers, that occurs soon after death.

a. Mechanism

ATP molecules continue to deplete from the surface of myosin. When they are completely consumed, "permanent", irreversible cross-linkages form between actin and myosin filaments (***actinomyosin complex***). There is no appreciable shortening of sarcomere [**Fig 9.7(3)**]. During life muscle is alkaline [pH 7.3]. Since the pH of muscle reaches 5.8 (acidic), the rigor (produced under initial conditions of abundant glycogen and ATP) is known as ***acid rigor***.

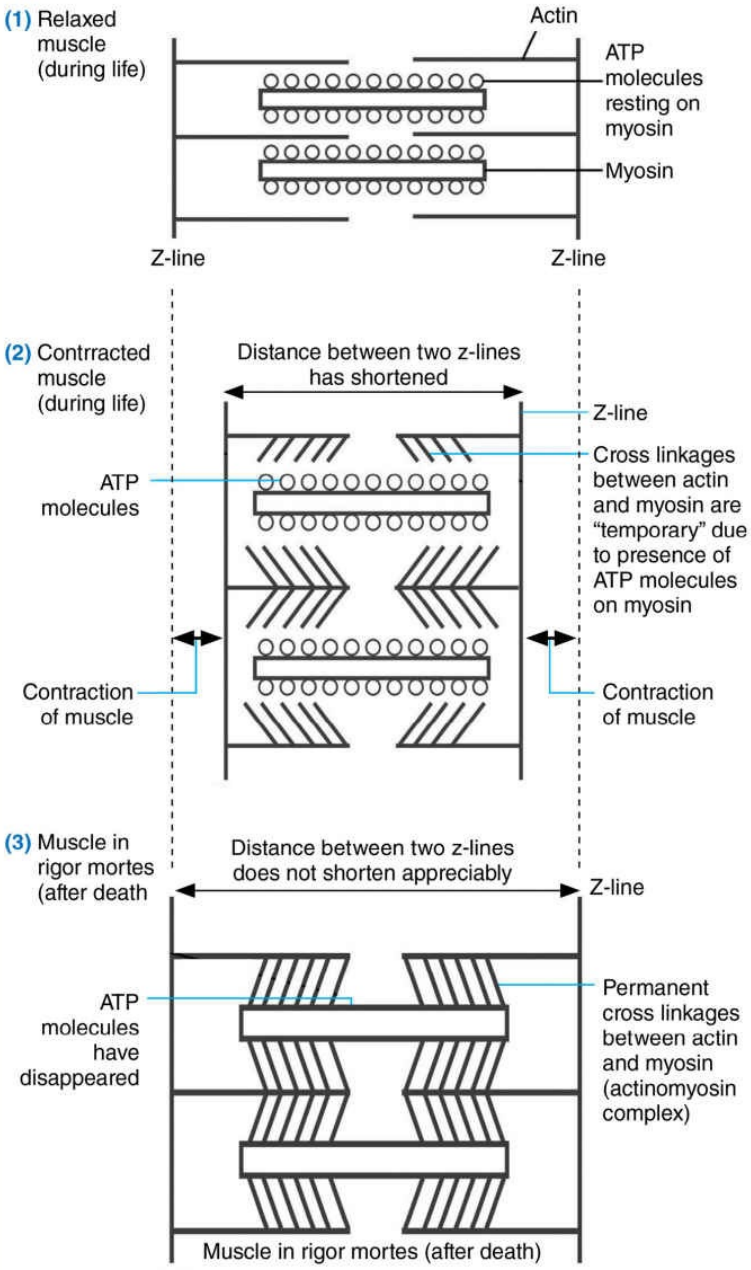

(1) Relaxed muscle (during life)
Actin
ATP molecules resting on myosin
Myosin
Z-line
Z-line
(2) Contrracted muscle (during life)
Distance between two z-lines has shortened
Z-line
Cross linkages between actin and myosin are "temporary" due to presence of ATP molecules on myosin
ATP molecules
Contraction of muscle
Contraction of muscle
(3) Muscle in rigor mortes (after death
Distance between two z-lines does not shorten appreciably
Z-line
ATP molecules have disappeared
Permanent cross linkages between actin and myosin (actinomyosin complex)
Muscle in rigor mortes (after death)

Fig. 9.7: (1) During life ATP molecules are present on the surface of myosin molecules **(2)** Muscular contraction during life involves temporary cross linkages between actin and myosin. The cross bridges do not quite reach the myosin molecule because of the presence of ATP molecules **(3)** After death, fresh ATP molecules are not synthesized. Those already present are consumed in about 2 hours. After that period, permanent cross linkages form between actin and myosin molecules, giving rise to an irreversible actinomyosin complex. There is rigidity but no appreciable shortening of muscle.

b. Order of appearance of rigor

It appears in **all muscles** in this order: Cardiac muscle [1 h]"Involuntary muscles"Voluntary muscles

c. Duration of RM

(1) **Rigor mortis appears** - in the entire body in 12 hours

(2) **Persists** – for another 12 hours

(3) **Then disappears** – in next 12 hours in the same proximodistal fashion in which it appeared. The body is again relaxed in 36 h [secondary relaxation].

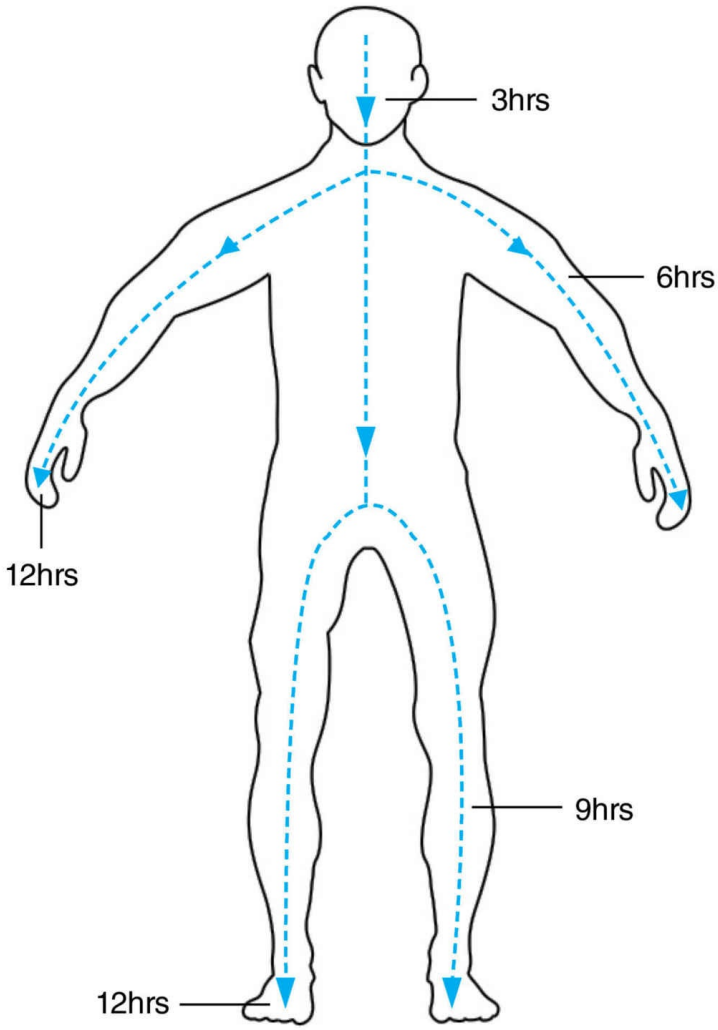


Fig. 9.8: Rigor mortis appears in a proximodistal fashion.

d. Conditions altering onset and duration of RM

All the following conditions can be easily explained on the basis of formation of

actinomyosin complex. If muscular ATP reserve was more at the time of death, and its consumption slower after death, it would take longer to deplete and RM would develop late, and vice-versa.

i. Age

(1) Rigor mortis appears in all ages, even in fetuses. It is wrongly taught sometimes that RM does not appear in fetuses <7 m. It is not true. Author has seen hundreds of fetuses below this age with well-developed RM. As long as there is muscle, the actinomyosin complex would form, and RM would set in. Muscles in a fetus begin to form at **8 weeks**. A fetus **<8 weeks** may not show RM simply because he does not have muscle. The rigor mortis is decidedly weak in young fetuses, and probably some authors confused a weak RM with no RM.

(2) Stillborn fetuses at full term – would develop RM

(3) In children and old people – feeble; develops rapidly; passes away early

(4) Healthy adults – strong and well marked; develops slows; lasts longer; passes away late.

ii. Nature of death

(1) **RM sometimes absent in** - septicemia

(2) **RM appears early and passes away early [duration short]** in

(i) Conditions causing great exhaustion and wasting -(a) bacterial infections [esp gas gangrene, where putrefaction begins early] (b) cancer (c) cholera (d) starvation (e) tuberculosis (f) typhoid

(ii) Death preceded by convulsions - (a) DDT (b) OP (c) Strychnine (d) Tetanus

(iii) Violent deaths - (a) Cut throat (b) Electrocution (c) Firearms (d) Lightning

(3) **RM appears late and passes away late in:** (i) Apoplexy (ii) Asphyxia (iii) CO (iv) Hemorrhage [severe] (v) Neuromuscular disorders (vi) Perfusion with normal saline (vii) Pneumonia.

iii. Muscular state

(1) **Onset slow, duration long, strength substantial** - Muscles healthy and were at rest before death

(2) **Onset slow, duration short, strength weak** – emaciated, weak persons

(3) **Onset rapid, duration short:** (i) Convulsions (ii) Exercise [heavy and violent] (iii) Exhaustion (iv) Fatigue (v) Running [excessive as to save from assailant. Rigor appears (vi) quicker in legs than other body parts] (vii) Strenuous work (viii) Struggle.

iv. Atmospheric conditions

(1) **Onset slow, duration long** – Cold dry weather

(2) **Onset rapid, duration short** – Hot weather.

Table 3: Differences between rigor mortis and cadaveric spasm

S.No	Feature	Rigor mortis	Cadaveric spasm
1.	Onset	3 h after death in skeletal muscles	Instantaneous
2.	Mechanism	Breakdown of ATP below critical level sometime after death	Complete exhaustion of ATP at the moment of death itself
3.	Predisposing factors	None. Occurs in all cases. But may be hastened or delayed by some factors	excitement, exhaustion, fear, nervous tension, sudden death, Violent struggle etc
4.	Molecular death	Occurs	Does not occur
5.	On touching	Body is cold	Body is warm
6.	Muscle reaction	Acidic	alkaline
7.	Muscles involved	All muscles. Both voluntary and involuntary	Voluntary muscles only; that too in a single group of muscles usually
8.	Simulated by	Heat rigor, cold rigor, gas stiffening, chemical stiffening	none
9.	Primary relaxation	Seen	Not seen
10.	Degree of muscle stiffening	Not marked. Moderate force can overcome it	Marked. Very great force is required to overcome it
11.	Electrical stimuli	Muscles will not respond	Muscles in cadaveric spasm would not respond, but other muscles would
12.	MLI	Indicates tsd	Indicates manner of death, and last action undertaken by victim

e. MLI of RM

(1) It is a sign of death

(2) Helps in estimation of TSD to some extent

(3) It indicates position of body at the time of death. If for examples arms are in rigidity extending up in air, it may indicate that they were initially resting on a chair, which was subsequently moved away.

f. Conditions simulating RM

i. Heat stiffening

ii. Cold stiffening

iii. Chemical stiffening

iv. Gas stiffening

(1) In about 2-3 days after death, putrefactive gases collect in s/c tissues, muscles and around joints to stiffen the joints [**putrefactive rigor mortis**].

(2) Both upper and lower limbs are abducted, flexed and rigid; hands are open

and fingers wide apart.

(3) The rigidity persists till gases escape during advanced putrefaction.

(4) This condition is especially seen in bodies recovered from water.

v. Cadaveric spasm

Cadaveric spasm [syn cataleptic rigidity, instantaneous rigidity, instantaneous rigor, postmortem spasm] is a rare condition, in which the muscles that were in contraction at the moment of death, remain in contraction after death without passing through the stage of primary relaxation.

Salient features:

(1) **Predisposing conditions:** (i) Cerebral hemorrhage (ii) CNS injuries [eg Firearm wounds to the head] (iii) Convulsant poisons [eg strychnine] (iv) Excitement (v) Exhaustion (vi) Fatigue (vii) Fear (viii) Pain [severe] (ix) Sudden death

(2) **Last action -** of the person is preserved or "frozen" for several hours after death

(3) **Mechanism** – Unclear. Some theories are (i) ATP depletion at time of death – appears most attractive. Muscular contraction starts during last moments of life, i.e. neuromuscular stimulation, but contraction is retained because of low levels of ATP

(4) **Muscles involved** – usually a single group of voluntary muscles. Rarely entire body as seen in soldiers shot in battle [**battle-field rigidity**].

(5) **Cannot be simulated** – eg by keeping the weapon in victim's hands.

(6) **Disappearance** – occurs along with rigor mortis in other muscles

(7) **MLI:** (i) Differentiation between AM and PM drowning – by grass, weeds [**Fig 9.9**], leaves etc found firmly grasped in hands (ii) Weapon [eg blade, knife, pistol etc] gripped tightly in hand – strongly favors suicide. Attempts to put weapon in victim's hands after murdering him does not produce the same tight grip (iii) Objects grasped in fist – of victim may give information about the assailant, eg hair, pieces of cloth [eg piece of shirt pocket], buttons etc.

3. Secondary relaxation

Secondary relaxation [or flaccidity] is complete relaxation of muscles after rigor mortis has passed away [**Table 4**].

Salient features:

(1) **Mechanism -** 3 theories (i) Action on muscles of alkaline liquids produced

by putrefaction (ii) Excessive acid produced during RM"Dissolution of myosin (iii) Autodigestion

(2) **Secondary relaxation in heart** – may mimic pathologic dilatation, or myocardial degeneration. Because of RM and secondary relaxation appearing in heart, it is not possible to opine if heart stopped in systole or diastole.

Fig. 9.9: Cadaveric spasm in drowning

Table 4: Differences between Primary and secondary relaxation

S.No	Feature	Primary relaxation	Secondary relaxation
1.	Duration	Begins immediately after death; lasts till the first skeletal muscle goes into rigor mortis [3 h]	Begins after rigor mortis passes off. Stays till gas stiffening starts
2.	Molecular death	Has not occurred	Has occurred
3.	Muscular response to chemical, electrical or mechanical stimuli	Yes	No
4.	Body temperature	Warm to touch	Cold to touch
5.	External features	None specific	Signs of decomposition

IV. LATE CHANGES

A. Decomposition

Decomposition involves two processes: (i) **Autolysis** and (ii) **Putrefaction**.

1. Autolysis

Autolysis is the breakdown of cells and organs through an aseptic chemical process caused by intracellular enzymes.

(1) **Cause** - Soon after death cell membranes break down with release of cytoplasm containing enzymes (glycolytic, proteolytic, lipolytic) . The enzymes autodigest the tissues even in the absence of bacteria

(2) **Modification** - Since it is a chemical process, it is accelerated by heat and slowed by cold.

(3) **Sequence** - Organs rich in enzymes (glandular tissue) will undergo autolysis faster than organs with lesser amounts of enzyme (eg, pancreas autolyses earlier than heart and spleen)

(4) **Dead fetus in utero** – there is aseptic environment. Autolysis results in maceration of fetus

(5) **Gastric mucosa** – Autolysis causes softening and rupture of stomach and lower esophagus. In **intracranial lesions**, when the person lies comatosed for long periods, this process may start even before death.

2. Putrefaction

Putrefaction is due to fermentation by bacteria. After death, the bacterial flora of the GIT spread throughout the body, producing putrefaction.

Salient features:

(1) **Commencement:** (i) **At Cellular level** – Putrefaction begins immediately after death at cellular level, but these changes are not visible grossly (ii) **Visible changes** - (a) Putrefaction usually is visible externally after rigor mortis (RM) has passed off. (b) In **summers** however, visible changes may commence before RM has passed off completely.

(2) **Cause: Bacterial enzymes** derived mostly from anaerobic organisms from the intestines (main is Cl. Welchii. Others are B. aerogenes, B.capsulatus, B. coli, B. proteus, Staphylococci and Streptococci).

a. External phenomena

Chief external phenomena of putrefaction are (i) Color changes and (ii) Development of foul smelling gases.

i. Color changes

Greenish discoloration of right iliac fossa (RIF) – This is the earliest sign of

putrefaction

(1) It is more clearly appreciated in **fair skinned people** than in the dark skinned

(2) **Time** - Occurs in **18 h** in summer and **36 h** in winter.

(3) **Mechanism** - Just below RIF lies cecum. It lies very superficially, its contents are in a fluid state and full of bacteria. They migrate out and (a) break up sulfur containing amino acids [cysteine, cystine and methionine] to form H_2S, which combines with Hb to form sulfhemoglobin (SulfHb), which is greenish in color. (b) cause deamination of L-phenylalanine. Produce NH_3 and phenylpyruvic acid which combine with Fe^{3+} to form a green complex.

Table 5: Differences between Putrefaction and gangrene

S.No.	Criteria	Putrefaction	Gangrene
1.	Pus	Not formed	Usually formed
2.	Phenomenon	Postmortem	Antemortem
3.	Inflammation and repair	-	+
4.	Line of demarcation between living and dead tissue	None. All tissue is dead	Present
5.	Area	All over	Localized

Fig. 9.10: Marbling over chest. Please also note strangulation mark over the neck.

ii. Development of foul smelling compounds

(a) **Chemical processes** in putrefaction are those of **reduction**. Proteins and carbohydrates are reduced to simpler compounds (amino acids, CH_4, CO, CO_2, H_2, H_2S, mercaptans, NH_3 and PH_3). Many of these gases are inflammable, and they can be ignited [these gases may give rise to **preternatural combustion** (Ch 14)].

(b) **Foul smell** – Foul smell from the cadaver is mainly due to the production of **ptomaines [**syn**, cadaveric alkaloids**]. Most ptomaines are **biologic amines** [low molecular wt compounds produced by bacteria through **decarboxylation of free amino acids**, releasing **CO_2** in the process]. Two most common ptomaines are **cadaverine** [$NH_2(CH_2)_5\ NH_2$ produced by decarboxylation of **lysine**], and **putrescine** [$NH_2(CH_2)_4\ NH_2$; produced by decarboxylation of **ornithine**].

(c) **Effects of gases:** (1) **Blisters** – (i) Form because of collection of gases between epidermis and dermis [**postmortem blisters]**. Fluids and liquid fat may also be pushed in the blisters (ii) Must be differentiated from antemortem blisters

[**ch 14**]. (iii) Blisters form first on areas where tissues contain more plasma due to hypostatic edema [back, lower surfaces of trunk, thighs] (iv) **Time of development**: Summers-36 h, Winters-48 h. (2) **Bloating of features** [2-3 d] – May be more marked when the head of the cadaver is in most dependent position [accumulation of fluids]. Appearance quite similar to that of strangulation: (i) **Eyes** – softened. May bulge from sockets (ii) **Lips** – swollen and discolored (iii) **Tongue** – blackened and thrust between the teeth (iv) **Face** – swollen and discolored. Identification becomes difficult or even impossible. In late stages, face is transformed into a bulbous green mass. (3) **Emptying of heart** – Blood is pushed out; blood for analysis not available in heart. (4) **Expulsion of fetus from uterus** [postmortem delivery]– may be seen in pregnant women in 2-3 d. Rigor mortis of uterus may be an additional reason. (5) **Expulsion of urine and feces** – in 2-3 d. Due to formation of gas and relaxation of sphincters. (6) **Floatation of body in water** – In drowned bodies, formation of gases cause the body to come up and float [please also see ch 19 - Asphyxia]. (7) **Postmortem aspiration** – Gases in the stomach force the food up in the throat, from where it may fall into larynx. Differentiation from true antemortem aspiration – Food not found beyond the bronchi in PM asp, while in AM asp, it will be found up to terminal bronchioles. (8) **Shifting of the position of PM staining** [36-48 h] – PM Staining displaced in any direction. Position of body immediately after death can not be ascertained, if body seen at this stage. (9) **Skin slippage:** (i) Epidermis becomes loosened, and separated (**Fig 9.11**). (ii) Skin of hands and feet may come off in a “glove and stocking” fashion. (iii) Exposed subdermal tissue dries giving a yellow parchment like appearance. Fingerprints can be taken from skin deprived of epidermis [please see ch on identification]. (10) **Skull** – Sutures of skull [especially of children] separated, with flowing out of liquified brain [3-5 d] (11) **Soft tissues become crepitant** – Entire body swells due to collection of gases in the tissues. If a suitable area, say, chest is pressed, the crepitant collection of gases can be felt. Feels like a sponge. (12) **Swelling of entire body** – May make even a thin body appear obese. (13) **Microscopically** – Breakdown of vessel walls and cell membranes leads to water logging of tissues with protein rich fluid. This helps in the growth of bacteria.

Fig. 9.11: Post mortem peeling.

iii. Other changes

(1) **Cornea** – becomes white, flattened or compressed [12-18 h in summers; 1-2 d in winters]

(2) **Fingertips** –become leathery and wrinkled. Nails become prominent [**36-48 hours]**

(3) **Fat** – Body fat, especially omental and mesenteric liquefies into a translucent yellow fluid.

(4) **Loosening of appendages** - Loosened and can be easily pulled out (i) Hair [3d] (ii) Nails [4d] (iii) Teeth [5d]

(5) **Postmortem luminescence** - Body may start emanating light in the dark. The light comes not from the body, but from luminescent bacteria [Photobacterium fisherii] and fungi [Armillaria mellea].

(6) **Fissures or splits** – may form in skin. May simulate antemortem incised wounds or lacerations.

iv. Appearance of maggots

(1) Maggots appear in **1-2 days**. They have proteolytic enzymes and penetrate the skin rapidly.

(2) This allows air to enter underneath the skin"More maggots enter body

cavity"Activity of maggots may -body temperature to above that of normal body temp.

(3) Decomposition and maggot activity -temp of body.

(4) May produce small round holes which may be confused with firearm wounds.

b. Internal phenomena

Internally decomposition proceeds more slowly than at the surface.

(1) **36 h - Inner surfaces of vessels** are discolored reddish brown, especially the aorta. Due to Hb released from RBC, which stains the walls. This is the earliest internal putrefactive change

(2) **36-48 hours:**

(i) Clotted blood becomes fluid.

(ii) Fluid blood starts collecting in serous cavities. Such effusions are usually less than 100 ml.

(3) **48-72 hours - Viscera** are discolored dark red to greenish yellow, greenish blue, greenish black and finally black. They are softer and greasier to touch

(4) **5-10 days:**

(i) Colliquative putrefaction begins.

(ii) Abdomen – Bursts. Stomach and intestines protrude Thorax – bursts especially in children.

(iii) Tissues – become loose and soft. Converted into a thick, semifluid, black mass. Separated from bones and fall of

(iv) Body fat – especially mesenteric, omental and perirenal liquefies into a translucent yellow fluid, which fills the abdominal cavity between organs. Marked in obese people.

(v) Cartilages and ligaments – softened towards the final stages

(vi) Miscellaneous – in ruptured ectopic pregnancy, small fetuses may completely disappear.

i. Order of putrefaction

(1) **General rule** - The softer the organ, the more blood it contains and the nearer to sources of bacteria it is, the faster it putrefies

(2) In general, organs show putrefactive changes in the following order - (a) Larynx and trachea (b) Brain of infants (c) Stomach, intestines (d) Spleen (e)

Omentum and Mesentery (f) Liver (g) Adult Brain (h) Heart (i) Lungs (j) Kidneys (k) Adrenals (l) bladder (m) Esophagus (n) Pancreas (o) Diaphragm (p) Blood vessels (q) Gall bladder (r) Skin, muscle, tendons (s) Prostate, virgin uterus (t) bones.

Memory Aid 2: Order of putrefaction

Large tree bird sits on liver. Adult bird has long kidneys, a bad Ependix and big muscles.

Large **tr**ee - Larynx and trachea

b i rd - Brain [infants]

s i t **s** - Stomach, intestines Spleen

On - Omentum and Mesentery

Liver - Liver

Adult bird - Brain [adults]

Has - Heart

Long - Lungs

Kidneys - Kidneys

a - Adrenals

bad - Bladder

E pen di x - Esophagus Pancreas Diaphragm and

b i **g** - BV Gall bladder

muscles - Muscles.

NB - The **last soft organs** to putrefy are **prostate in males** and **virgin uterus in females**. **Bones are the last organs [including both soft and hard] to putrefy**. These facts are easily remembered on their own.

Liver

Timings are for summer. In winter same changes take twice the time

(1) **Soft and flabby** – 12-24 h

(2) **Multiple blisters** – 24-36 h

(3) **Honeycomb liver** [**Fig 9.12**]:

(i) Cl.welchii collect in tissue spaces in characteristic small clumps

(ii) Produce gas which soon -in size

(iii) Resulting lesions first appear as small, opaque, yellowish grey, dendritic figures in parenchyma.

(iv) Later form bubbles making liver look like a honeycomb [foamy liver, honeycomb liver, vesicular liver; **Fig 9.12**].

(4) **Color:**
(i) Initially greenish; later coal black
(ii) In early stages, discoloration is seen around the branches of portal vein
(5) **Newborns** - Liver putrefies earlier in newborn children than in adults
(6) **Gall-bladder** - putrefies much later than liver.

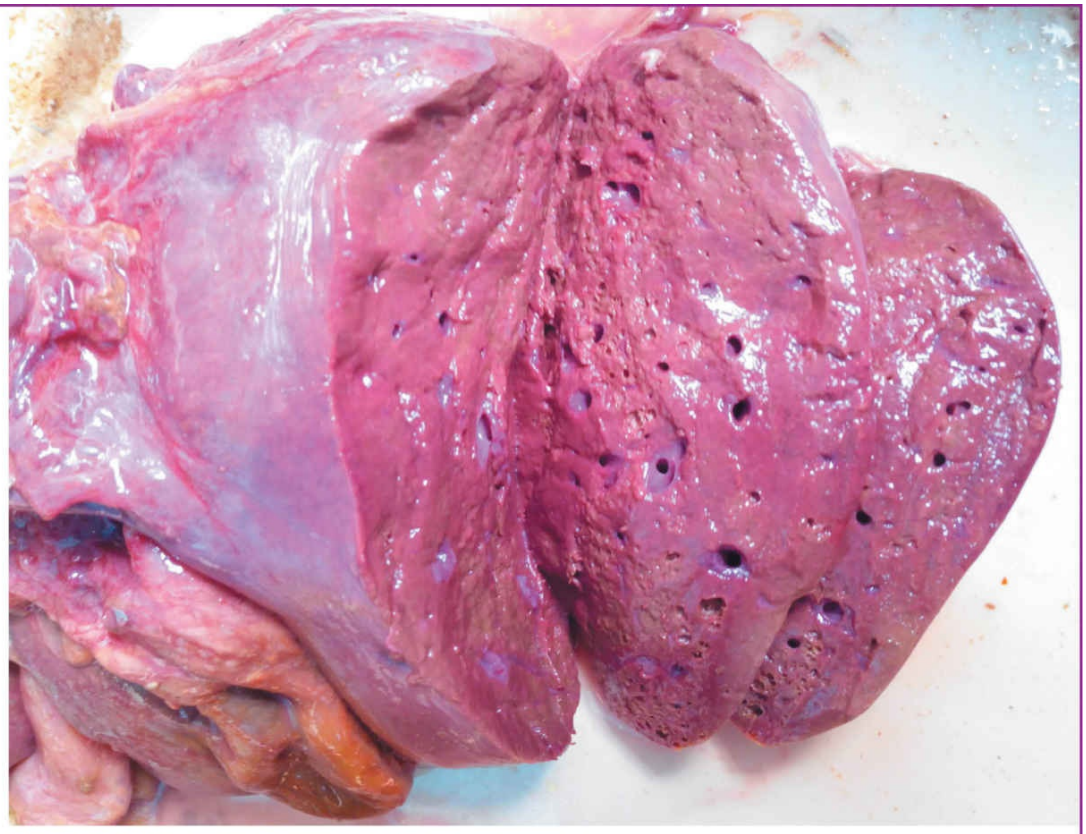


Fig. 9.12: Honeycomb liver

c. Skeletonization (last stage)

Skeletonization is the last stage of putrefaction, when the body is reduced to a bare skeleton.

Salient features:

(1) **Time required** – varies considerably
(2) **Body lying exposed in air** – Carnivores [Dogs, Jackals, vultures], Flies and their larvae [Maggots], Insects [Ants, Cockroaches] and Rodents [rats] reduce the body to a skeleton within a few days
(3) **Body in water** – body attacked by aquatic animals [crabs, crustaceans, fishes etc]. Clothing protects body from animals for long periods. In warm weather

algae may deposit on exposed parts of body offering some protection against aquatic animals.

(4) **Buried bodies:**

(i) Factors modifying rate of skeletonization (a) Access to air (b) Acidity of soil and soil water (c) Amount of soil water (d) Climatic and seasonal variation

(ii) Bodies buried without coffins - (a) in shallow grave – Putrefaction delayed to a moderate extent (b) in deep grave – Decomposition is markedly delayed [due to .temp, exclusion of air, absence of animal life]. (c) Mass burials in a common grave - Bodies lying in the center of grave are better preserved than those at the periphery (d) Average time period for complete skeletonization in a buried body in India – 1 y

(iii) Bodies buried with coffins - (a) If coffin is of good quality and airtight – disintegration does not complete till several decades (b) Poor quality coffin which admits air and water – bodies may decompose within months

(5) **Bones lying on surface** – As proteinaceous portion decomposes, the bones are reduced to just calcium, which then crumble. Bones which decompose faster – flat bones, bones of infants and old.

(6) **Buried bones** – decay according to acidity of soil. Neutral soil does not destroy bones. Acidic soil causes decay in 25-100 y depending upon acidity.

B. Conditions Affecting the Rate of Putrefaction

1. External

a. Air

(1) **In normal conditions** – unbroken skin acts as an impermeable barrier to bacteria

(2) **Free access to air**"hastens putrefaction [air conveys organisms to the body].

(3) **Moist air**"hastens putrefaction.

b. Clothing

(1) **Loose clothing:**

(i) **Initially** - -putrefaction [because body temperature is maintained and is not allowed to fall]

(ii) **Later** - .putrefaction [because access of airborne organisms, flies, insects etc is prevented]

(2) **Tight clothing** – includes belts, boots, socks, suspenders and undergarments [tight fitting]

(i) **Initial and late stages both** - .putrefaction [tissues compressed"blood driven out"organisms do not find medium to grow]. Also access to organisms is prevented as above.

c. Manner of burial

Putrefaction is less: (i) Body buried soon after death (ii) Buried in dry soil (iii) buried in sandy soil (iv) Buried in lime.

d. Moisture

(1) Moisture is necessary for putrefaction

(2) **Conditions associated with -ed moisture -the rate of putrefaction:** (i) anasarca (ii) bodies recovered from water (iii) Organs which contain more water putrefy faster.

e. Temperature

(1) **Putrefaction begins at**"10°C.

(2) **Temp increase of 10°C**"doubles the rate of all chemical reactions, including putrefaction

(3) **Season** – rate of putrefaction is twice in summer as in winter.

(4) **Putrefaction is optimum between**"21°C-38°C.

(5) **Putrefaction is arrested**"<0°C and at >48°C.

(6) **Dead bodies are best preserved at**"4°C.

2. Internal

a. Age

(1) **Newborns:**

(i) If unfed – slow, because body is sterile

(ii) If fed – fast

(iii) If IV drips etc were given – fast, because bacteria can enter quickly through punctures

(2) Bodies of children putrefy faster than those of older people [less water content].

b. Sex

No effect on putrefaction.

c. Condition of the body

Fat and flabby bodies putrefy faster [-fat and fluid, -er retention of heat].

d. Cause of death

(1) Putrefaction is **rapid** in deaths due to: (i) Anasarca [generalized] (ii) Asphyxia (iii) Inflammatory conditions (iv) Peritonitis and (v) epticemia

(2) Putrefaction is **delayed** in deaths due to: (i) Anemia (ii) Debility and (iii) Wasting diseases. (iv) Poisons which either have preservative action on tissues or a destructive [or inhibitory] action on bacteria or both.

i. Poisons which delay putrefaction

(1) Carbolic acid

(2) Heavy metal poisoning [chronic], especially arsenic

(3) Strychnine [if death occurred following a few seizures]

(4) Zinc chloride. These should not be confused with poisons which ***resist putrefaction*** [i.e. which are not destroyed by putrefaction; can be detected even in putrefied bodies].

ii. Poisons which resist putrefaction

(1) Barbiturates [**ch 40**] (2) Corrosives (3) Cyanide (4) Datura (5) KCN (6) Metals (7) Nicotine (8) Pancuronium [Pavulon] (9) organophospnates (10) Phosphorus (11) strychnine. Heavy metals and strychnine both delay and resist putrefaction. Cyanide both appears after and resists putrefaction.

iii. Poisons which are destroyed by putrefaction

(1) Aconitine (2) morphine (3) succinylcholine.

Memory Aid 3: Poisons which are destroyed by putrefaction

Destroy is same as **SMA**SH

S - **S**uccinylcholine

M - **M**orphine

A - **A**conitine

iv. Poisons which hasten putrefaction

(1) Chronic alcoholism

(2) H_2S

(3) Strychnine [if death occurred after prolonged and repeated seizures"-heat production].

v. Poisons which appear due to putrefaction

(1) Alcohol - Bacterial and fungal fermentation yields ethyl alcohol, which may vitiate the interpretation of postmortem alcohol concentration. Levels of alcohol

produced by putrefaction are almost always **<200 mg%**. (2) CO [**ch 44**] (3) cyanide (4) Ptomaines (5) Substituted phenols, especially p-hydroxy phenyl derivatives.

Memory Aid 4: Poisons which appear due to putrefaction

APPEAR

A – Alcohol

P – Ptomaines

P – Phenols [substituted]

3C – **C**arbolic, **C**O, **C**N

e. Mutilation

(1) **Mutilated bodies**, or bodies with excessive wounds "Rapid putrefaction [(i) organisms gain easy access]

(2) **Dismemberment**"Putrefaction of limbs is slower than that of trunk [(i) Blood drains out of limbs (ii) intestinal organisms cannot gain access].

C. Casper's Dictum

Casper's dictum [syn Casper's law, Casper Regel, Casper's Rule] states that the time taken for same amount of putrefaction to occur when the body is in air, water and buried in earth is in the ratio of 1:2:8. Putrefaction occurs fastest in air and slowest in earth.

Memory Aid 5: Casper's Dictum

AWE (**A**ir, **W**ater, **E**arth)

D. ML Aspects of Putrefaction

(1) **Time of death** - can be ascertained

(2) **Cause of death** – becomes difficult to ascertain, except in cases of Firearm injuries, fractures, drowning and Poisoning. Hair, teeth and bones resist putrefaction and several poisons [esp **metals**] can be detected in hair and bones. Several poisons resist putrefaction [please see list above]. **Diatoms** may be demonstrated in bones.

(3) **Infanticide** - level of diaphragm may spuriously be altered by gases of decomposition [**ch 27**].

(4) **Poisons:**

(i) can hasten or delay putrefaction,

(ii) can resist putrefaction and
(iii) may destroy during putrefaction
(iv) may appear after putrefaction [defense lawyer may argue that the poisons appeared due to putrefaction]. These lists are given above.
(5) **Raygat's test** - May be vitiated by putrefaction [**ch 27**].

E. Modifications of Putrefaction

1. Adipocere

Adipocere (Latin adipo fat, cera wax) is a modification of putrefaction in which a friable, crumbly, water insoluble grayish-white to brown wax-like material is produced in the dead body by the breakdown and conversion of body fats to fatty acids (FA), mainly oleic, palmitic, and stearic acids.

Salient features:

(1) **Nomenclature** - Adipocere is also known as corpse wax, grave wax or mortuary wax.
(2) **Areas where formed [Distribution]:**
(i) It is most prominently formed in areas where fat is excessive [face, cheeks, breasts, abdomen, buttocks].
(ii) Limbs, chest wall and other parts of the body are less commonly affected.
(iii) Can occur wherever fat is present.
(iv) Rarely entire body be converted into adipocere.
(v) Internal adipocere – Starts earlier, because body's own water is used. Fatty tissue present between individual fibres of (a) Skeletal muscle - Small muscles are dehydrated, very thin and acquire a uniformly grayish color. Large muscles show a pinkish or reddish color in depths. (b) nerves and (c) Myocardium. Fat in the substance of (d) liver [especially if fatty liver] – Retains its shape (e) kidneys (f) other internal organs. Adipocere then forms a matrix for remnants of tissue fibres, nerves and muscles. Grossly the organs retain their shape; histologically only cell outlines can be made out. (g) Lungs and intestines – thin, parchment like (h) May rarely form in the fat of bone marrow in fractured bones
(vi) Skin – As adipocere forms, (a) epidermis disappears due to decomposition and shedding (b) Dermis becomes darkened (c) Multiple whitish-grey, rounded outgrowths [1-10 mm in diameter], resembling moulds, are seen on

the surface. (d) These are protruding clusters of crystals from underlying adipocere

(3) **Conditions required:** (i) Hot and humid environment.

2. Mummification

Mummification is a modification of putrefaction in which the skin becomes brittle, contracted, dried, firm, leathery tough, parchment like, shrunken and wrinkled, turning yellow-brown to black in color. [from Latin mumia, bitumen; because of the blackened skin, bitumen was once thought to be used by Egyptians to produce mummies].

Salient features:

(1) **Conditions required:** (i) Hot and dry environments, eg air heated buildings, deserts. Hot environment helps evaporate water away from the corpse, making it desiccated and dry (ii) enclosure in warm and dry environments – unwanted newborn children have been killed and hidden in trunks and kitchen cupboards, where their mummified bodies are recovered a few years later.

(2) **Internal organs:** (i) Consistency - Hard and shrunken. May adhere together to form a single mass. Individual organs may lose their identity and disappear (ii) Color"Dark brown and black

(3) **Entire body:** (i) loses wt, becomes brittle, stiff and thin (ii) emits no foul smell (iii) if unprotected – gradually breaks into fragments, becomes powdery and disintegrates. May also be attacked by flies, insects, larvae and moths (iv) if protected [as by wrapping in bandages (done by Egyptians)] – preserved for centuries

(4) **Histologically demonstrable tissue** are (i) Cartilage and bone (ii) Collagen (iii) Elastic tissue and (iv) Muscle [cardiac and skeletal]

(5) **Partial mummification:** (i) occurs in some cases, with only some parts [eg head, limbs, trunk] mummified and others not. (ii) Occasionally some parts show mummification [arms and legs], while others adipocere [cheeks, abdomen, buttocks]

(6) **Medicolegal Importance:** (i) **Identification** - Features are relatively well preserved (ii) **Determination of cause of death** is possible – Because injuries are preserved. (iii) **Time and place of death** - can be ascertained. Time required is same as that of adipocere formation.

V. EMBALMING

Memory Aid 6: Composition of a typical embalming fluid

PG Bounced a **WAD** in **VP**'s **M**outh

1. **P**reservatives – **FM** [**F**ormaldehyde, **M**ethanol]
2. **G**ermicides – **BGP** (similar sounding to BJP) [**B**enzalkonium chloride, **G**lutaraldehyde, **P**henol]
3. **B**uffers
4. **W**etting agents – **W**i**GS** [**G**lycerine, **g**lycols; **s**odium lauryl sulphate, **s**orbitol]
5. **A**nticoagulants
6. **D**yes – **T A PE R** - Toluidine red, Acid fuchsin, amaranth, erythrosine, rhodamine, rose Bengal
7. **V**ehicle
8. **P**erfuming agents – **LOC**U**M** – Lavender, Oil of cloves, Cinnamon oil, Menthol, Methyl salicylate
9. **M**uscle relaxants – **M**gCl2

VI. ESTIMATION OF PM INTERVAL (PMI)

[A] ***Postmortem interval (PMI)*** or ***time since death*** (TSD) is the time period between death and examination of the body. It is important because of following reasons: (i) **credibility of suspect** - one can check the credibility of a suspect's statement (ii) **Moment of crime** - It informs when the crime was committed (iii) **Vital leads to the police** - It gives police a vital lead and a starting point for their enquiries (iv) **Exclude suspects** – if a suspect was not in the city during the time of death, he can be excluded (v) **checks alibi** – It can confirm or disprove an alibi. [B] **Importance of PMI** - Determination of PMI is important both in criminal and civil cases. In **criminal cases** it is important to catch the killer; in **civil cases**, it may become important in deciding (i) who inherits the property (please see the example under the heading "commorientes" below) and (ii) whether at the time of death the **insurance policy** was in force. [C] **Determination of PMI:** (i) Exact time of death **can not be determined**, except in very rare cases, where the wrist watch of the victim stopped due to drowning, the blow of a weapon or fall from height. In most cases, the enormous biological and environmental variations make it very difficult (ii) The pathologist should give a **range of PMI** rather than a fixed PMI. To ***estimate PMI*** one must make use of ***immediate***, ***early*** and ***late*** changes (as the case may be) as mentioned

above. In addition, one can make use of following parameters.

A. Electrical Contractility of Muscles

Can be used to calculate TSD up to first 2-3 h [supravital period. Please see ch 8].

B. Nasal Ciliary Motility

Nasal cilia maintain motility up to 18 h after death.

C. Food in the Stomach

(1) **Average emptying time of stomach** -approx **3 h** [one feels hungry 3 h after last meal]. Thus if the time of last meal is known, tsd can be calculated.

(2) **State of digestion** – of food inside stomach also indicates time of death.

(i) Digestion is an active antemortem process, which does not continue after death. Although acids and enzymes are present, the peristaltic movements necessary to churn food with them are absent.

(ii) Recognition of meal - If complete digestion has taken place [>4 h], nature of food cannot be recognized. This generally happens when food has passed in the intestine. As long as food is in stomach, it can generally be identified. Identification of meals generally corroborates or negates the story of witnesses, especially if the death was sudden.

D. Postmortem Chemistry [Thanatochemistry]

1. Blood

Carbohydrates:

(1) Glycogenolysis in liver"Glucose diffuses in nearby vascular channels "-ed glucose [in vena cava and R side of ht]"Spurious pm diagnosis of diabetes mellitus may be made.

(2) Right atrial blood is sampled, which contains glucose diffusing from glycogenolysis in the liver

(3) Glycolysis in the PM period occurs at a rate of **12.8 mg/100 ml/h**.

(4) However blood glucose levels are not useful as PMI indicator because of several variables – Diabetes, cause of death [-glucose levels occur in asphyxia,

CO poisoning, -ed Intracranial pressure].

2. CSF

About **15mL** is removed through LP. Specimen of choice in child abuse cases when eyes are to be examined histologically.

Carbohydrates:

(i) Glucose - .es very rapidly after death

(ii) Lactic acid - -es sharply and regularly up to 10 h. Thereafter continues to -, but at a lower rate

(iii) Inositol – Normal level during life : 1.7±0.45 mg%. -es regularly after death, with levels reaching up to 72 mg%.

3. Vitreous

Interpretation

Variation between two eyes may be up to 10%.

(1) **Carbohydrates:**

(i) Glucose – Normal vitreous glucose levels are approximately half the serum levels. With -ing PMI, there is a consistent .in levels. Within 4-5 h, levels are zero in non-diabetics.

(ii) Lactic acid – normal values are 80-160 mg%. After 20 h, they - to 210-260 mg%.

(2) **Electrolytes:**

(i) Potassium – Found to be most useful so far. During life, K^+ concentration is low in the vitreous humor [5.8 mmol/L] but much higher in peripheral tissues such as retina. After death K^+ from peripheral tissues [retinal cells and vascular choroid] starts diffusing in the vitreous -ing its concentration. This occurs in a linear fashion. Formulae most popular in this regard are: (a) Time since death [in hours] = 7.14 # [K^+]in mmol/l – 39.1 (**Sturner's formula**) (b) Time since death [in hours]= 5.26 # [K^+]in mmol/l – 30.9 (**Madea's formula**) (c) Time since death [in hours]= 4.32 # [K^+]in mmol/l – 18.35 (**James' formula**) (ii) Sodium – Normal levels are 2480-3500 mg/L [or 118–154 mmol/L] Postmortem change is too insignificant to be of any help.

4. Pericardial fluid

(1) More quantities [10-15 mL] can be obtained as opposed to 5mL of CSF and 1-2 mL of vitreous.
(2) **Potassium** - -es at an average rate of 0.30 mmol/h.
(3) **Sodium** -.es at a rate of 0.37 mmol/h in the first 85 h after death.
(4) **Calcium** – Doesn't change appreciably after death.

5. Synovial fluid

Levels of sodium, potassium, calcium, chloride, urea, creatinine, and glucose in synovial fluid show that they behave in the same way as in vitreous.

E. Hair

(1) **Hair does not grow after death**; yet it appears to have grown in dead individuals. It is due to shrinkage of skin, which make the hair look taller.
(2) **Time since death:**

Fungal Growth - (a) Over 30 genera of fungi have been detected on hair after death. (b) Order of succession - (I) Initial stage colonization - Hair is first occupied by fungi with high competitive saprophytic ability able to rapidly utilize the less complex nutrients [for hair composition, please see **ch 3**] (II) Mid stage colonization - occupied by fungi with less competitive saprophytic ability. Both initial and mid stage colonization is by non-keratinophilic fungi (III) Last stage colonization - Keratinophilic fungi. Utilize keratin, the most resistant part of hair.

F. Muscle Proteins and Enzymes

(1) **Calcineurin A (CnA)** - .es in skeletal muscle after death.
(2) **Creatine phosphokinase (CPK)** -.es after death
(3) **Myofibrillar protease** - -es after death
(4) **Protein phosphatase 2A (PP2A)** - -es after death in first 24 h.

G. Entomology of the Cadaver

Entomology is the scientific study of insects. ***Forensic entomology*** is the application of insect biology for the purposes of law and administration of justice. An important area of forensic entomology is estimation of time of death, where the order of appearance of flies on **dead body** often indicates the probable

time of death.

Table 6: Life cycle of Musca domestica (housefly)

S.No	Developmental stage	Summers	winters
1.	Flies lay eggs	Within minutes	Within hours
2.	1st instar hatches from eggs	8 h	1 d
3.	1st instar to 2nd instar	1 d	4 d
4.	2nd instar to 3rd instar	1 d	4 d
5.	3rd instar to pupa	3 d	8 d
6.	Pupa to adult	5 d	5 d
	Total	10-11 days	22 days

H. The Scene of Death

(1) **Cell phone records** – Death must have occurred between last call attended and first call unattended. Not foolproof, since unattended call may be intentional and attended call may be by someone else.

(2) **Milk bottles** - Number of uncollected milk bottles at door step indicate number of days person is dead. Useful if there was a **lonely occupant living in a house.**

(3) **Newspapers** - Earliest newspaper uncollected at door step gives the possible date of death. Death must have occurred one day previously. **Disposal of body parts wrapped in newspapers** may indicate the day of death. Please see

Ruxton case [**ch 6**].

(4) **Stopped wrist watch** – in cases of drowning, fall from height, blunt trauma etc where weapon struck watch and stopped it, may rarely give exact time of death. Firearm wounds of chest may stop a pocket watch exactly at the time of death. **Case study** - The author was once involved in a case, where a person was shot in the chest. He used to keep a pocket watch in the upper left hand pocket of his vest. The bullet had scraped the watch before entering the chest. The watch had stopped showing **8:47pm**.

(5) **Dates** on uncollected mail

(6) **Sales receipts and dated slips of paper** in deceased's pockets

(7) **Statements from neighbors** – when they last saw the deceased

(8) **State of food on the table** – fully completed, half eaten etc.

(9) **State of dress** – whether the person was in normal dress or night dress

(10) **Corpse lying on grass** – Look at grass underneath the corpse. The state of drying, yellowness etc can indicate time of death.

VII. PRESUMPTION OF SURVIVORSHIP AND DEATH

[B] **S.107 Indian Evidence Act – Presumption of survivorship** - When the question is whether a man is alive or dead, and it is shown that he was alive within **30 years**, the burden of proving that he is dead is on the person who affirms it.

Memory Aid 7: S.107, IEA

Section hundred and **S**even [**S.107, IEA**] deals with **S**urvivorship

[C] **S.108 Indian Evidence Act – Presumption of death** - When the question is whether a man is alive or dead, and it is proved that he has not been heard of for **7 years** by those who would naturally have heard of him if he had been alive, the burden of proving that he is alive is shifted to the person who affirms it.

Fig. 9.13: **(1)** Wall clock stopped by a stray bullet indicating the exact time of death. **(2)** In this case of death by burns, the watch on left hand was found stopped at the exact time of burns, due to intense heat. **(3)** A pocket watch completely burnt and stopped at the time of burns shows the exact time of burns.

1 Balci Y, Basmak H, Kocaturk BK, Sahin A, Ozdamar K. The importance of measuring intraocular pressure using a tonometer in order to estimate the postmortem interval. Am J Forensic Med Pathol. 2010 Jun;**31(2)**:151-5.

10. Artifacts

I. GENERAL

[A] An artifact [L. arte, by skill; factum, thing made] is (i) any change caused to a body after death ***unintentionally*** and which is (ii) physiopathologically unrelated to the natural state of the body tissues or to the disease process to which the body was subjected to before death.

II. CLASSIFICATION

Table 1: Classification of artifacts

1. Artifacts introduced between death and autopsy
2. Artifacts introduced during autopsy

A. Artifacts Introduced Between Death and Autopsy

1. Agonal artifacts (produced just after death)

Regurgitation of gastric contents - in respiratory tract after death may be confused with genuine antemortem aspiration.

2. Resuscitation artifacts

Injection marks

3. Artifacts due to handling of the body

Abrasions – Due to inept dragging of the body by the police from the scene of crime to their vehicle for transportation.

4. Artifacts related to postmortem lividity

Prinsloo and Gordon artifact - Generally thought to be due to pm lividity, but is actually due to "leakage" of blood outside the vessels. Described under the heading "**Postmortem hemorrhage**".

B. Artifacts Introduced During Autopsy

1. Air in blood vessels

Can be introduced by: pulling the dura in the sagittal line [air enters in bv at the top of brain].

2. Skull fractures

Using chisel and hammer to open skull – May produce artificial fractures [usually in the middle fossae], or may cause fractures already present to enlarge. Presence of a blood streak within the fracture can sometimes help to differentiate artifactual fractures.

3. Visceral damage

(1) **Aorta** – Descending aorta may show transverse intimal tears, if neck structures are pulled too hard to eviscerate thoracic viscera. May cause tears of neck structures also.

(2) **Brain** – rough handling during removal may produce tears of midbrain

(3) **Liver** – Rough handling during removal may produce tears of diaphragmatic surface which resemble antemortem lacerations.

4. Extravasation of blood

Spurious air embolism – cutting of bv during autopsy" sucking of air in circulation"may enter cerebral, coronary and other bv"appear like a genuine antemortem air embolism.

11. Injuries: Classification and Medicolegal Aspects

I. INTRODUCTION

A. Injury

1. Legal definition

Any harm whatever illegally caused to any person, in body, mind, reputation or property (section 44 IPC). In general, it is **violation of another's rights** for which the **law** allows an action **to recover damages**. **Ex** - (i) "Z" inflicts burns on "P's" body. He has caused injury [**harm to body**] (ii) "Z" threatens "P" on phone. He has caused injury [**harm to mind**] (iii) "Z" spreads false rumors that "P's" character is bad. He has caused injury [**harm to reputation**] (iv) "Z" burns "P's" car. He has caused injury [**harm to property**].

Memory Aid 1: Definition of Injury

BMR is an important concept of **P**hysiology - **B**ody, **M**ind, **R**eputation, **P**roperty

2. Medical definition

Damage inflicted on the body by an external force. Only the 1st part of legal definition constitutes medical definition. For other significant terms which have different **medical** and **legal** definitions, pl see **ch 26**.

B. Wound

A break in the natural continuity of any of the tissues of the living body.

C. Trauma

[from Gk traumata, "to wound", "I injure"] A physical or psychological injury [**physical trauma**, **psychological trauma**].

II. CLASSIFICATION OF INJURIES

A. Medical Classification

Doctors when communicating among themselves would refer to this classification.

(1) **Mechanical injuries** - (a) Due to blunt force - (i)Abrasions (ii) Contusions (iii) Lacerations (iv) Fractures and dislocations (b) Due to sharp force - (i) Incised wounds (ii) Chop wounds (iii) Stab wounds (c) Firearms - Firearm wounds

(2) **Thermal injuries** - (a) Due to cold - (i) With tissue freezing (I) Frostbite (II) Frostnip (ii) Without tissue freezing (I)Trench foot (II) Chilblains (b) Due to heat - (i) Burns (ii) Scalds

(3) **Chemical injuries** - (a) Due to corrosive acids (b) Due to corrosive alkalis (c) Due to corrosive salts

(4) **Physical agents** - (a) Electricity - (i) High tension (ii) Low tension (b) Lightning (c) X-rays (d) Radioactive substances

(5) **Explosions**.

B. Legal Classification

Courts would generally follow this classification.

1. Hurt

Hurt would fall **u/s 319, IPC**. Law does not mention the word "**simple injury**" anywhere, and thus this term should best be avoided. By common usage, "simple injury" is used as a synonym of **hurt**.

2. Grievous hurt

Grievous hurt would fall **u/s 320, IPC**. All injuries which are not simple are grievous and vice-versa.

C. Medicolegal Classification

Doctor when advising the court would follow this classification.

1. Suicidal injuries

Hesitation cuts etc.

2. Homicidal injuries

Fatal wounds on head, chest, abdomen. Would not include non-fatal defense wounds [**ch 12**].

3. Accidental injuries

4. Self inflicted injuries

Caused by self to mislead police (Fabricated injuries)

5. Defense wounds

Received by victim, mostly on his hands, during defense. Generally non fatal [**ch 12**].

6. Iatrogenic injuries

Surgical incisions, thoracotomy wounds etc.

III. HOMICIDE

Homicide is killing of a human being by another human being. It can be classified according to the following scheme (**Table 1**).

Table 1: Classification of Homicide

1. **Lawful:** (a) Justifiable (b) Excusable
2. **Unlawful:** (a) Rash and negligent act (b) Culpable homicide not amounting to murder (c) Culpable homicide amounting to murder

A. Lawful

1. Justifiable homicide

Justifiable homicide is one in which the person committing it is at ***no*** fault in a ***legal sense***. It is commanded or authorized by law. Usually a ***legal officer*** is involved who commits the homicide in order to carry out a ***perfectly legal [justified] task***.

2. Excusable homicide

Excusable homicide differs from ***justifiable homicide*** in that one who commits an excusable homicide is at fault to some degree, but the degree of fault is ***not enough to constitute a culpable homicide***.

B. Unlawful

In **unlawful homicide**, the **degrees of culpability** (degrees of fault) would differ in different cases.

1. Culpable homicide (S.299, IPC)

Culpable homicide is causing death by (1) doing an act with the intention of causing death, or (2) with the intention of causing such bodily injury as is likely to cause death, or (3) with the knowledge that he is likely by such act to cause death, commits the offence of **culpable homicide**.

2. Murder (S.300, IPC)

Culpable homicide is **murder** (1) if the act by which the death is caused is done with the **intention** of causing death, or (2) if it is done with the **intention of causing such bodily injury** as the **offender knows to be likely to cause the death** of the person to whom the harm is caused, or (3) if it is done with the **intention of causing bodily injury** to any person and the bodily injury intended to be inflicted is **sufficient in the ordinary course of nature to cause death**, or (4) If the person committing the act knows that it is so **imminently dangerous** that it must, in all probability, cause death or such bodily injury as is likely to cause death, and commits such act without any excuse.

IV. LEGAL PROVISIONS REGARDING HOMICIDE, SUICIDE AND RELATED ACTS

***"imp; **"v.imp**

A. IPC

(1) **S.299** IPC***"Definition of culpable homicide

(2) **S.300,** IPC***"Definition of murder

(3) **S.301,** IPC"Culpable homicide by causing death of person other than person whose death was intended

(4) **S.302,** IPC***"Punishment for murder. Death or life imprisonment and fine. **Honor killing** - An ***honor killing*** is the murder of a person by one or more fellow family members when they believe the victim brought dishonor upon the family. Victims are usually unmarried females who engage in lover affairs, or marry someone from a different caste. These cases are tried u/s 302, IPC.

Honor killings had been reported in Egypt, India, Iran, Jordan, Lebanon, Morocco, Pakistan, Syrian Arab Republic, Turkey, Yemen, and other Mediterranean and Gulf countries.

(5) **S.303,** IPC"Punishment for murder by life-convict. This is the only section in the entire IPC, where only death has to be given. ***No fine***.

(6) **S.304,** IPC*****"Punishment for culpable homicide not amounting to murder (imprisonment for life or fine or both)

(7) **S.304A,** IPC******"Causing death by negligence. 2 y, or fine, or both

(8) **S.304B,** IPC******"Dowry death. Punishment minimum 7 y. ***No fine***.

(9) S**.305,** IPC"Abetment of suicide of child or insane person. Punishment death or imprisonment for life and fine.

(10) **S.306,** IPC"Abetment of suicide. Punishment"10 y+fine

(11) **S.307,** IPC"Attempt to murder. Punishment"10 y+fine

(12) **S.308,** IPC"Attempt to commit culpable homicide. Punishment"3 y, or fine or both (if hurt is not caused to the victim); 7 y, or fine or both (if hurt is not caused to the victim)

(13) **S.309,** IPC*****"Attempt to commit suicide. Punishment"1 y, or fine or both.

Salient features:

(i) This is the only section in the entire IPC, where a person ***cannot be prosecuted*** if he is successful in his crime. (ii) **Dyadic death** ("pairs of death") is homicide followed by suicide or a double suicide pact (e.g. a pair of lovers committing suicide together, because they were not allowed to marry. Other reasons may be infidelity, jealousy, poverty, property disputes or unemployment). (iii) **Multiple methods of suicide** – If a person is unable to commit suicide by one method, he may resort to another method. At autopsy evidences will be found of all attempts [e.g. a person consuming rat poison first. When he finds he is still alive, he may slash his wrists, and finally jump out of the window] (iv) **Suicide attack** – Human bomb in order to kill a large number of persons. (v) **Most common method of suicide** – Varies from place to place, and from culture to culture. In one South Indian study organophosphorous poisoning was found to be the most commonly used method for suicide (25.4%). (vi) **Decriminalization of suicide** – **S.124** of the proposed **Mental Health Care Bill 2013** attempts to decriminalize suicide. It says "Notwithstanding anything contained in section 309 of IPC, any person who attempts to commit suicide shall be presumed, unless proved otherwise, to be suffering from mental illness

at the time of attempting suicide and shall not be liable to punishment under the said section" [**S.124(1)**]. Furthermore it says, "The appropriate Government shall have a duty to provide care, treatment and rehabilitation to a person, having mental illness and who attempted to commit suicide, to reduce the risk of recurrence of attempt to commit suicide." [**S.124(2)**] However, pending the passage of the Mental Health Care Bill 2013, **S.309, IPC** is yet to be limited or repealed.

Table 2: Types of homicide according to decreasing culpability

S.No	Type of homicide	IPC section applicable (definition)	Punishment	Equivalent term in some foreign countries
1.	Culpable homicide amounting to murder	S.300, IPC	Capital punishment or life+fine (S.302, IPC)	First degree murder
2.	Culpable homicide not amounting to murder	S.299, IPC	Life imprisonment +fine (S.304, IPC)	Second degree murder (**voluntary manslaughter** in the UK)
3.	Causing death by negligence	S.304A, IPC	2 years +fine (S.304A, IPC)	Third degree murder (**Involuntary manslaughter** in the UK)
4.	Participating in a related act, which led to murder	-	-	Fourth degree murder

B. Other Acts

S.25, *Hindu Succession Act, 1956* - A person who commits murder or abets the commission of murder shall be disqualified from inheriting the property of the person murdered, or any other property in furtherance of the succession to which he or she committed or abetted the commission of the murder.

V. HURT (S.319, IPC)**

Hurt means bodily pain, disease or infirmity caused to any person.

Memory Aid 2: Def inition of hurt

BiDI " **B**odily pa**i**n, **D**isease, **I**nfirmity

VI. GRIEVOUS HURT (S.320, IPC)**

[A] ***Grievous hurt*** is hurt of a more serious nature. There are **8 clauses** in the

definition of **grievous hurt**(GH). Clause 8 has further 3 sub clauses. (1) Emasculation (2) Permanent privation of the sight of either eye (3) Permanent privation of the hearing of either ear (4) Privation of any member or joint (5) Destruction or permanent impairing of the powers of any member or joint (6) Permanent disfiguration of the head or face (7) Fracture or dislocation of a bone or tooth (8) Any hurt (i) which endangers life or (ii) which causes the sufferer to be during the space of twenty days in severe bodily pain, or (iii) unable to follow his ordinary pursuits.

Memory Aid 3: Def inition of Grievous hurt

E[1]very **S[2]**tudent **H[3]** as re **member[4]** ed **I[5]**njury's **D[6]**efinition, but **F[7]**orgot it within **20days[8]** of **P[8]**assing **E[8]**xaminations.

[B] **Illustrations** (1) **Emasculation** – Refers to taking away the masculine power (i.e. penetrating power during intercourse) of male. This is the only clause that applies only to males. (i) **Cutting of penis** - X cuts penis of Y"X has emasculated Y"caused GH. (2) **privation of the sight of either eye** – Privation means even partial loss of sight. Operative interference is not to be taken into account. **Ex**. (i) **Retinal detachment** (ii) **Gouging out eyes**. (3) **Permanent privation of the hearing of either ear** – Same examples and reasoning as above, but w.r.t. ear. (i) **Tympanic membrane** - Teacher X slaps student Y"Y's tympanic membrane is ruptured and he cannot hear"X has caused GH (4) **Privation of any member or joint** – A member is any part of the body, which is capable of performing a distinct function, and/or is not able to regrow. Hands, feet are members, but not nails and hairs. Blood is not a member, because it can regenerate. For the same reason skin and body fluids (milk, saliva, tears, urine, CSF, semen etc) are not members. (i) **Nails** - If nail is trimmed, it is not GH (because it can regrow), but if avulsed from base, it is GH (because it cannot regrow). (5) **Destruction or permanent impairing of the powers of any member or joint** – This clause is attracted if privation is not caused to member or joint (i.e. not amputated), but has been destroyed or permanently impaired (i) X strikes a hammer on Y fingers, so that his fingers are reduced to pulp"GH (6) **Permanent disfiguration of the head or face** – **Ex** (i) **Abrasions v lacerations** – Abrasions of face are simple (as they heal without scarring); lacerations of face are grievous (as they heal with scarring) (7) **Fracture or dislocation of a bone or tooth** – **Ex.** (i) **Cut on a bone** is a fracture and thus GH. (viii) **Dislocation of shoulder** – Grievous hurt, because dislocation of a bone occurs. It is also a rare

example of grievous hurt which can be corrected within minutes. (8) **Any hurt (i) which endangers life or (ii) which causes the sufferer to be during the space of twenty days in severe bodily pain, or (iii) unable to follow his ordinary pursuits**.**Exp – Any hurt which endangers life – Pointers** [A] It is an injury which has a 50% chance to end in fatality.

VII. DOWRY DEATHS

A. Legal Provisions Relating to Dowry Deaths

(1) **S. 304B, IPC**"Where the death of a woman is caused by any burns or bodily injury or occurs otherwise than under normal circumstances (i) within **seven years** of her marriage **and** (ii) it is shown that **she was subjected to cruelty or harassment** by her husband or his relative for dowry, such death shall be called "dowry death", and such husband or relative shall be deemed to have caused her death. Punishment is imprisonment **minimum 7 years**; **maximum life**. There is no fine.

(2) **S.498A, IPC**"**Treating a married woman with cruelty** – If the woman's husband or any of his relatives treats her with cruelty, he/she will be imprisoned for up to 3 years and will also be fined. Cruelty has been defined as any willful conduct which is of such a nature as is likely to drive the woman to commit suicide or to cause grave injury or danger to her life. It also includes her harassment with a view to coerce her for dowry. Habitual drunkenness by husband has been regarded as cruelty to woman (please see ch 40).

VIII. TORTURE

A. Definitions

[A] **General Definition** - ***Torture*** (Latin tortus, to twist) is infliction of intense pain (e.g. from burning, crushing, wounding etc) to punish, coerce, or afford sadistic pleasure. [B] **Declaration of Tokyo (1975)** - Torture is deliberate, systematic or wanton infliction of physical or mental suffering by one or more persons acting alone or on the orders of any authority, to force another person to yield information, to make a confession, or for any other reason.

B. Methods

1. Physical torture

(1) **Beating:**

(i) Commonest form of torture

(ii) Instrument used – blunt instruments like belt, bicycle chains**,** lathi, metal or wooden bar, whip etc.

(iii) Target and injuries produced - (a) Back [Most common] - abrasions, contusions, lacerations are produced (b) Abdomen – rupture of viscera may occur (c) Head – head injury, skull fractures, intracranial hemorrhages.

(iv) Detection (a) In the living - via ultrasound (b) In the dead- Bruising in the back is not seen superficially because of thick fascial planes [same is true of soles also – please see falanga below]. Deep dissection is necessary to reveal bruising. Aseptic necrosis may be seen.

(2) **Chepuwa** - A Bhutanese torture technique. Both thighs or legs are tightly clamped with bamboo for a number of days. The 2 sides of the clamps are pressed with torturer's legs or he may stand on the 2 sides of the clamps.

(3) **Electric torture** - Also called cattle prod. Types

(i) Picana: (a) victim is strapped to a wooden table and wetted to aid the flow of current. (b) Current [DC, usually powered by an automobile battery] is applied to sensitive parts of the body eg head, temples, eyes, mouth, teeth, tongue, breasts, nipples and genitalia (c) a doctor is present to make sure that the victim has no heart problems and can survive the interrogation (d) Effects - May result in the loss or fracture of teeth.

(ii) La parrilla (metal grill): Electric shocks given while the victim is tied to a metal bed frame.

(4) **Falanga** - [also known as bastinado, falaka, falaqa, foot whipping and phalanga]

(i) Beating of the soles of the feet with canes or rods.

(ii) Favored by perpetrators, because (a) Extremely painful and debilitating [there is clustering of nerve endings in the feet] (b) Foot being weight bearing organ, injuries take long time to heal (c) leaves few physical marks

(iii) Detection – As in beating above. Dissection of soles may be necessary

(5) **Force Feeding** – of boiling water, mustard oil, pepper, saturated salt water,

vinegar, urine and feces

(6) **Forced standing:**

(i) Types (a) standing on both feet (b) On one foot [could be in hot sun to increase pain]

(ii) Called Planton in Latin America and stoika in the Soviet Union. An attractive option for torturers because like water torture, it leaves no marks

(iii) Effects (a) Swelling of the ankles and feet to twice their size within 24 hours (b) moving becomes agonizing (c) Development of large blisters (d) Increasing of the heart rate and fainting.

(7) **Knee capping** - Victim is shot through the knee joint. Common in Northern Ireland.

(8) **Submarino:**

(i) Dry submarino (submarino seco)-Near suffocation by pulling a plastic bag [usually a shopping bag sometimes containing chillies and/or petrol] over the head and face

(ii) Wet submarino (submarino mojado)-Face of the victim is forced under water usually contaminated with excrement, urine, vomit or blood. Victim may die of drowning. Feces [or other extraneous material] in lungs at autopsy.

2. Mental torture

(1) **Blindfolding**

(2) **Deprivation techniques** – Deprivation of sleep by use of continuous high pitched sound. Deprivation of the use of toilet, clean habitable place, isolated captivity [social deprivation]

(3) **Mock execution** - Victim is deliberately but falsely made to feel that his execution or that of another person is imminent.

(4) Providing **false information** to victim – e.g. tragedy involving wife and children

(5) **confinement** - in a dark place

(6) **Threat** of permanent, severe disfigurement to victim or his loved ones [e.g. wife, children]

(7) **Witness torture** – Victim is forced to witness the torture of another person.

C. Medicolegal Aspects

Torture is punishable in Indian law under following sections (a) **S.330, IPC** -

Voluntarily causing hurt to extort confession, or to compel restoration of property is punishable with imprisonment up to seven years, and also fine. (b) **S.331, IPC** - Voluntarily causing grievous hurt to extort confession, or to compel restoration of property is punishable with imprisonment up to ten years, and also fine.

IX. CAUSES OF DEATHS FROM WOUNDS

A. Immediate Causes

1. Shock

Shock is a circulatory disturbance characterized by hypoperfusion of cells and tissues, which in turn may be due to . in the vol of blood or cardiac output. In wounds it is mainly due to hemorrhage, but also because of other causes.

a. Causes

i. Cardiogenic shock

(1) Due to myocardial failure".cardiac output.
(2) Myocardial failure in turn may be due to
(i) extrinsic compression [cardiac tamponade]
(ii) intrinsic myocardial damage [MI]
(iii) outflow obstruction [eg pulmonary embolism]
(iv) Pulmonary embolism
(v) ventricular arrhythmias
(vi) Ventricular rupture.

ii. Hypovolemic shock

Hypovolemic shock is due to loss of blood [eg massive hemorrhage] or plasma volume [eg vomiting, diarrhea, severe burns (***burn shock***)].

Salient features:

(1) When it is due to excessive bleeding, it is known as hemorrhagic, traumatic or wound shock. It is the most common cause of hypovolemic shock in forensic practice.
(2) Bleeding diathesis – Victims with hemophilia or other bleeding diathesis may succumb to relatively minor injuries
(3) Quantity of blood loss – may be approximately judged by observing (a) scene of death – amount of blood lying around the victim
(4) Classification - It could be classified according to (a) etiology (b) source of

bleeding (c) destination of bleeding (d) Time period of bleeding (e) Quantity of blood loss.

Important points of each are as below:

(1) **Etiology** – (a) **Traumatic** – Due to wounds involving blood vessels, or severe laceration of viscera (b) **Spontaneous** – Bleeding diathesis etc.

(2) **Source of bleeding** - (a) Cardiac (b) Arterial (c) venous (d) capillary

(3) **Destination of bleeding** - (a) External [outside the body] (b) Internal [within the body].

(4) **Time period of bleeding** - (a) Primary hemorrhage - Occurs immediately after injury (b) Secondary hemorrhage - Delayed for several hours (up to 24 hrs) after injury. Due to (i) -in BP, which accompanies recovery from shock (ii) muscular movements, which loosen blood clots, and (iii) erosion and sloughing of vessel walls due to infection [cf; **ch 17**"Primary and secondary head inj and intracranial hemorrhages].

(5) **Quantity of blood loss:** (i) Sudden loss of blood is more dangerous that the same quantity of loss occurring slowly. (ii) Measuring blood loss - (a) Clot of a fist size – contains 400-500 ml.

iii. Septic shock

(1) **Causes:** (i) Overwhelming microbial infections [bacterial and fungal] (ii) Superantigens [eg toxic shock syndrome]

(2) Vasodilation and peripheral pooling of blood occurs as part of a systemic immune reaction to infection.

(3) **Mechanism of death** - is unclear; aside from -ed lymphocyte and enterocyte apoptosis there is only minimal cell death, and patients rarely have refractory hypotension.

(4) Septic shock due to Gram –ve infections are sometimes called **endotoxic shock**.

iv. Less common causes

(1) **Anaphylactic shock** - denotes systemic vasodilation and -ed vascular permeability caused by an IgE–mediated hypersensitivity reaction. Acute widespread vasodilation results in tissue hypoperfusion and hypoxia.

(2) **Neurogenic shock** - anesthetic accident, spinal cord injury. Loss of vascular tone occurs"peripheral pooling of blood.

(3) **Surgical shock** – Occurs during or immediately after surgical procedures. Occurs due to combination of:

(i) anesthesia
(ii) loss of blood and plasma
(iii) infections
(iv) emotional factors
(v) liberation of substances from severely traumatized tissues.

b. Stages of shock

(1) If the injury is massive (eg a massive hemorrhage from a ruptured aortic aneurysm), death is immediate.
(2) In all other cases, shock evolves through **three** general stages:
(i) **Initial nonprogressive stage**- Reflex compensatory mechanisms are activated and perfusion of vital organs is maintained
(ii) **Progressive stage** - characterized by tissue hypoperfusion and onset of worsening circulatory and metabolic imbalances, including acidosis
(iii) **Irreversible stage** - sets in after the body has incurred cellular and tissue injury so severe that even if the hemodynamic defects are corrected, survival is not possible.

c. PM appearances

i. General

(1) **Circulatory changes:**
(i) Congestion of all internal organs
(ii) Edema of viscera
(iii) Petechial hemorrhages in serous membranes
(2) **Degeneration and necrosis** - of various organs.

ii. Brain

(1) Changes occur if victim survives for 12-24 h.
(2) **Findings:**
Ischemic encephalopathy: Sequence – shrinkage or swelling of neurons"nucleus becomes pyknotic"death of neurons"replaced by fibrillary gliosis.

iii. Heart

(1) **Fatty changes:** (i) After 18 h of survival - seen in about 20% cases. (ii) 3-4 days – become well marked in all cases
(2) subendocardial hemorrhages.

iv. Lungs

Findings are same as in ARDS [please see below].

v. GIT

(1) **Contents** – blood stained fluid. Blood is dark red and fluid.

(2) **Mucosa** – congested and edematous, shows hemorrhagic erosions, ulcerations and necroses [Hemorrhagic enteropathy].

vi. Liver

(1) **Gross** – no appreciable change

(2) **microscopic** – Due to perfusion deficit. Fatty changes after 18 h. Gradually extend from central areas to lobules.

vii. Spleen

Enlargement and softening [in septic shock].

viii. Kidneys

(1) **Pathology** - Changes are due to .in renal blood flow (RBF)"ischemic renal injury. Changes begin within 24 h. Become marked in 7-10 days.

(2) **Shock kidney:**

(i) Gross – Normal in size. May be slightly enlarged in some cases

(ii) Microscopic - Acute tubular necrosis.

ix. Adrenals

Focal lipid depletion in cortical cells – begins after 24 h. Well marked in 3-4 days.

x. Other organs

Necrotic foci – in lymph nodes, pancreas, spleen

2. Reflex vagal inhibition

Please see **ch 8**.

3. Air embolism

Air embolism [syn, gas embolism] is entry of air bubbles in vascular system. It is an immediate cause of death in certain wounds e.g. wounds of neck.

Types:

(1) Venous – more common. So common that when only the phrase “air embolism” is mentioned, it is presumed to be venous air embolism [please also see **ch 11**].

(2) Arterial – caused when air in sufficient quantities enters (i) a vein of the pulmonary system"reaches left side of the heart"blocks arterioles and

capillaries in different parts of the body [brain, heart] or (ii) directly into the arteries of the systemic circulation.

a. Causes of air embolism

i. Venous embolism

(1) **Artificial respiration** – forced respiration by mechanical respiratory aids"rupture of overinflated alveoli"interstitial pulmonary emphysema"air penetrates pulmonary lymphatics"enters venous system via thoracic duct.

(2) **Criminal abortions** – when air or soap solutions are injected in the uterine cavity [**ch 26**]

(3) **Crush injuries** of chest

(4) **Decompression sickness** or Caisson's disease [may cause both arterial and venous embolism]

(5) **Oral genital sex**, especially in a pregnant woman [**ch 25**]

(6) **Surgical** and other **iatrogenic procedures:**

(i) **Central venous catheterization** (CVC), including central lines, pulmonary catheters, subclavian artery catheterization, hemodialysis catheters and Hickman (long-term) catheters

(ii) cesarean delivery, **manual extraction of placenta**

(iii) hemodialysis

(iv) **Injection of air under pressure** in fallopian tubes (to test patency)

(v) **IV injections** given with a faulty technique

(vi) **Neurosurgical procedures**, especially those performed in the Fowler's (sitting) position (e.g., air encephalography, posterior fossa surgeries)

(vii) **Otolaryngological surgeries**

(viii) **positive pressure ventilation** in newborn infants [over distension of airspaces"alveoli rupture+ pulmonary vascular damage"entry of air into pulmonary vasculature]

(ix) thoracocentesis

(x) During **transfusion**, when positive pressure is being used.

(7) **Wounds of**

(i) neck involving jugular or subclavian veins

(ii) craniofacial region (especially wounds of sagittal sinus)

(iii) chest and abdomen.

ii. Arterial embolism

(1) Crushing and penetrating wounds the chest [may penetrate a lung vein and a bronchiole and allow air to enter vein]
(2) Surgical procedures on the thorax [any cardiac surgical operation that uses extracorporeal bypass, thoracentesis, thoracic surgery]. Because the pulmonary vein pressure is low [10 cm H_2O], any factor producing an -ed pressure gradient between airway and pulmonary vein allow entrance of air into the vein.
(3) Paradoxical embolism
(4) overexpansion of the lung by decompression barotrauma.

b. Pathophysiology

If volume of air is large - Air enters venous system" carried to right heart and pulmonary arteries" mechanical obstruction and churning [turbulent flow] of blood"Production of erythrocyte and platelet aggregates, fat globules, fibrin and froth [complexes of air bubbles, microbubbles]"further occlusion of vasculature"death.

c. Paradoxical air embolism

(1) Occurs when air passes into the arterial system via anomalous structures
(i) atrial or ventricular septal defect
(ii) patent foramen ovale [incidence in general population "**ch 27**]
(iii) pulmonary AV malformations [AVM].
(iv) In the absence of AVM, gas bubbles may pass when (a) there are large volumes of venous air (b) administration of vasodilators and (c) volatile anesthetic agents [have a pulmonary vasodilating effect].
(2) Arterial tree is embolized paradoxically. The paradox is how the air entering the veins can embolize arteries. Mechanism is same as that of fat embolism [please see below].

d. Minimum amount of air that causes fatal air embolism

(1) **Venous embolism - 100 ml** in adults. If injected in a limb vessel, comparatively larger volumes are required, as air is absorbed rapidly.
(2) **Arterial embolism – 2-3 ml**, because air travels directly from lungs to the left side of the heart, from where it is forced into the coronary or cerebral arteries [**ch 5**].

e. Fatal period

Death from air embolism [venous or arterial] occurs within a few minutes, and is rarely delayed beyond an hour.

f. Diagnosis at PM

Please see **ch 5**.

4. Mechanical injury to a vital organ

(1) **Most vital organs - brain**, **heart** and **lungs** [**ch 8**]. Severe injury to any of these would result in rapid death.

(2) **Other vital organs** – if severely injured may also result in death, eg liver, spleen etc

(3) **Slight injury:**

(i) May result in death, if the organ was previously diseased.

(ii) Some examples where minor trauma may cause death (a) Opening up of recently stitched wounds [eg vital abdominal wounds] (b) Perforation of ch intestinal ulcer (c) Rupture of an aneurysm, enlarged liver or spleen.

(iii) In such cases, if the offender (a) did not have intention to kill (b) did not know the condition of victim and (c) amount of injury caused would not be fatal to a normal person, then he cannot be charged for murder or even culpable homicide. He would be charged with causing hurt or grievous hurt [please also see “culpable homicide and murder compared” above].

5. Natural diseases

A natural disease may be aggravated by trauma. Please see below "Trauma and disease.

B. Remote Causes

(1) These are cases when victim dies from the effects of trauma, long after it was inflicted.

(2) **Liability:**

(i) If cause of death can be traced to the original trauma, assailant is responsible.

(ii) If medical attention was provided and was allegedly negligent in nature, liability may lie on the doctor also [please see “surgical operation” below].

1. Crush syndrome

Crush syndrome (traumatic rhabdomyolysis, Bywaters' syndrome) is a life threatening medical condition characterized by shock and renal failure following a crushing injury to skeletal muscle.

2. Disseminated intravascular coagulation (DIC)

DIC is a state of hypercoagulation that occurs in a variety of disease states, including severe trauma, particularly to the brain.

3. Embolism

An ***embolus*** is defined as a detached intravascular physical mass that is carried by the blood to a site distant from its point of origin. 95% of all emboli consist of thrombi dislodged from an intravascular thrombus.

4. Gangrene or Necrosis

Gangrene is death of an area of the body accompanied by putrefaction.

Salient features:

Types: (i) Dry gangrene – (a) Mechanism – Injury" thrombosis"loss of blood supply (ii) Wet [moist] gangrene - (a) Mechanism - untreated infected wound"bacterial infection"Swelling"stoppage of blood flow"invasion of muscles by bacteria. Gas gangrene is a special type of wet gangrene caused by Clostridia.

5. Infection

Caused by:

(1) Organisms normally present on body surfaces - (i) B. pyocyaneus (ii) Friedlander's bacilli

(2) Organisms invading tissues from environment - (i) C. diphtheriae (ii) Cl. tetani

(3) Organisms from both body and environment - (i) Cl. welchii (ii) Coliform bacilli (iii) Staphylococci (iv) Streptococci.

6. Neglect

(1) **Negligence:**

(i) of doctor, eg not paying attention to complications

(ii) of patient himself. Willful disobedience, eg not taking medicines
(2) **Improper treatment**

7. Surgical operation

Surgical operation, anesthesia or other medical intervention–for treating injury may cause death.

8. Supervention of disease from a traumatic lesion

(1) **Damage of artery by bullet**"false aneurysm" rupture"death
(2) **Fibrous scar tissue formation** - Traumatic lesion" healing"scar tissue formation"scar tissue contracts"complications
(i) fibrous scar in hollow muscular organ–obstruction, stricture
(ii) fibrous scar in arteries"under constant tension"traumatic aneurysm"rupture" death
(iii) Abdominal wound"heals"fibrous scar" strangulated hernia"death
(3) **Injury to spinal column**"paraplegia"bed sores, septic cystitis, general exhaustion"death
(4) **Thoracic trauma**"injury to coronary artery"coronary thrombosis"death.

X. AGE OF WOUNDS

Age of wounds can be determined by observing the amount and extent of healing. Age of wounds can be determined by (i) Gross changes (ii) Histopathology (iii) Enzyme histochemistry (iv) Immunohistochemistry. For ageing by gross changes, please refer to individual types of injuries (abrasion, contusions etc).

A. Enzyme Histochemistry

[A] ***Enzyme histochemistry*** is a technique to demonstrate enzymes in tissues.
[B] **Sequence** - Immediately after a wound is produced, two zones can be demonstrated around it 1.a central zone 0.2-0.5mm wide and (1) a peripheral zone 0.1-0.3mm wide. The enzymatic activity .s in central zone and -s in the peripheral. The technique can be combined with **biochemical examination** to detect vasoactive amines (serotonin and histamine), which are the earliest to appear (**Table 3**).

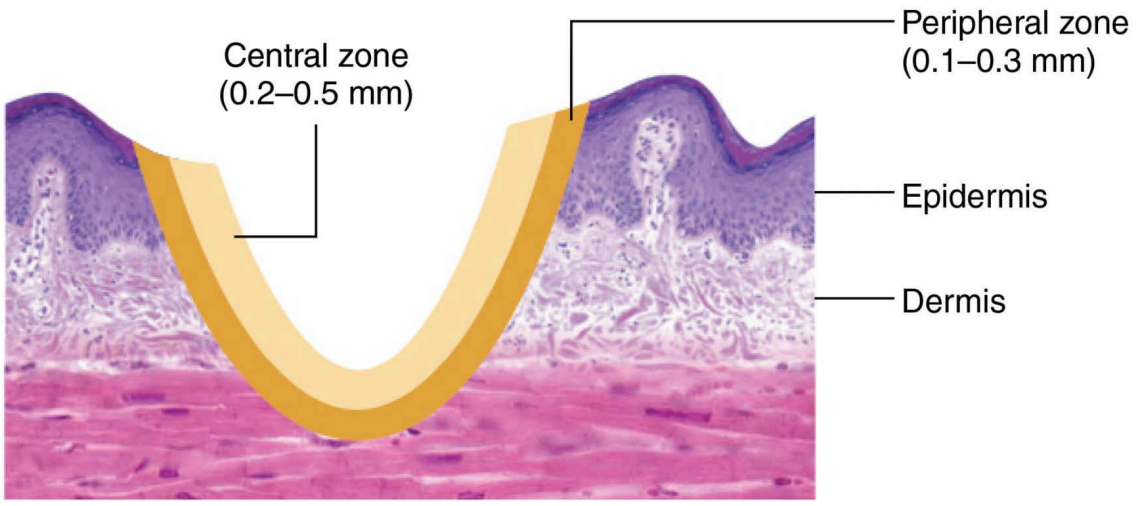


Fig. 11.1: Histochemical changes according to zones in an antemortem wound

Table 3: Age estimation of wound by histochemistry

S.No	Age of wound	Vasoactive amine/Enzyme
1.	10 minutes	Calpains, cathepsins, Serotonin
2.	30 minutes	Histamine
3.	1 hour	ATPase and esterase
4.	2 hours	Aminopeptidase
5.	4 hours	Acid phosphatase
6.	8 hours	Alkaline phosphatase

Memory Aid 4: Histochemistry of wounds

Remember like this.

• **F**irst to appear corresponds with **F**ree histamine; and

Se**C**ond to appear corresponds with **S**erotonin, **C**alpains and **C**athepsins. But this is not so.

So transpose them. Thus we get following:

✦ **F**irst to appear [**10 min**] " **S**erotonin, **C**alpains, **C**athepsins

✦ **S**e **C**ond to appear [**30 min**] " **F**ree histamine

• Last two are **acids** and **alkalis** respectively. Thus we have:

✦ **4 h** " Acid phosphatase

✦ **8 h** " Alkaline phosphatase

B. Immunohistochemistry

Table 4: Immunohistochemical determination of age of wound

S.No	Age of wound	Protein which can be demonstrated by IHC
1.	5 minutes	P-selectin
2.	10-20 minutes	Fibronectin
3.	1 hour	E-selectin
4.	1.5 hours	ICAM-1 (Inter-Cellular Adhesion Molecule 1)
5.	2 days	Tenascin
6.	3 days	Collagen III, V, VI
7.	5 days	Collagen I

XI. VOLITIONAL ACTS AFTER INFLICTION OF FATAL WOUNDS

In severe and fatal injuries, the question often arises, if the victim could have performed some volitional acts involving physical and mental exertion eg speaking, walking, throwing, killing someone back in revenge etc. after receiving the injuries.

(1) **Length of time victim can live depends on**

(i) nature of injury
(ii) Rate of bleeding
(iii) Degree of associated shock
(iv) Different individuals may react differently to same injury
(2) **Trauma to brain** -
(i) Frontal lobe damage "does not incapacitate immediately.
(ii) If brain stem is destroyed [as in judicial hanging]" immediate incapacity
(iii) Severe injuries to brain "(a) victim may survive rarely although the extent of damage is expected to have caused instant death. (b) May remain unconscious for several minutes before finally succumbing to injuries (c) Death may occur with no obvious naked eye changes to the brain
(iv) Single stab wound of skull and brain"May not be immediately fatal, and victim may walk, run or do some other voluntary activity before death.

XII. ANTEMORTEM VS. POSTMORTEM WOUNDS

(1) **AM v PM wounds** – [L. ante, before; post, after; mortis, death]. PM wounds may be caused by animals [dogs, cats, rodents etc], deliberate attempts to confuse police, mutilomaniacs, ***lust murderers*** [**ch 25**] etc.
(2) **Differences** - In many situations, it becomes vital to know if injuries are antemortem [AM] or postmortem [PM].
(3) **Diff** - **Table 5**.
(4) **Perimortem wounds** [L. peri, around; mortis, death] – Wounds caused during supravital period [**ch 8**]. Since molecular death has not occurred, cellular reaction and enzyme histochemistry may be +ve. However since heart has stopped, spurting etc may not be present.

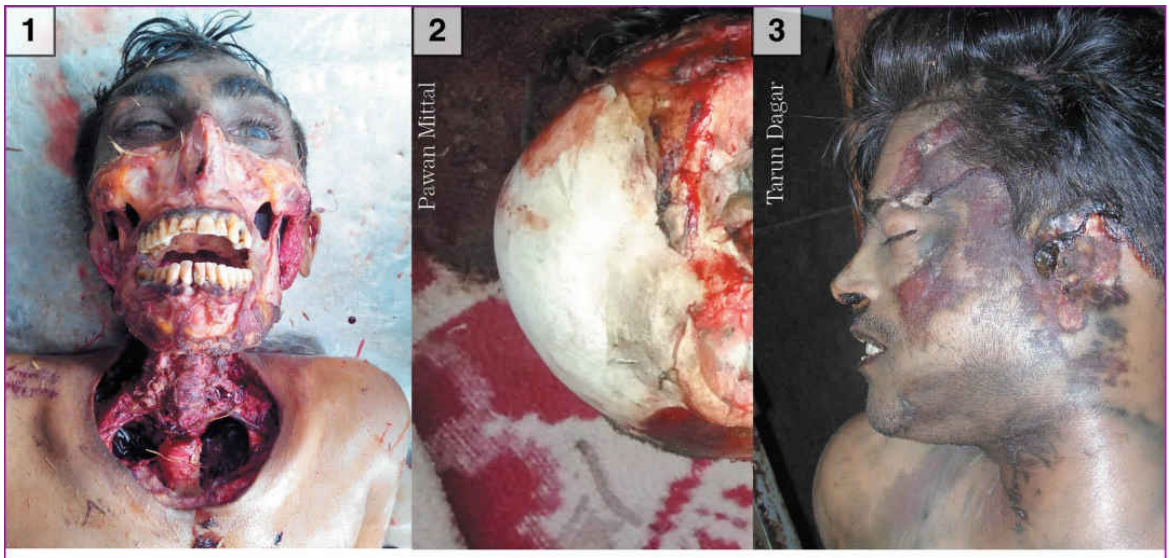


Fig. 11.2: **(1)** Gnawing by rodents **(2)** GPM Mutilation by dogs. Typical PM injuries. Note absence of vital reaction. Projecting soft parts, eg nose, eyelids, ear lobes, penis etc are chosen. **(3)** GPostmortem removal of auricle by assailants for golden ear rings. Please note absence of bleeding and other vital signs excluding antemortem injury. Postmortem gnawing by rats is excluded because much softer eyes are unharmed, as well as nose and lips.

Table 5: Differences between antemortem and postmortem wounds

S.No	Feature	Antemortem wounds	Postmortem wounds
1.	Hemorrhage	Abundant, copious,	Slight or none
2.	Source of hemorrhage	arterial	Venous
3.	Spurting	Present on body, clothes and in the vicinity, eg walls	No spurting
4.	Extravasation of blood	Into surrounding tissues	No such extravasation
5.	Staining of wound edges	Yes; resist washing	No staining; even if stained, can be washed easily
6.	Coagulation	Blood firmly coagulated	No coagulation, or soft clot
7.	Wound edges	everted, gaping retracted, Swollen	Gaping is less [muscles are not in tension. Skin elastin fibres retain their tension even after death. Inadvertent cuts made by prosector during reflection of neck flap show wide gaping]
8.	Sings of vital reaction, eg inflammation and repair	Yes	No
9.	Histology	Infiltration by leucocytes, macrophages, fibroblasts etc. Formation of new capillaries	No sign of cellular proliferation and infiltration
10.	Enzyme histochemistry	-ed activity of vasoactive amines and enzymes	No activity.

XIII. MEDICOLEGAL QUESTIONS ON TRAUMA

Following questions on trauma are frequently asked by lawyers in courts.

A. Can There be No Signs of Trauma Externally, Yet Internal Injury May Be so Great as to Cause Death?

Yes, this is possible. In blunt injuries to abdomen (pliable body surfaces), there may be no external signs of trauma, yet liver and spleen may be damaged, and cause enough bleeding to cause death.

B. How Would You Quantify Degree of Trauma?

This is mostly subjective. Trauma is roughly estimated as mild, moderate, severe and extreme.

C. What are the Situations in Which Acceleration-Deceleration Forces Play a Vital Part?

(1) Vehicular accidents [**ch 18**]
(2) Shaken baby syndrome [**ch 27**].

D. Can the Wounds be Altered from their Original Appearance?

Yes, it is possible both in the living and in the dead (i) **In the living**: (a) **by medical treatment**- Kennedy phenomenon (please see chapter on firearms) (b) by healing (ii) **In the dead**: (a) by resuscitative measures (b) by postmortem predators (insects, animals) (c) by decomposition.

E. Is it Possible for a Person to Die During Altercation?

Yes, if the deceased already suffered from heart disease. Sudden altercation may -BP and heart rate. If the coronaries are already thrombosed, it may precipitate a subintimal hemorrhage leading to death.

F. Were More than One Weapon Employed in this Case?

(i) **Gang wars and group fights** - Generally several people attack with different

weapons. Different weapons would produce different injuries, which would enable the pathologist to opine that more than one weapon were used (e.g. if an incised wound and a bruise are present on the body, it can be opined that both a sharp and a blunt weapon were used) (ii) **Suicide** – If a suicide fails by one method (e.g. cutting wrists), he may employ a firearm to end his life. Presence of incised and firearm wounds would indicate, two different weapons.

12. Mechanical Injuries

Mechanical injuries are injuries produced by mechanical force – blunt, sharp or firearms.

I. ABRASIONS

An ***abrasion*** is an injury involving destruction of epidermal layers of the skin and dermal papillae.

Salient features:

(1) **Force** - producing abrasions can be of two types (a) tangential (b) perpendicular.

(2) **Effects** - Tangential force generally produces scratch and graze abrasions, while perpendicular force produces pressure and imprint abrasions (**Fig 12.1**). Most commonly the force involves a mixture of the two.

(3) **Healing** - Abrasions always heal without scarring. If the injury extends to subepidermal areas [below the level of dermal papillae], it would result in superficial scarring. They are termed as superficial lacerations (**Fig 12.2**).

(4) **Weapons** - with rougher surfaces and with more movement produce more severe abrasions.

(5) **On a wet skin** – difficult to see. Become more prominent after the skin dries. Body recovered from drowning must be allowed to dry before inspection begins.

(6) **On drying** – abrasions become dark brown or black

(7) **Description** – Each abrasion must be described separately.

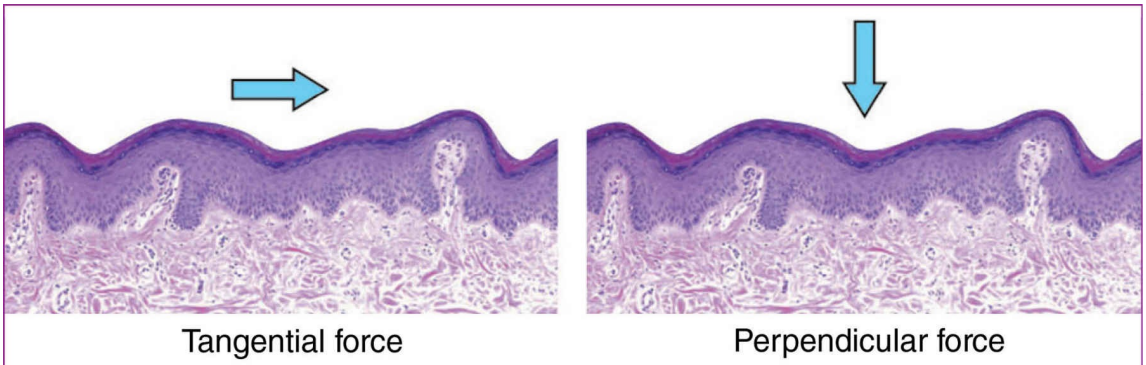


Fig. 12.1: Tangential and perpendicular forces, producing abrasions. Please see text for details

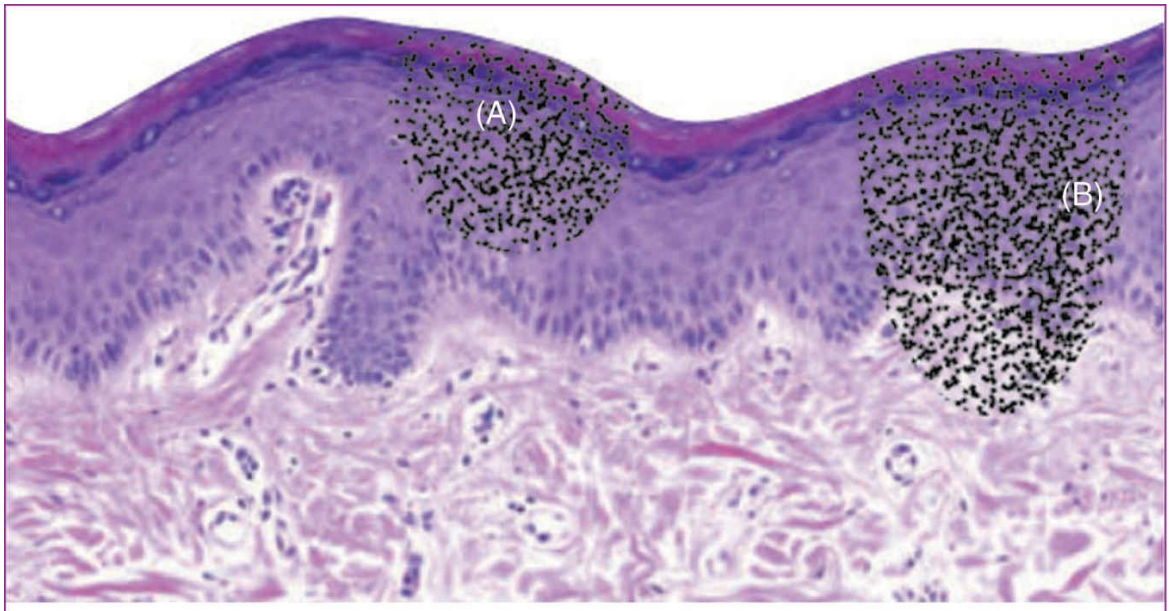


Fig. 12.2: **(A)** A typical abrasion is confined to the epidermis. **(B)** Injury below the level of papillae are superficial lacerations. Please see text for details.

A. Types of Abrasions

1. Scratch abrasions

Scratch abrasions [syn, linear abrasions, scrapes, scratches] are abrasions with appreciable length, but no significant width (**Fig 12.3**).

Salient features:

(1) **How produced** - They are typically produced by sharp, pointed objects such as fingernails, pins or thorns. (a) When produced by tip of knife or razor, they are called point scratches. (b) Nails produce typically curved scratch abrasions, known as semilunar abrasions. Abrasions produced by fingernails may barely be recognizable when very fresh and moist. Upon drying they become dark brown or black and prominent.

(2) **Direction** - Surface layers of the skin are "heaped up" in the direction of the movement. This leaves a clean area at the start of abrasion and epithelial tags at the end.

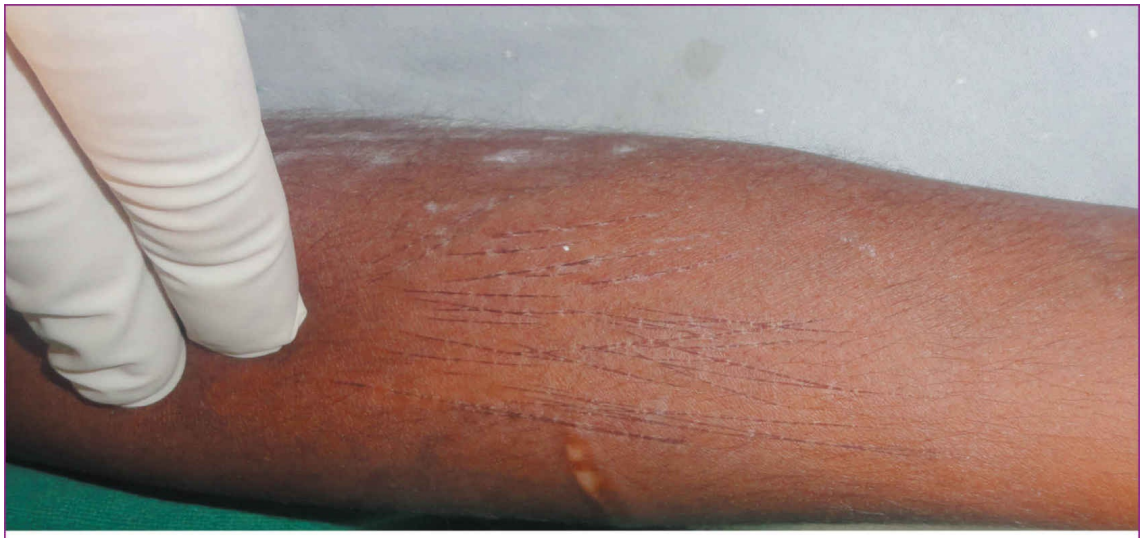


Fig. 12.3: Almost completely healed longitudinal linear abrasions. Pl note that the scab is coming off at places. It is a self-inflicted injury.

Fig. 12.4: Graze abrasions over **(1)** forehead **(2)** Shoulder. Please note exposed dermal papillae as small dark spots **(3)** back.

2. Graze abrasions

Graze abrasion [syn, glancing, grinding, scraping or sliding abrasions] is

essentially a collection of innumerable scratch abrasions.

Salient features:

(1) **Most common** abrasions.

(2) Comprise of uneven, longitudinal parallel lines (grooves or furrows) with the **epithelium heaped up** at the ends of these lines. This heaping up indicates the direction in which force was applied.

(3) Abrasions may extend partially into the dermis because of ***corrugations of the dermal papillae***. Bleeding occurs in such abrasions.

a. Typical examples

(1) **Dragging** at rough road side, as occurs in road side vehicular accidents [**brush burn**, **friction burn**, **gravel rash**, **road rash**, **scuff abrasion**]. Dirt, grit or gravel may be found sticking to abrasions, which would indicate the surface, where the person was dragged.

(2) **Glancing kick** with a boot [application of tangential pressure]

(3) Pedestrian walks into the side of a moving vehicle [**ch 18**"injuries to pedestrians"primary impact inj].

3. Pressure abrasions

Pressure abrasions [syn, crushing or friction abrasions] are abrasions produced by crushing of epithelium, when a ***relatively small force*** is applied perpendicularly to the skin for ***large time periods***.

Salient features:

Impact abrasions also occur by perpendicular forces to the skin. The difference is that in pressure abrasions, the **pressure may not be much**, but the **time period** during which pressure is applied is **considerable**. Because of this, tissue fluid is gradually extruded out and the skin becomes dry and **parchment like**.

Memory Aid 1: Pressure abrasions

Parchmentization is the hallmark of **p**ressure abrasions.

In **impact abrasions**, the **time period is negligible**, but the **pressure applied is tremendous**. Substantial extrusion of fluid is not there and skin is **not dry and parchment like**. (1) **Typical examples:** (i) Corneal abrasion - Use of contact lens for more hours than indicated leads to abrasion of the outer layer of the cornea. (ii) Nappy abrasions (nappy rashes) - in babies due to pressure of

nappies on soft baby skin (iii) Ligature marks - of hanging and strangulation (iv) Shoe bite (v) Teeth bite marks.

4. Impact abrasions

Impact abrasions [syn, contact or imprint abrasions] are abrasions produced by crushing of epithelium, when a ***relatively large force*** is applied perpendicularly to the skin for ***minuscule time periods***.

Salient features:

(1) Impact abrasions are **slightly depressed** below the surface, unless an underlying bruise or local edema bulges the tissues.

(2) **in forcible impacts** – (i) dermis is damaged (ii) there is an underlying bruise

(3) **by a solid object** – often produces abrasions only at the periphery, where the skin is forced downwards with more force.

(4) **Impact by a smooth surface** – abrasion without any pattern. Object cannot be identified [cf patterned abrasions]

(5) **Typical examples:** (i) Headlamp rim marks, or radiator grille marks - in head-on vehicular collisions. (ii) Recoil/muzzle impression - in tight contact wounds [**ch 13**]. (iii) Shoe sole marks – in kicking (iv) Tire marks of a car, bus or truck on pedestrian - in run over accidents. Marks may be soiled with grease, paint flakes and road dirt.

Patterned abrasions

Patterned abrasions are a special subset of pressure and impact abrasions, whereby the ***pattern of the object*** causing them is ***faithfully reproduced on the skin, enabling its identification***.

Examples – (i) bicycle chain (ii) Cat-o-nine tails – It is a special whip having 9 thongs, each containing a number of knots. It produces a very typical patterned abrasion consisting of a series of linear abrasions showing knot marks at specific distances. Sometimes superficial lacerations are also produced. (iii) falling down on a metal grid, (iv) serrated knife, (v) shoe (vi) spiral weaves of electric wires or ropes, (vii) striking by a weapon having a highly patterned surface (viii) weave of coarse fabric.

Fig. 12.5: Patterned abrasion produced by a square based tin can.

5. Miscellaneous

(1) T**attooing** and **pseudo-tattooing - ch 13**
(2) D**icing injuries** – **ch 18**.

B. Age of Abrasions

Abrasions heal from periphery to the centre, by new growth of epithelial cells. Age of abrasions can be determined by (i) gross examination and (ii) microscopic examination [histologically].

1. Gross examination

(1) **Fresh** – Bright red
(2) **1 day** – Blood and lymph dries up. **Bright red scab** forms
(3) **2-3 days** – Reddish brown scab
(4) **4-7 days** – (i) Scab becomes dark brown (ii) Epithelium grows under the

scab and covers defect. Scab becomes ready to fall

(5) >**7days** – Scab dries, shrinks and falls off, leaving depigmented area. Gradually the area would get pigmented.

2. Microscopic (histological) examination

(1) **4-6 hours** – Cellular infiltration in abraded area

(2) **12 h** – Three layers are seen

(i) Surface layer – fibrin and RBCs

(ii) Middle layer – infiltrating polymorphs

(iii) Deep layer – abnormally staining collage

(3) **48 h** –

(i) Scab well formed

(ii) Epithelial regeneration is seen at the margins of scab

(4) **4-5 days** – If abrasion is small, it is completely covered by epithelium

(5) **5-7 d** – subepithelial formation of granulation tissue

(6) **8 d** – Reticulum fibres appear

(7) **9-12 d** – Collagen fibres appear

(8) **>12 d** – Regression starts. Remodelling of epithelium occurs. Becomes thinner.

C. Postmortem Abrasions

(1) **Cause** –

(i) when dead body is dragged to conceal it,

(ii) rolled down a hill

(iii) thrown in water and it buffets against reefs, stones and other objects within water

(iv) animals gnaw or scratch the dead body

(2) **Differentiation** between AM and PM is difficult or even impossible when -

(i) produced just before or after death [impossible even by microscopic examination]

(ii) superficial in nature

(iii) advanced decomposition

(iv) burns occur as added injury. **Table 1** gives some imp differences between the two.

Table 1: Differences between antemortem and postmortem abrasions

S.No	Feature	Antemortem abrasion	Postmortem abrasion
1.	Site	Anywhere on the body	Usually over bony prominences
2.	Colour	Bright reddish brown	Yellow. Translucent. Parchment like
3.	Exudation	More. Scab slightly raised	Less. Scab lies below the level of skin surface
4.	Microscopic	Congestion. Vital reactions	No congestion or vital reaction

Fig. 12.6: Antemortem abrasion during healing stage. Pl note contracture of tissues around abrasion.

D. Medico Legal Importance of Abrasions

(1) **Direction of force** – can be estimated from tissue tags

(2) **Force of impact** – can be judged

(3) **Foreign matter sticking over abrasions** – may give an idea of scene of crime [eg grass, gravel, sand etc]

(4) **Imp and only sign** – of internal injury sometimes

(5) **Manner of production** –

(i) Generally accidental or homicidal in nature

(ii) May be self-inflicted for leveling false charges against enemies

(6) **Nature** –

(i) Usually simple, because they heal without scarring (ii) Do not pose danger to life, until and unless multiple and massive, or involve underlying vital organs

(7) **Time of assault** – can be calculated

(8) **Type of offence** – is indicated by site of abrasions

(i) around anus - homosexuality

(ii) around breast, cheeks, neck, inner side of thighs – indicate sexual assault

(iii) over face – indicate struggle

(iv) over mouth and nose – in smothering

(v) over neck – indicate throttling, especially crescentic abrasions

(9) **Weapon** – can be identified from patterned abrasions. Sometimes peculiarity of the offender's nails can be estimated [eg long, crooked, broken, irregular etc).

E. Differential Diagnosis

1. Burns

After death abraded epidermis becomes brown, leathery, parchment like, prominent and stiff and may begin to resemble burns.

2. Erosions produced by ants

(1) Ants gnaw the body after death and produce whitish or brownish erosions.

(2) **Differentiating features:**

(i) Margins - abrasions have irregular margins

(ii) Site –Since ants attack moist areas, the abrasions are found at

mucocutaneous junctions, around eyelids, nostrils, mouth, ears, axillae, knuckles, groins, genitalia and any moist folds of the skin.

(iii) Examination by hand lens – may reveal crescent shaped ant bite marks

(iv) Vital reaction - Absent.

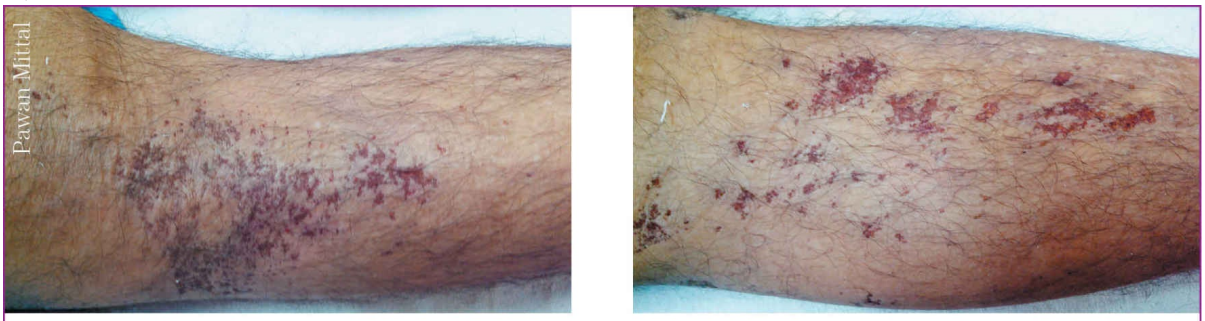


Fig. 12.7: Ant bites on legs. They are brownish indicating bites during perimortem period. Microscopy will reveal weak vital reaction typical of perimortem period.

3. Excoriations of skin by excreta

Seen in infants – persistence of excreta around napkin area"slight excoriation"these areas dry up and become depressed and parchment like. Color varies from pale-yellow to deep copper.

4. Drying of the skin

Scrotal area may dry up and produce reddish brown or yellowish discoloration resembling abrasions.

5. Pressure sores

Seen only in pressure areas. History of long immobilization may be there.

II. BRUISES (CONTUSIONS)

A ***bruise*** [Fig 12.8] is an effusion of blood into the tissues ***underneath the skin*** due to rupture of blood vessels (arterioles, venules and veins). When the effusion of blood is in other tissues and organs (muscles, lung, heart, brain, spleen, mesentery etc), it is called a **contusion**.

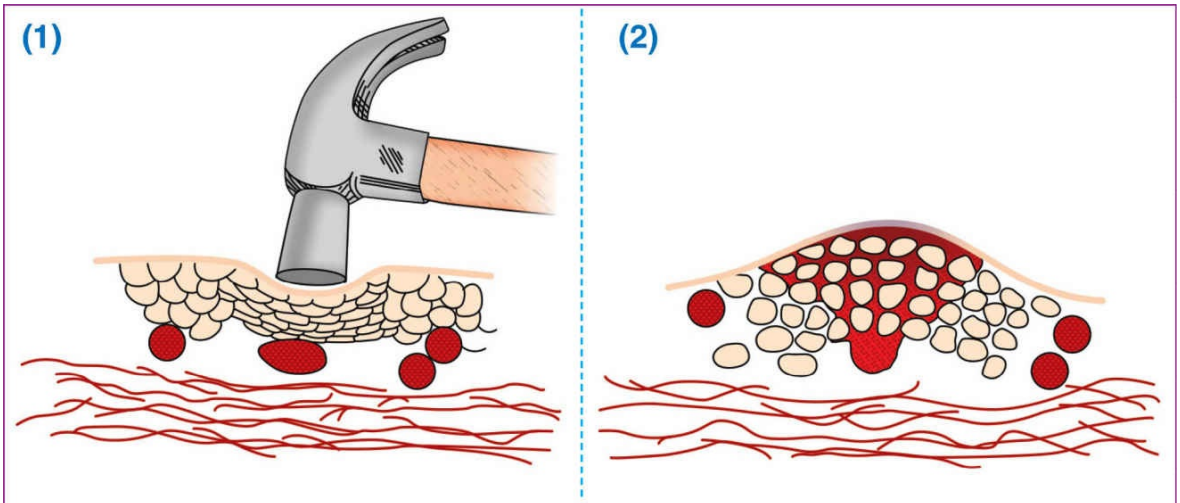


Fig. 12.8: Mechanism of production of a bruise. **(1)** A heavy blunt force like a hammer injures blood vessels. **(2)** Blood extravasates and fills up tissue spaces. This is seen through the skin as a reddish blue bruise. Beating somebody "black and blue" refers to production of several contusions.

Salient features:

(1) **Situation** – Bruise is usually situated in the **dermis** and **subcutaneous tissues** – some-times in the **fat layer**

(2) **Color** – Lighter in the center, than at the periphery, because extravasated blood is pushed outward

(3) **Cause** – Bruises (and contusions) are caused by **blunt force**, eg iron rod, lathi, fist, stone, cricket bat, hockey stick, whip, boot etc.

(4) **Associated findings** - (a) painful, tender (b) crushing and tearing of s/c tissues; (c) usually no destruction of skin (d) may occur in association with abrasions or lacerations. If abrasions and contusions occur together, it is customary to call them "**abraded contusion**" if contusion is more prominent and "**contused abrasion**" if abrasion is more prominent.

(5) **Size** - (a) **Petechial hemorrhages** – 0.1-2 mm (b) **Ecchymosis** – 2-5 mm (c) **Bruise** – more than 5 mm. Size of a bruise may be larger than the surface of the weapon which caused it, because blood continues to ooze (d) **Hematoma** – (i) Usually associated with substantial trauma, when a large BV is injured. (ii) More than 5 mm with appreciable swelling. (iii) Lesion rises above surface of skin. (iv) Forms a fluctuant mass (v) Unlike a standard bruise, a hematoma can be aspirated in the same way a collection of pus is aspirated.

(6) **Shape** –(a) Does not correspond with offending weapon, because of (i) constant oozing of blood (ii) tissue swelling. However if death occurs immediately after infliction, the oozing stops and shape may be similar to that of weapon (b) Often a bruise has a doughnut like configuration, with the center a lighter shade. This results from outward pressure of extravasated blood by the impact.

(7) **Margins** – blurred

(8) **Extent** – -force"bigger bruise

(9) **Compare** with corresponding opposite part – may display minor swelling.

(10) **D/d** – **Mongolian spot** (hyperpigmented skin in the lumbosacral region). Mongolian blue spots in a child. They may look like bruises and raise suspicion of child abuse.

(11) **Differentiating features**-

(i) Present since birth.

(ii) Location is always lumbosacral.

(iii) Color changes will not occur in Mongolian spot.

A. Classification

Bruises are classified in 3 types depending on their location [**Fig 12.9**]

(1) **Intradermal bruise** - (a) **Location** - Bleeding occurs in dermis (**Fig 12.9**). (b) **Amount** - of extravasated blood is small, but because of its superficial position, and relative translucency of epidermis above it, the pattern is distinct (c) This type of bruising occurs especially when the impacting object has **alternating ridges and grooves**, as the skin will be forced into the grooves and be sharply distorted [**ex**: vehicle tire running over body, kick by ribbed rubber soles of “trainer” shoes. **Stomping** (kicking and jumping on a person together) may also produce intradermal bruises].

(2) **Subcutaneous bruise** - Bleeding occurs in subcutaneous tissue. Most common type of bruising. Appears soon after injury

(3) **Deep bruise** - (a) Bleeding occurs in deep subcutaneous tissue just above muscle, or between muscular bundles. (b) Not apparent immediately after injury, but 1-2 days after (**delayed bruising**). (c) In suspected cases, infrared photography [**ch 30**] is essential (d) If this facility not available, examine again after 48 hours.

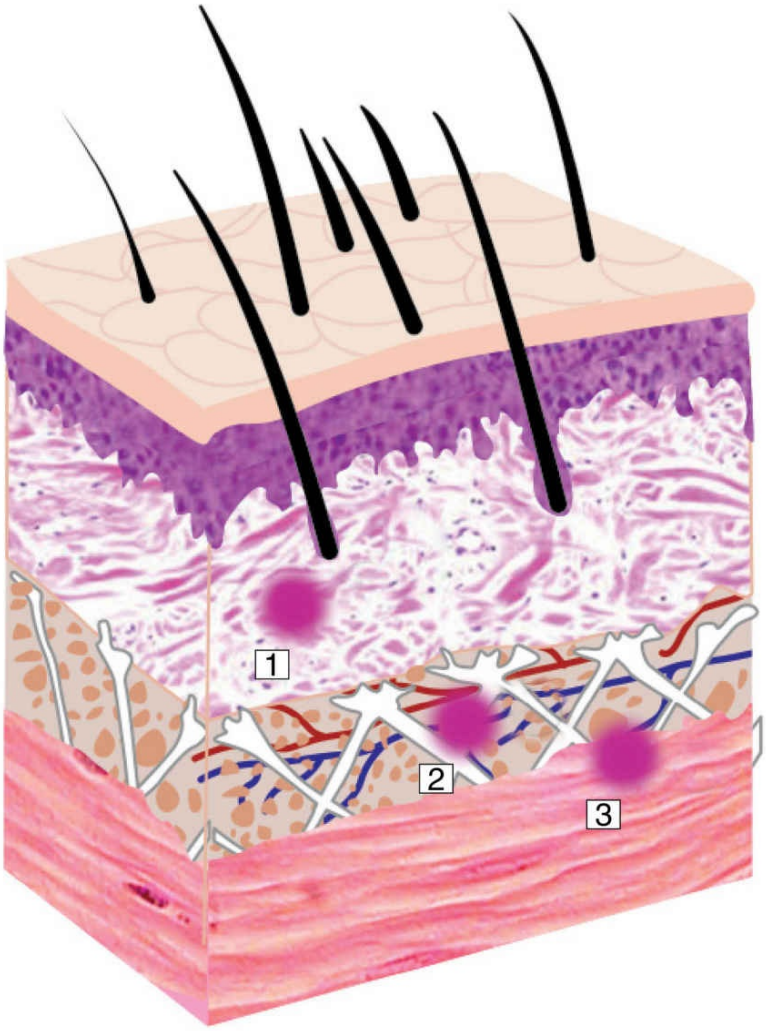


Fig. 12.9: **(1)** Intradermal **(2)** subcutaneous and **(3)** deep bruise

B. Factors Modifying Size and Shape

1. Age

(i) **Children** and **old** people bruise easily [soft and delicate subcutaneous tissues in children. Loss of flesh and atherosclerotic changes in old] (ii) **Adults** (especially muscular) bruise less easily.

2. Sex

(i) Women bruise more easily than men [more s/c fat. More delicate tissue]. Even a firm grip on the arms of a delicate woman may produce bruising (ii) Men

bruise less easily

3. Obesity

Fat people bruise more easily [greater amount of s/c fat]

4. Color of skin

(i) **Fair skinned people** – bruising is better appreciated **visually** (ii) **Dark skinned people** – Bruising is not easily appreciable. May be better appreciated on touching (raised area due to tissue swelling can be felt). Always compare with the identical area on the opposite side.

5. Effect of embalming

Bruises are more prominent in embalmed bodies because (i) embalming fluids form a **dark pigment complex** with blood (ii) Since **pressure** is used to force embalming fluid into BVs, it also forces blood through the damaged areas (iii) Transparency of blood is increased.

6. Condition and type of tissue

(i) **Vascular and loose tissue** (eg face, vulva, scrotum)" More bruising, because there is sufficient space for blood to collect (ii) **Firm, fibrous, strongly supported tissue** (scalp, back, palms, soles)"Less bruising (iii) **Good muscle tone** (boxers, athletes)"Less bruising because of good muscle tone, which prevents blood from extravasating (iv) **Resilient areas** (abdomen, buttocks) bruise less.

7. Weapon used

(i) If weapon is a yielding weapon (eg sandbag), bruising may not be seen. (ii) If it is a cane or whip, **railroad bruise** may be seen (please see under patterned bruising).

8. Associated diseases

(i) **Arteriosclerosis** – bruising occurs easily (even on coughing and slight exertion), because diseased vessels rupture easily (ii) **Blood dyscrasias** – may cause prominent bruising even on minor trauma because of defective clotting mechanism (eg hemophilia, leukemia, prothrombin deficiency, purpura hemorrhagica, scurvy, vitamin k deficiency). Purpuric spots can be **differentiated** from true bruises by following points (a) Purpuric areas are clearly demarcated from surrounding skin (b) They never show tissue swelling (iii) **Poisonings** – Hepatotoxic poisons may cause major bruise to appear from minor trauma because of prothrombin deficiency (eg alcohol, phosphorus). Chronic alcoholics bruise more easily.

9. Gravity shifting of blood

The site of bruise does not necessarily indicate the site of violence. Sometimes the two may be entirely different. When extravasated blood at the site of injury tracks along fascial planes or between muscular layers ***under the influence of gravity*** to reappear at a site below the injured site, it is known as ***ectopic***, ***migratory*** or ***percolated bruise***. **Ex**: (i) **Spectacle hematoma** - [**Fig 12.10**] (a) Blunt injury to the forehead"blood extra-vasates in forehead region"gravitates down and collects around the eye (b) Blunt injury to skull"fracture of anterior cranial fossa (ACF)"blood extravasates below ACF"gravitates down and collects around the eye (black eye). Because of its peculiar position, known as **spectacle hematoma**, **panda eyes** or **raccoon eyes**. Contusion of eye may also be as a result of direct trauma (punch in the eye). (ii) Bruise behind the ear [**mastoid ecchymosis, Fig 12.11**] may indicate fracture of posterior cranial fossa rather than a direct blow behind the ear. (iii) Injury to the scalp"bruising of the face (iv) fracture of jaw"Bruise appears in neck (v) fracture of pelvis"Bruise appears in thigh (vi) fracture of femur"Bruise appears on lower outer aspect of thigh (vii) Blunt injury to upper part of thigh"Bruise appears above knee (viii) Kick on the calf"Bruise appears around ankle.

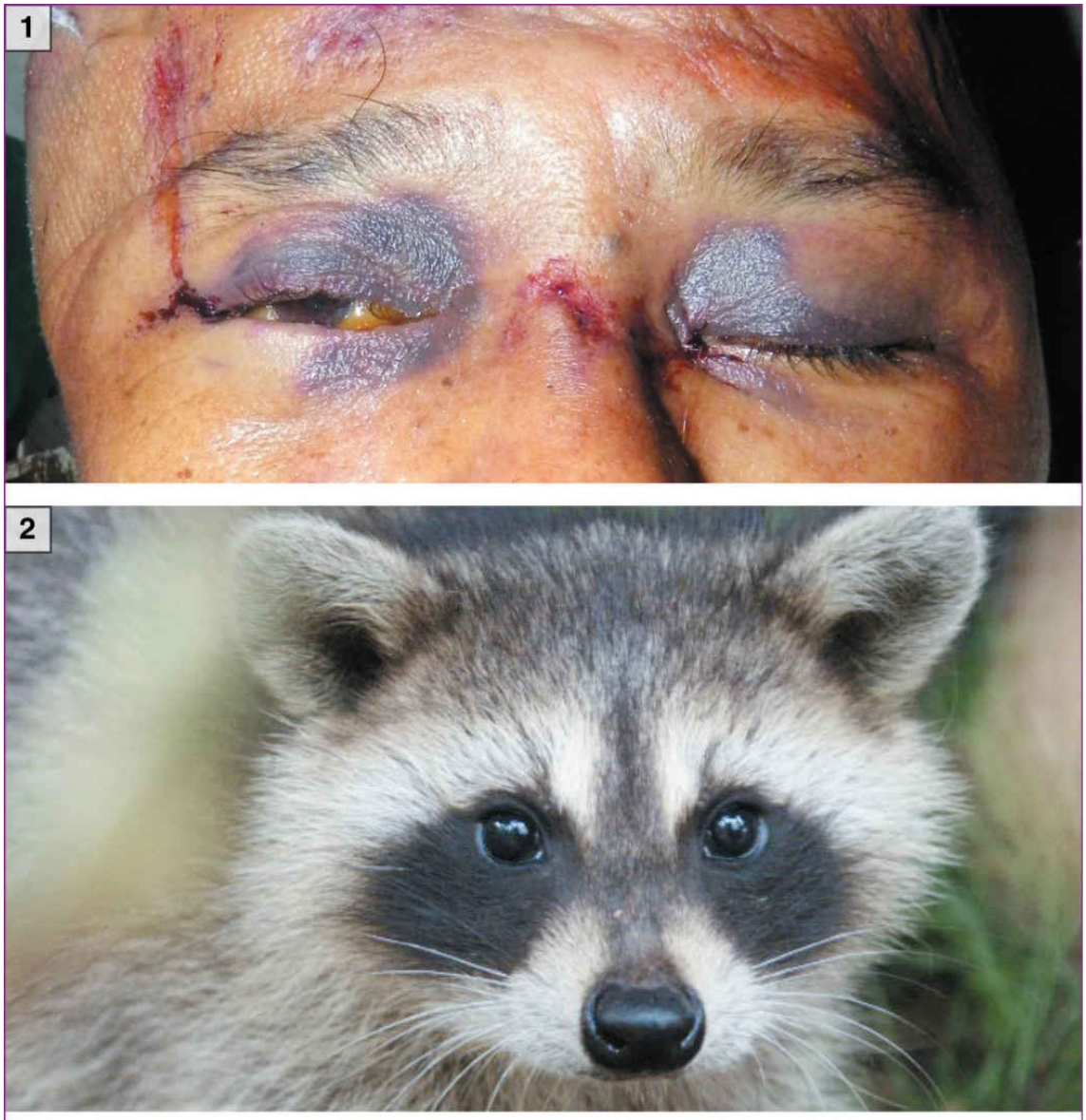


Fig. 12.10: **(1)** Bilateral black eyes. **(2)** They look similar to the eyes of a raccoon, hence the name "raccoon eyes".

Fig. 12.11: Battle's sign.

10. Miscellaneous factors

(i) If injured part is thickly clothed, bruising may not be seen (ii) Persons on **anticoagulants, antiplatelet** drugs, **SSRI**"More extensive bruising. SSRIs inhibit platelet activity.

C. Patterned Bruising

A ***patterned bruise*** is that which reflects the pattern of the striking surface. Such bruises are very medicolegally very important, because they can tell about the striking object. **Ex.**: **Railway track bruise** - Also known as **railway line** or **tram line bruise**. Caused when the skin surface is struck by a pliable instrument such as a broom handle, cane (as that used by police), narrow planks, wood and metal rods or whip. The bruise appears as two parallel lines of bruising with an undamaged zone in the centre (like a railway track).

Fig. 12.12: A railroad bruise on front of chest.

D. Age of Bruise

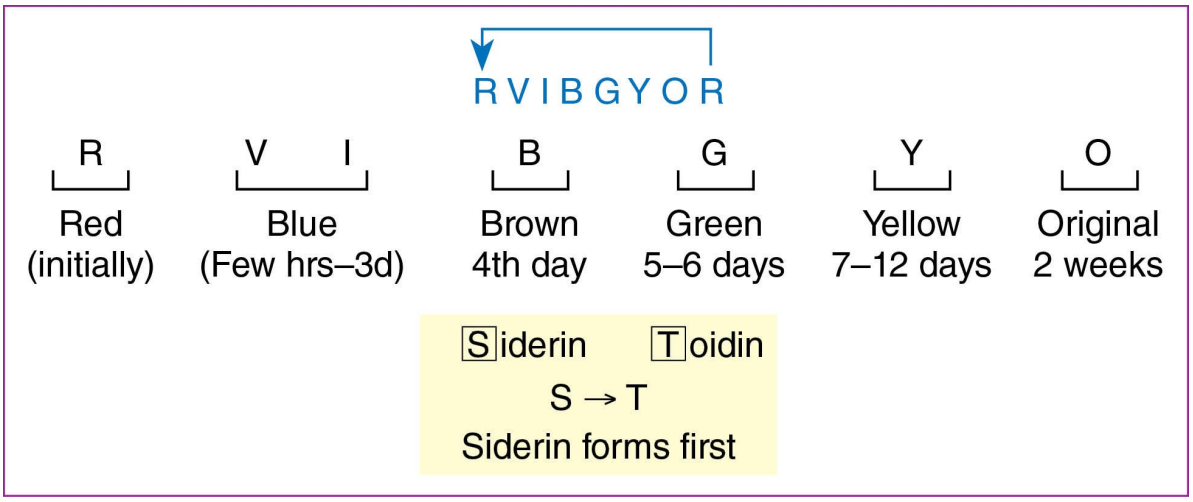
(1) **Mechanism** - Bruise heals by destruction and removal of the extravasated blood. RBCs disintegrate by hemolysis"Hb liberated"broken down into hemosiderin, hematoidin and bilirubin by the action of enzymes. Because of formation of different pigments, color of the bruise changes with its age.

(2) **Rate of healing** – Healing faster if
(i) area is more vascular
(ii) Contusion is smaller
(iii) Individual is young, well-nourished and healthy.
(3) **Color changes:**
(i) Start - at the periphery and extend inwards towards the center.
(ii) Color changes [**Table 2**]
(iii) Rate of color change [healing]– Highly variable, and one should be very cautious in interpreting age from colors [please see above for factors affecting rate of healing]. In extensive and deeply situated bruises, color takes a longer time to appear externally.

Table 2: Age of bruise

S.No	Time	Color	Pigment
1.	Initially	Red	OxyHb
2.	Few hours – 3 days	Blue	Hb
3.	4th day	Bluish-black to brown	Hemosiderin
4.	5-6 days	Greenish	Hematoidin
5.	7-12 days	Yellow	Bilirubin
6.	2 wks	Normal	OxyHb

Memory Aid 2: Age of bruise



(4) **Special cases** -

(i) Subconjunctival bruising – color changes do not occur. They change straight from red to yellow

(ii) CO poisoning – bruise have a cherry red color.

E. Deep Tissue and Organ Contusions

(1) All organs may be contused

(2) **Contusion of brain:**

(i) may cause impairment of function, confusion

(ii) ***Contusion of vital centers*** [eg those controlling BP and respiration]- Even small contusions may cause coma and death.

(iii) ***Pathogenesis*** – (a) causes swelling (b) Metabolic by products accumulate which are toxic to the brain

(3) **Contusions of the heart** –

(i) Prevent adequate cardiac emptying"Ht failure

(ii) If involve SA or AV node or Bundle of His"may cause serious disturbance of normal rhythm or stoppage of cardiac function"Death

(4) **Contusion of visceral organs** – may cause rupture" bleeding into body cavity"Death.

F. Postmortem Bruises

Postmortem bruise is one that appears after death.

Salient features:

(1) **Causes** - It may be due to (i) an antemortem injury or (ii) postmortem injury.

(i) **Antemortem injury** - Sometimes if death occurs soon after blunt impact, bruise may appear after death. This is due to (a) passive oozing of blood from damaged BV under the influence of gravity (b) hemolysis of stagnant blood"pigment diffusing out locally and producing a stain on the surface [**come-out bruise**] (c) Pressure of putrefactive gases may further push blood out of damaged BV. (d) Blood gravitates down to the most dependent areas, rendering non dependent areas much more paler. Paler areas in non dependent parts may reveal bruises [because of greater contrast], which were not visible earlier. Therefore even if no bruise was detected during life, it may be worthwhile to search for it at autopsy.

(2) **Differences** between AM and PM bruise are given in **Table 3**.

G. Artificial Bruises

Artificial or ***false bruises*** are fake bruises produced by rubbing some irritant substances on the skin.

Table 3: Differences between an antemortem and postmortem bruise

S.No.	Trait	Antemortem bruise	Postmortem bruise
1.	Time of formation	During life	Within 2-3 hours after death; not later
2.	Swelling in and around the area of bruise	Present	absent
3.	Damage to epithelium	Present	Absent or minimal
4.	Size	Small to large. Always proportional to the force used	Always small. Wholly disproportionate to the degree of force used
5.	Extravasation of blood	More	Less
6.	Site	Anywhere	On bony prominences
7.	Tissues underlying bruise	Blood infiltrates the tissue and stains it. Tissue stains resist washing	Tissue stains do not resist washing
8.	Histological examination	Inflammatory reaction Present	Absent
9.	Color changes	Older bruises will show different color	Will always be of same color, i.e. dull bluish
10.	Histochemical examination	Shows appearance of various reparative enzymes	No such enzymes can be detected

Salient features:

(1) They produce discolorations which look like bruises, but can be differentiated (**Table 4**).

(2) **Reasons** - They are produced (i) to bring a false charge of assault against an enemy (ii) by prisoners to bring a false charge of torture against policemen (iii) by malingers to go on leave

(3) **Substances** used for producing artificial bruises are (i) Calotropis gigantea (ii) Plumbago rosea (iii) Semecarpus anacardium [marking nut] of these at least Calotropis gigantea [madar] has a **wrong reputation of producing artificial bruise**. Author has rubbed the juice of madar several times on his own arms, with absolutely nothing happening. He has in his possession several videos of this too. (iv) Unusual Agents like Dithranol have also been used.

Memory Aid 3: Substances used for producing artificial bruise

Can **M D P**ass – Calotropis [wrongly attributed], Marking nut, Dithranol, Plumbago

Table 4: Differences between true and artificial bruise

S.No.	Trait	True bruise	Artificial bruise
1.	Cause	Blunt force	Irritant juices (calotropis, marking nut, plumbago rosea)
2.	Site	Anywhere	Exposed accessible parts. May be on inaccessible parts also if an accomplice is used
3.	Color	Changes with time	Dark brown
4.	Shape	Usually round or ovoid	Irregular
5.	Margins	Diffuse, irregular	Well defined and regular
6.	Itching	No	Yes (due to irritation)
7.	Vesicles	No vesicles	Vesicles present on site, as well as on fingertips due to scratching
8.	Redness and inflammation	Seen over the bruise	Seen in the surrounding site
9.	Contents (can be examined in dead only)	Extravasated blood	Acrid serum
10.	Chemical tests (washings over the bruise)	No chemical detected	Irritant substance detected

H. Demonstration of Bruises at Autopsy

(i) Examination of whole body under **UV light** [please see **ch 30** for all things visible in UV light] (ii) **Contusions of scalp** – Demonstrated by reflecting the scalp (iii) **Contusions of neck** – layer dissection of neck (iv) Contusions due to **torture** and **beating** – Long parallel incisions through the skin (v) **Microscopy** in cases of doubt.

I. Complications

(1) **Death from shock** - An average bruise contains about 20-30 ml of blood. Multiple bruises can cause death from shock.

(2) **Infection** - stagnant blood serves as a good culture medium for bacterial growth, especially of clostridial group

(3) **Fat embolism** - Rarely a large bruise may cause sudden compression of the subcutaneous tissue"release of s/c fat"Pulmonary fat embolism

(4) **Necrosis and Gangrene** - may sometimes result from extensive bruising.

J. ML Importance

(1) **Age of bruise** – Determined by color changes. Can confirm or refute the story of victim

(2) **Alcoholics** – are more prone to bruising because

(i) keep falling due to unstable gait

(ii) cirrhotic disease causes bleeding diathesis. With the passage of time they accumulate bruises all over their body. This may give an appearance as if they have been involved in a fight. All bruises will however be of different ages.

(3) **Decomposition** – can cause merger of contusions with blurring of their patterns.

(4) **Differentiation from PM staining** - They must be differentiated from pm staining (hypostasis) [**Table 5**].

(5) **Distribution of bruises** – Can indicate the character and manner of injury

(i) **Child abuse** – Bruises of different ages all over the body indicate that the child was beaten regularly. Indicates child abuse

(ii) **Manual strangulation** – Position of finger tip and thumb bruises over the neck (as in the above case) can indicate method of asphyxia, as well as position of assailant

(iii) **Pinning on the ground** – deduced by bruising of shoulder blades

(iv) **Restraint signs** - 3-4 coin sized bruises on one side of arm (produced by finger tips) and one larger bruise on the opposite side (produced by thumb) would indicate that arms were held together by the assailant. From the relative positions of smaller bruises with the larger one, the position of the assailant can be deduced. This can be sign of forcible restraint.

(v) **Sexual assault** – Can be deduced by bruising of inner aspect of thighs

(6) **Extraneous material** – Same ML value as in lacerations

(7) **Manner of production:**

(i) Self inflicted – Very rare because painful. May be seen in hysteria and mentally ill persons.

(ii) Accidental – (a) Very common (b) Seen on prominences [forehead, nose, elbows, knees] (c) Manner of causation – can be deduced from presence of dirt, Grease, Mud, Oil and Sand

(iii) Homicidal - (a) On any part of the body (b) Associated injuries eg lacerations, stabs etc can reveal their manner of production

(8) **Medicolegal value** - Bruises are medicolegally less valuable than abrasions because:

(i) Ectopic bruises - appear away from the site of injury. Thus exact site of injury is not denoted by location of bruise

(ii) Direction of force – not indicated

(iii) Shape and size - does not correspond to the offending weapon [due to constant oozing of blood]. Thus bruises caused by different weapons, fist, fall etc cannot be differentiated very well

(iv) Time of appearance – of deep bruises may be too long after infliction of injury

(v) Visibility - not very well visible in dark skinned persons

(9) **Micro-contusions** - are seen in firearm entry wounds surrounding each individual tattoo point [**ch 13**].

(10) **Patterned bruises** – May indicate the surface features of offending weapon. May connect the victim with the weapon. May help recover it [eg cane, chain, ligature, shoe, tire, whip]

(11) **Six penny bruises:**

(i) These are discoid shaped bruises of about 1-2 cm in diameter, usually in a group of three to five.

(ii) They resemble English six penny coin, and are caused from fingertip pressure.

(iii) Causes - (a) Child abuse [**ch 27**] (b) criminal abortion, rape and attempted rape - present over thighs, lower abdomen, perineum (c) throttling - over neck: [**ch 19**]

(12) **Size** – can indicate the degree of violence.

Table 5: Differences between PM Staining and bruise

S.No	Feature	PM staining	Bruise
1.	Cause	Pm change after death	Blunt force injury
2.	Collection of blood	within the vessels	outside the vessels
3.	Extent	Extensive	Generally localized
4.	Site	Most dependent parts	Anywhere on the body on which blunt force is applied
5.	Application of pressure	Deep pressure for a minute applied with thumb, would cause the pm staining to blanch, if it is not fixed	Will not blanch.
6.	Incision	no staining of s/c tissues. Blood within vessels	S/c tissues heavily stained with blood [can be done in dead bodies only]
7.	Swelling	None	Often swollen because of extravasated blood and edema
8.	Epidermis	Not abraded	May be abraded
9.	Margins	Merge with surrounding areas	Clearly defined. Accurate dimensions are always noted and written in MLC
10.	Microscopy	RBCs within blood	RBCs outside

III. LACERATIONS

Lacerations are tears or splits of skin, mucus membrane, muscle or internal organs, produced by application of blunt force to the body, which stretches tissues beyond their limits of elasticity.

Salient features:

(1) Also called **ruptures**

(2) Displacement of tissues is most common – when soft tissues are crushed against bone, e.g. scalp, facial bones, shoulders and shin.

A. General Characteristics

(1) **Margins** – irregular, ragged, uneven

(2) **Edges:**

(i) Angular impact - (a) skin on the side of wound opposite to the direction of motion is usually torn free and undermined. (b) Skin on the other side, i.e.

the side from which blow was delivered is abraded and bevelled

(ii) Perpendicular impact - Equal undermining on all sides.

(3) **Depth** – depends on

(i) degree of force

(ii) depth of soft parts at the site of injury

(4) **Shape and size:**

(i) May not correspond - with the weapon which produced it

(ii) Linear – produced by long, thin objects eg crowbars, pipes.

(iii) Irregular, ragged or Y-shaped – produced by objects with flat surfaces

(iv) if curved - convexity of curvature points towards the direction of application of force

(v) Stellate [star shaped] – produced by blunt round object

(vi) Crescentic– Produced by a blunt object with an edge, eg hammer head

(vii) Semicircular – striking head against wall or other hard surface. Due to convexity of head

(viii) Patterned lacerations

(ix) Swallow tails at one end – seen sometimes due to tearing at angles of lacerations [**Fig 12.13**].

(5) **Gaping** – present due to pull of elastic and muscular tissues.

(6) **Bruising:**

(i) in skin or s/c around the wound

(ii) If the force is exerted by the object moving in a downward direction, the lower margin of the wound shows more bruising, and undermining.

(7) **Tissue bridges** [**Fig 12.14**]:

(i) Tissue bridges or bridging fibres are seen at the base of wound. This is because deeper tissues [BV, connective tissue, elastic fibres, nerves etc] are unevenly divided.

(ii) Tissue bridges become apparent, when the edges of the wound are gently separated manually

(iii) MLI - their presence immediately indicates blunt force

(iv) Internal organs - Bridging is also seen in internal organs eg liver, kidneys, pancreas and spleen.

(8) **Hair bulbs** – crushed

(9) **Hair and epidermal tags** – Deeply driven in the wound

(10) **Hemorrhage:**

(i) less in laceration. Because vessels are crushed and torn across irregularly. (a) They can retract (b) Blood clots readily

(ii) Exceptions – scalp lacerations, where hemorrhage is more. Temporal artery is firmly bound"unable to retract.

(11) **Foreign matter** – usually present in the wound.

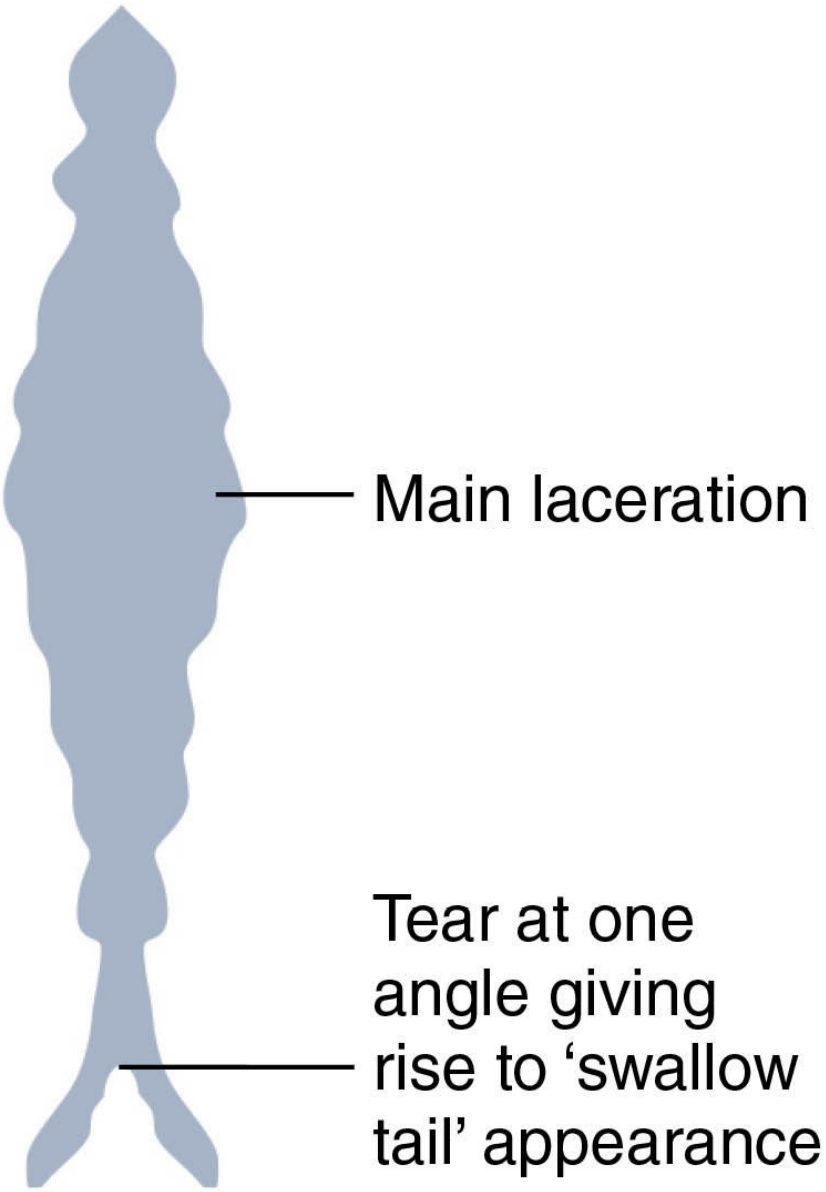


Fig. 12.13: Swallow tail appearance at one of the angles of a laceration.

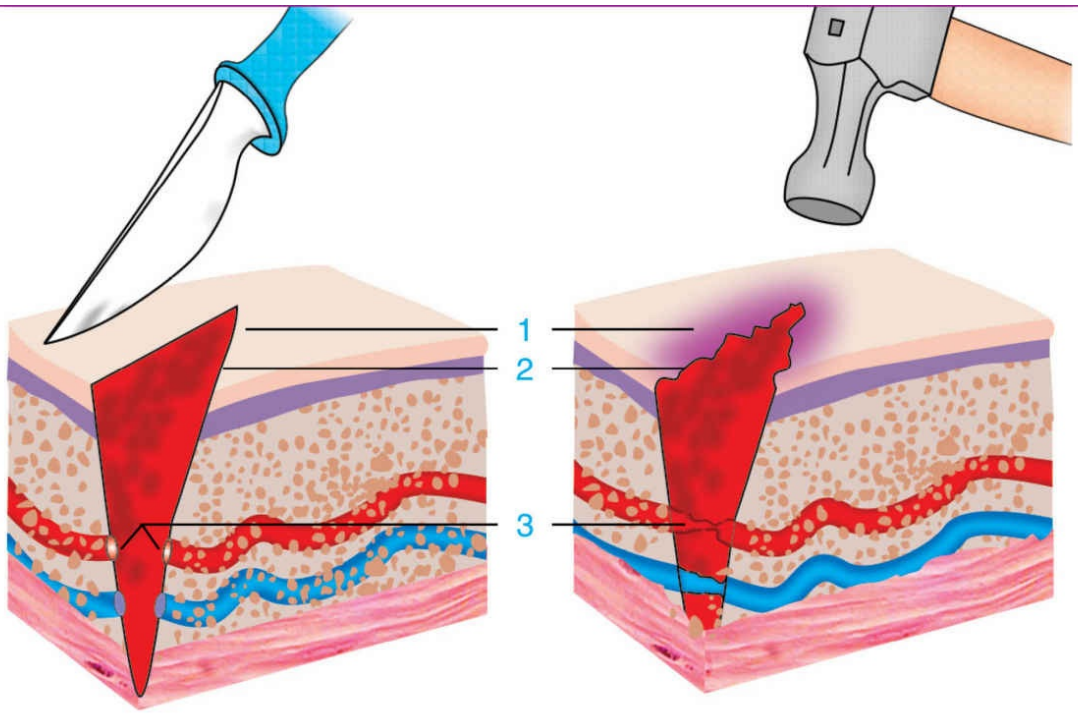


Fig. 12.14: Differences between sharp and blunt force injury **(1)** No contusions around sharp force injury; contusion around blunt force injury **(2)** Former has sharp margins, latter, irregular margins **(3)** in former, vessels clean cut and no tissue bridges. In latter, vessels are crushed and there are tissue bridges

B. Types

(1) ***Tear***, the most general type of laceration, and encountered most commonly, occurs when blunt force is applied to skin having ***sufficient amount*** of underlying fat and muscle (eg thigh).

(i) caused by a heavy blunt weapon (cricket bat, hockey stick, lathi)

(ii) has ragged and bruised margins.

(2) ***Split laceration*** occurs when blunt force is applied to skin direcly overlying a bone, with ***miminal amount*** of underlying fat and muscle. (scalp, zygomatic process, chin, tip of shoulder, back of elbow, anterior superior iliac spine, shin).

(i) **Mechanism** - Skin is split between the weapon and the underlying bone.

(ii) **Appearance** – mimics an incised wound [called incised looking lacerated wound. Examination with hand lens is helpful (**Table 6**).

(iii) **Table 7** lists differences between a true incised and true lacerated wound.

Table 6: Differences between an incised and incised looking lacerated wound

S. No	Criteria	Incised wound	Incised looking lacerated wound
1.	Margins, when seen under a hand lens	clean cut	Ragged and bruised
2.	Edges	No, or minimal bruising	Heavily bruised
3.	Blood vessels at the base of the wound	Blood vessels at the base are clean cut	Blood vessels at the base are crushed.
4.	Other tissues at the base of the wound	Tissues such as fascial bands and nerves are clean cut	Fascial bands and nerves are either crushed or seen intact. They show continuity
5.	Hair around the wound	Clean cut	Crushed

Table 7: Differences between a true incised and true lacerated wound

S. No	Criteria	True incised wound	True lacerated wound
1	Edges	Clean cut	Ragged
2	Margins	Not bruised	Bruised
3	Injuries to bv, nerves, tendons, fascia at the base of wound	Clean cut	Crushed
4	Hair bulbs	Clean cut	Crushed
5	Bleeding	More	Less

(3) ***Stretch laceration*** is caused if the force is applied in such a manner that skin is overstretched, resulting in a large flap. **Ex**. a glancing kick by foot.

(4) ***Cut laceration*** is produced by heavy cutting weapons [axe, boat propeller, bush knife, chopper, hatchet, industrial and farm machinery, lawn mower blade, machete (a large heavy knife used for cutting sugarcane), meat cleaver] wielded with a tremendous amount of force.

(i) It may best be conceived as a "cross" between a true laceration and a true incised wound. The edges of the weapon do cut the skin, but since the edges are heavy, they crush and bruise the margins of the wounds too.

(ii) The wounds show an ugly, wide gape

(iii) The underlying bones are frequently fractured, or show deep grooves or cuts.

(iv) Also called **chop wounds.**

Fig. 12.15: A cut laceration showing a deep cut on the underlying mandible.

(5) ***Avulsion*** is produced if grinding compression [eg fast moving tire of a heavy motor vehicle (eg run over inj; ***ch 18***), or a rotating heavy industrial machine], is applied, causing avulsion (separation) of large areas of skin from its underlying attachments and is lost (**Fig 12.16**).

(i) Wound is devoid of any overlying skin.

(ii) also known as **flaying**.

(iii) Underlying muscles are heavily bruised. **Table 8** summarizes various types of laceration and how they are typically caused.

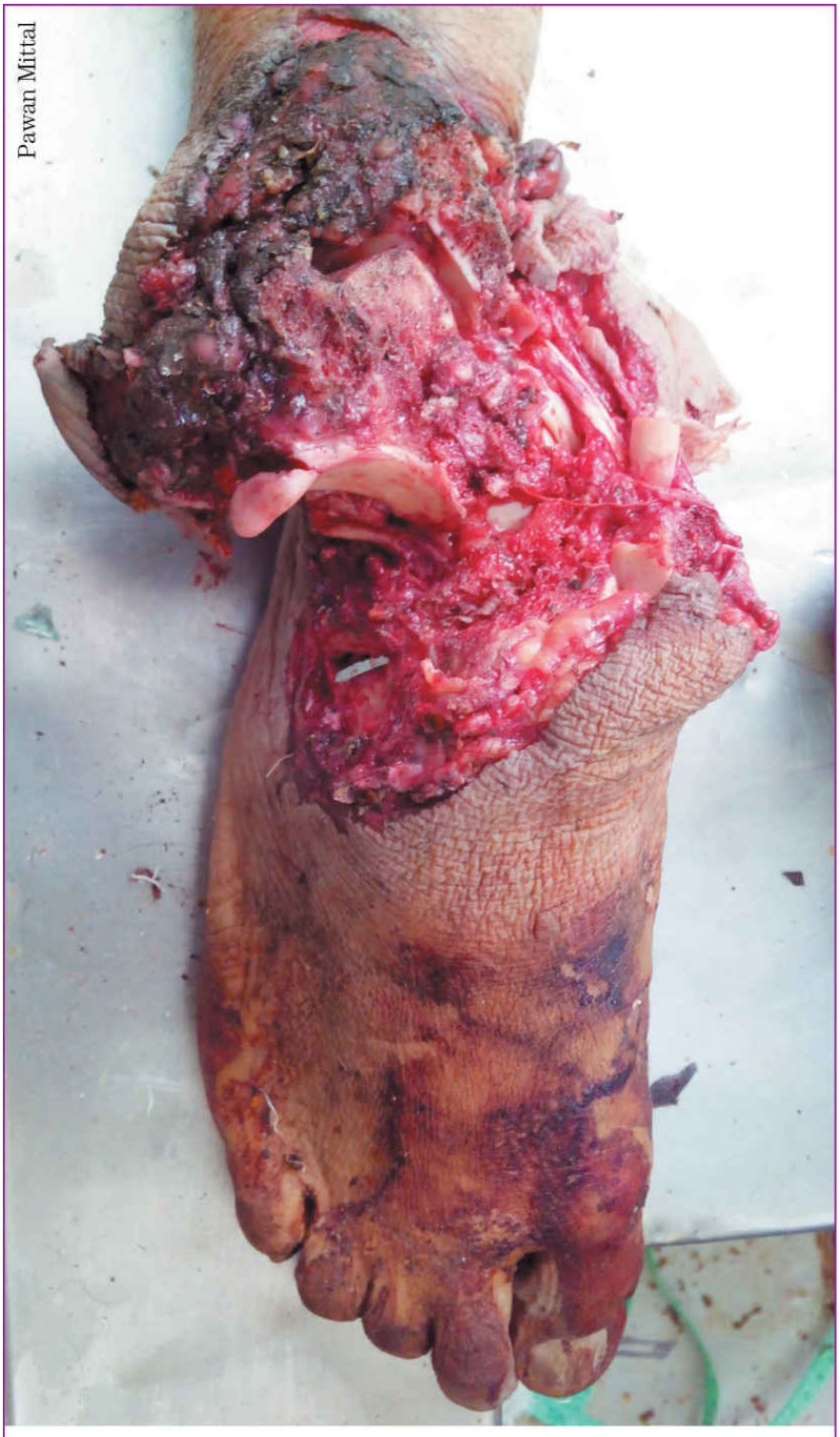


Fig. 12.16: An avulsed laceration of foot.

Table 8: Different types of lacerations and how they are typically caused

S.No	Type of laceration	How typically caused
1.	Tear	Any heavy blunt instrument (e.g. a hockey stick or a cricket bat)
2.	Split laceration	Any heavy blunt instrument, on an area of body which has some bone directly underneath with a scanty layer of intervening tissue (e.g. scalp)
3.	Stretch laceration	A heavy force acting on the body in such a manner that a portion of skin is overstretched
4.	Cut laceration	Heavy cutting weapon with an edge which may not be too sharp
5.	Avulsion	Heavy weights (e.g. a truck) passing over the body, causing large areas of skin to be avulsed and lost. Also called "flaying"

C. Age of Lacerations

Age determination is difficult unless clear signs of healing are present [eg Fibroblasts, Granulation tissue, Organizing infiltrate].

D. AM and PM Lacerations

Antemortem lacerations would show (1) Bleeding (2) Blood-staining of margins (3) Bruising (4) Eversion (5) Gaping (6) Vital reactions. PM lacerations would show none of these.

E. MLI

(1) **Age of lacerations** –

(i) Fight - indicates time of struggle

(ii) Disputed pregnancy - age of perineal lacerations indicates the possible date of delivery [please see **ch 24** for more details]

(2) **Combinations of lacerations with abrasions and bruises** – Abrasions, bruises and lacerations may be seen together. Each injury may be produced by different blows by the same weapon, or they may be produced at the same time (as in explosions). Punching (with a fist), kicking and stomping (with a shoe wearing foot) can produce all three injuries with different blows.

(3) **Extraneous material** – may be present in lacerations (eg dirt, dust, grass, grease, sand, stone particles). This may connect the injuries with the place of occurrence

(4) **Firearms** [**Lacerations produced by**]– By pistol whipping [buffaloing].

(i) It is the act of holding a handgun by its barrel, and clubbing the victim with the butt, in effect using it as a blunt weapon.

(ii) location - Predominantly on forehead.

(5) **Manner of production** -

(i) Accidental - falls etc

(ii) Suicidal lacerations – are very rare, because a person virtually never uses a blunt object for suicide (very painful and rarely effective). May be seen in suicidal jumping from high buildings.

(iii) Homicidal - (a) Lacerations on the vertex generally indicate homicide. (b) ***Bagh Nakh*** [tiger claws] is a claw-like weapon inspired by the armature of big cats. It fits over the knuckles or is concealed under and against the palm. It consists of four or five curved blades affixed to a crossbar or glove, and is designed to lacerate skin and muscle. Some incorporate a spike or blade on one end of the crossbar. This form is known as a **bichawa bagh nakh** because the blade is based on that of the bichawa (scorpion knife).

(6) **Shape of lacerations** –

(i) can very rarely indicate the nature of offending weapon. (**patterned lacerations**).

(ii) Causes – (a) chains (b) Pistol whipping [please see below]. Please also see above under the heading “shape and size”.

(7) **Tendons and muscle lacerations without corresponding lacerations on**

skin – Can indicate violent muscular contractions and spasm (convulsant poisons like strychnine, convulsive natural disorders like epilepsy, tetanus). In such cases, lacerations in hollow viscera may also be seen.

IV. INCISED WOUNDS

An ***incised wound*** (cut, slash, slice) is a clean cut wound through the tissues, caused by a sharp-edged instrument. In an incised wound, length is its maximum dimension [cf. stab wound].

Salient features:

(1) **Force** – delivered over a very narrow area, corresponding with the cutting edge of the instrument

(2) **Skips in pattern** - A series of incised wounds, separated from one another by bridges of normal skin are produced - (i) when there are bony ridges (ii) tendons or (iii) if a dull weapon was used over areas of loose, thin, atrophic, folded and wrinkled skin, eg back of hand in an elderly victim..

A. Causes

(1) **Typical instruments which cause incised wounds** – Blade, knife, razor, scalpel, sword

(2) **Single or multiple strikes** – Usually caused by a single strike. A "sawing motion" may produce a more complex wound.

(3) A **curved sharp weapon** (eg a sickle) produces a stab from the pointed end, and an incised wound from the blade with an intervening intact skin.

(4) **Glass** – Wounds by glass (eg broken bottles, broken glass pieces) are typically **incised**. If the bottle is very thick [eg a cola bottle], the broken pieces would be equally thick, and may produce bruising around margins, giving the appearance of a **cut laceration**.

B. Characteristics

1. Length

(1) **Greatest dimension** - Length is the greatest dimension in incised wounds [cf stab wounds].

(2) Length has no relation to the cutting edge of the weapon, because a cutting

weapon may be drawn to any distance creating an incised wound of any length.

2. Width

Width may be greater than the edge of weapon - due to

(i) retraction of divided tissues

(ii) shaking of blade.

3. Margins

(1) Clean-cut, well-defined

(2) if knife is serrated – may produce a saw-toothed cut

(3) Edges -

(i) usually everted

(ii) Inverted – if a thin layer of muscle fibre is adherent to the skin, pulling it inwards [eg in scrotum]

(4) Abrasions and contusions -

(i) Not seen on margins if weapon was perfectly sharp

(ii) may be seen if the cutting edge was blunted; because then the incision is produced more by pressure than by the sharpness of the cutting edge.

4. Shape

(1) **usually spindle shaped** – due to greater retraction of edges in the center

(2) Crescentic – if blade is curved or struck surface is convex

(3) **Zigzag** – if skin folds are loose, as in axillary folds, scrotum etc

(4) **Gaping** – is greater if underlying muscle fibres are cut transversely or obliquely [due to retraction]. Lesser when cut longitudinally.

5. Direction

(1) Incised wound is deeper at the beginning [**Fig 12.17 (A)**], because more pressure is applied on the knife at this point. This is known as the **head of the wound**.

(2) As the assailant draws the knife away from head, it is gradually withdrawn. This causes the wound to become increasingly shallow towards the tail [**Fig 12.17 (A)** and **(B)**]. Finally as the knife leaves the tissue, the depth is so less that only the skin is cut alone. The portion where the epithelium alone is cut is known as **tailing of the wound**. It indicates the direction of the incised wound. Because only epithelium is injured, the tail of the incised wound typically shows no bleeding.

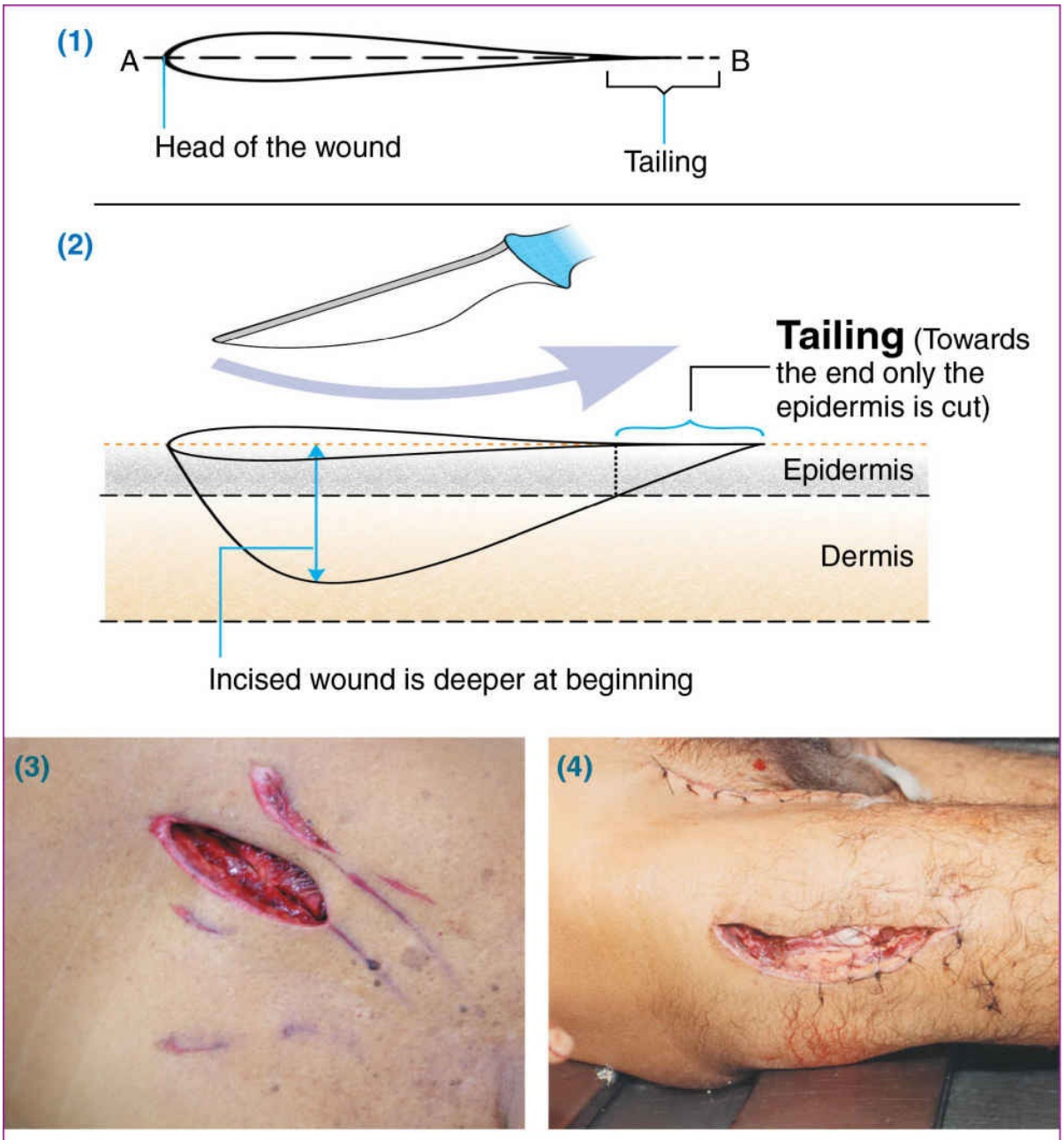


Fig. 12.17: Incised wound is deeper at the beginning and less deep towards the end **(1)**. **(2)** is a vertical section of the incised wound along the broken line "AB" showing its depth at various points. **(3)** two incised wounds showing tailing. Sometimes a stitched wound may have to be opened to reveal tailing **(4)**. White dotted circle highlights tailing.

6. Beveling cut

[A] A ***beveled cut*** refers to a cut which is not perpendicular to the skin surface, but at an angle [Fig 12.18]. [B] **Direction of weapon:** (1) **Vertical** – If weapon

enters vertically the edges of wound would make a nearly 90° angle with the skin surface [**Fig 12.18(1)**] (2) **Oblique** – If weapon enters obliquely [**Fig 12.18(2)**], one edge would make an angle >90° with the skin surface (**beveled edge**). The other edge (under mined edge) makes an angle <90° with the skin surface. It mostly lies submerged under the surface. (3) **Nearly horizontal** – If weapon enters nearly horizontally [**Fig 12.18(3)**], a wound with a flap is produced. [C] **Manner** – Beveling usually indicates homicide, and may indicate relative position of the assailant and victim.

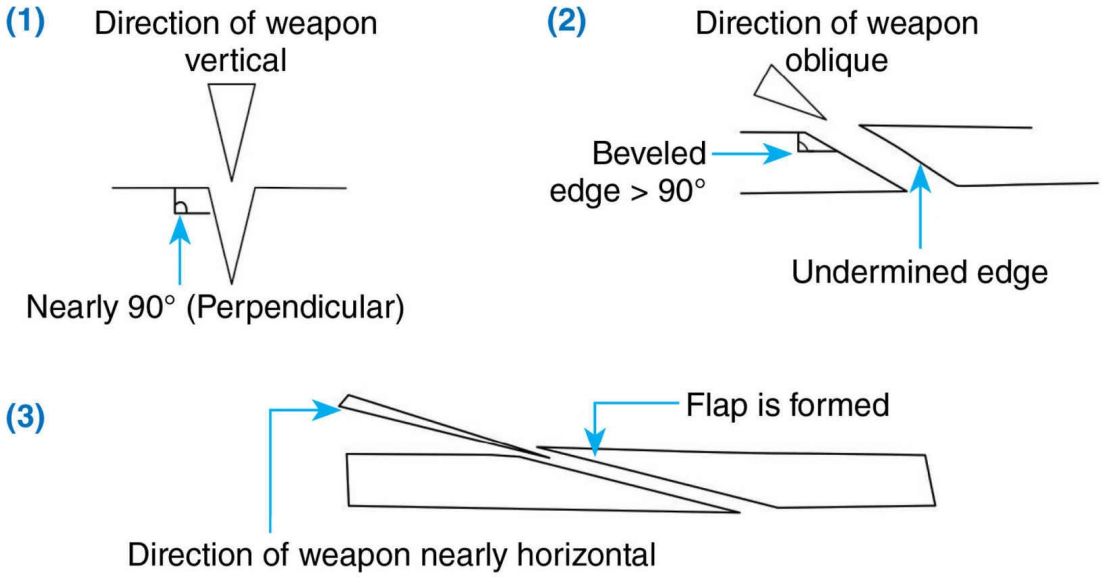


Fig. 12.18: Edges of the wound would indicate the direction of weapon. Please see text for details

7. Hemorrhage

(1) Hemorrhage is **more** as the vessels are cut cleanly.

(2) Spurting will occur if an artery has been cut.

C. Cut Throat Wounds

1. Suicidal and homicidal

Please see **Table 9**.

Table 9: Differences between suicidal and homicidal cut throat

S.No	Feature	Suicidal cut throat	Homicidal cut throat
1.	Site	Left side of neck (in right handed persons and vice-versa); passes across the front to throat. Rarely on both sides	Usually on both sides
2.	Level	High. Above the thyroid cartilage	Low. On or below the thyroid cartilage
3.	Direction	Above downwards and from left to right in right handed persons.	Transverse or from below upwards. If attacked from front, the wound runs from left to right
4.	Edges	Ragged (due to overlapping of multiple incisions)	Sharp and clean cut. Beveling may be seen
5.	Number of wounds	Multiple (10-15 or more) superficial parallel wounds (**hesitation cuts**) with **1 final deep wound** (fatal).	Usually single deep wound. May be multiple in "overkill". But all are deep
6.	Hesitation cuts	Present	Absent
7.	Tailing	Present	Absent
8.	Severity	Several less severe wounds are present. There is one final fatal deep wound, but it never reaches vertebrae	Severe. May reach up to vertebrae, or even cut through them
9.	Neck vessels	As the suicide recedes his head back, carotid artery is drawn beneath sternocleidomastoid and escapes injury. Jugular vein is injured	Both carotid arteries and jugular veins injured
10.	Blood stains	(i) Person usually stands in front of mirror to see position of hand. Splashing on mirror is seen (ii) Blood stains on front of body and clothes. May also be seen on palms (iii) direction of blood from above downwards (iv) splashes on feet may be seen	(i) no splashing on mirrors (ii) if sleeping, the blood flows down both sides of neck and collects on the back. May be seen on palms, if victim tried to stop bleeding (iii) direction of blood from before backwards on neck, as blood flows backwards (iv) splashes on feet may only be seen if victim was standing
11.	Weapon	Present at the scene	Always absent
12.	Clothes	Not cut or damaged	May be torn, if struggle ensued. Clothes may be disarranged, torn and may show loss of buttons
13.	Hands	May be in cadaveric spasm holding weapon of offence	May be in cadaveric spasm holding hairs of the assailant, some piece of his clothing, buttons etc
14.	Wounds in other areas of the body	Several superficial wounds may be present in other parts of the body, as the suicide experiments with pain. May be present on back of neck, wrists, thighs, groin, ankles and knees	Very deep if present. No superficial wounds are seen
15.	Defense wounds	Not present	May be present on palms and wrists
16.	Scene of crime	(i) Usually room locked from inside (ii) Body may be lying in front of mirror (iii) no disturbance of surroundings (iv) Suicide note (farewell letter) may be found	(i) Room never locked from inside (ii) Body not in front of mirror (iii) Surroundings may be very much disturbed (iv) Suicide note never found
17.	Circumstantial evidence	Victim may be found depressed. May have talked to relatives about the futility of life. May have started distributing his costly belongings among friends and relatives	Victim may be found to be in financial or other dispute with the killer

D. Age of Incised Wounds

1. Gross

(1) **Fresh** – Hematoma formation
(2) **12 h** –
(i) Edges red, swollen, adherent with blood and lymph
(ii) Leukocytic infiltration
(3) **24 h** – Dried clot in the form of crust or scab.

2. Histopathology

(1) **Few min** –
(i) Reactive changes in tissue histiocytes
(ii) Dilatation of capillaries
(iii) Swelling of vascular endothelium
(iv) Margination and emigration of neutrophils
(2) **12 h** –
(i) Reactive changes in fibroblasts
(ii) Monocytes appear in exudate
(3) **16 h** – Mitotic divisions begin in monocytes
(4) **24 h** –
(i) Epithelium begins to grow at the edges
(ii) A continuous layer of endothelial cells cover the surface
(iii) Vascular buds begin to form
(5) **72 h** – Formation of vascularized granulation tissue
(6) **4-5 d** – New fibrils are formed
(7) **1 wk** – scar formation if wound is small.

E. MLI of Incised Wounds

(1) **Reconstruction of crime** – Incised wounds help to reconstruct the crime and to corroborate or refute the story of the victim by enabling to determine (a) the nature of the weapon – sharp edged (b) Age of injury and (c) Direction of force
(2) **Decomposed body** - Differentiation between incised and lacerated body is difficult
(3) **Body with incised wounds immersed in water soon after death** - Blood in wounds is lysed by water. It is difficult to make out if the wounds were AM or PM. Same is true of lacerations and stab wounds also.

(4) **Manner of injury** –may be accidental, suicidal or homicidal.

Fig. 12.19: Hesitation cut on right forearm. It also indicates that the person was left handed. Pl note that the wound is deeper at the radial side and gradually thins out at the ulnar side, where it finally shows tailing. This is expected if the deceased used his left hand.

V. CHOP WOUNDS

Chop wounds (or **slash wounds**) are same as cut lacerations. Please see under the heading "Type of lacerations".

VI. STAB WOUNDS

A ***stab wound*** is one which has ***depth*** as its maximum dimension [cf. incised wound]. It is produced when force is delivered along the long axis of a narrow or pointed object, such as an arrow, dagger, knife, nail, needle, screw driver, spear etc. into the depths of the body.

Salient features:

(1) **How caused** - either when the instrument is driven in body, or the body is

pressed against or falls against the instrument.

(2) - (a) **Punctured wound** (**Fig 12.20**) is a stab wound caused by a circular "ice pick" shaped object. Wound caused by other instruments (eg daggers etc) would be called stab wounds. *A punctured wound typically terminates in flesh.* It may be due to (i) Insertion of pins, needles or threpeutic injections in flesh (ii) Snake bite (iii) Drug addiction [there will be older scars around from earlier injections]. In case of deaths due to therapeutic injections, drug addiction or snake bite, excised tisse around the punctured wound must be sent frozen for chemical analysis. **Homicidal needle punctures** may often be missed, especially when inflicted at base of skull near nape of the neck (injurying the medulla), axilla (injurying a major b.v.), or through natural orifices (fornix of upper eyelids, inner canthus of the eye, ears, nostrils, vagina, rectum). The locations of such punctured wounds are intentionally chosen to hide their location. These are known as **concealed puncture wounds** [concealed trauma – **ch 8**; concealed firearm wounds – **ch 13**]. Needle punctures are a common method of infanticide (through anterior or posterior fontanelles). (b) **Penetrating wound** (**Fig 12.20**) enters into a body cavity (chest, abdomen, skull, knee joint, orbit, scrotum, etc) without coming out. Should be described in a sequential order, i.e. entrance wound, depth and direction of track, specific location of termination **Ex** - (i) Stab wound to the chest or abdomen [having no exit] (ii) Gunshot wound to the head [having no exit] (iii) Needle injury to the eye (iv) Tracheostomy wound (v) Intracardiac injection [as in resuscitation attempts] (vi) Drainage tubes in chest and abdomen (c) **Perforating wound** (**Fig 12.20**) has both an entry and exit wound [same terminology is used in relation to **fracture of skull** [**ch 17**] and **firearm wounds**; penetrating wounds occur when a bullet enters the body and does not exit – has only an entry wound; perforating wounds occur when the bullet passes completely - has both an entry and exit wounds]. Should be described in a sequential order as above, i.e. entrance wound, path of wound, exit wound.

(3) **Entry and exit** Ex - (i) Stab wound to the chest or abdomen [with exit] (ii) Gunshot wound to the head [with exit] (**Fig 12.21**)- In perforating wounds, wound of **entry** is (a) larger (b) has inverted edges. **Exit** is (a) smaller [due to tapering of blade] (b) has everted edges. In firearm wounds entry is smaller; exit is larger [**ch 13**].

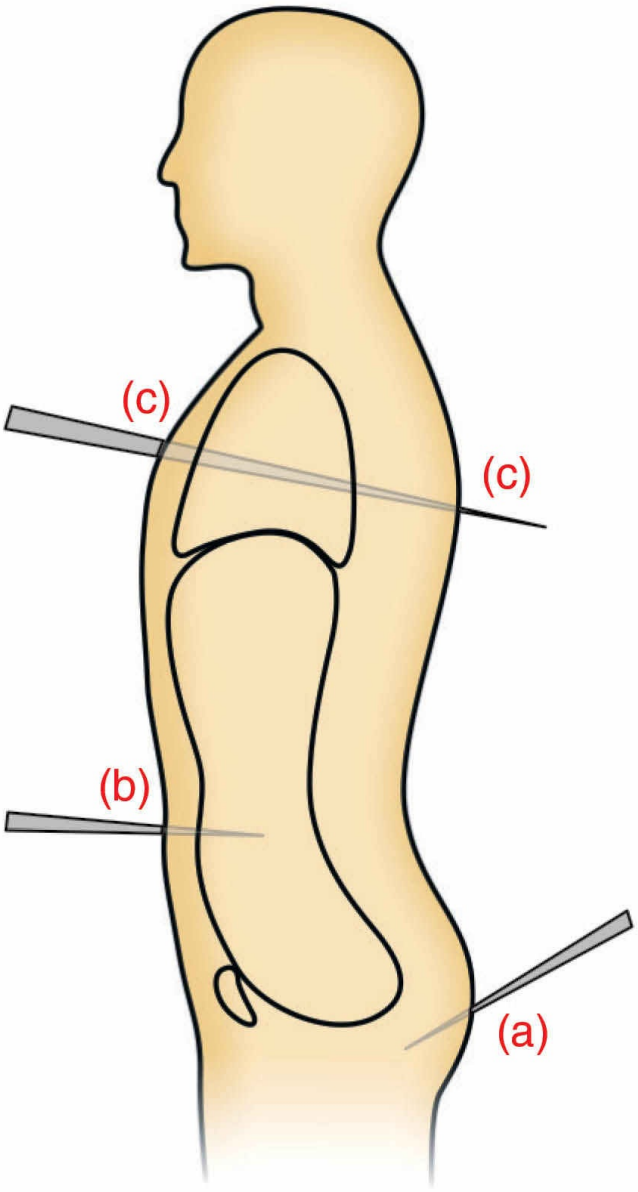


Fig. 12.20: (a) Punctured, (b) penetrating and (c) perforating wounds. Please correlate nos (given in red). with text.

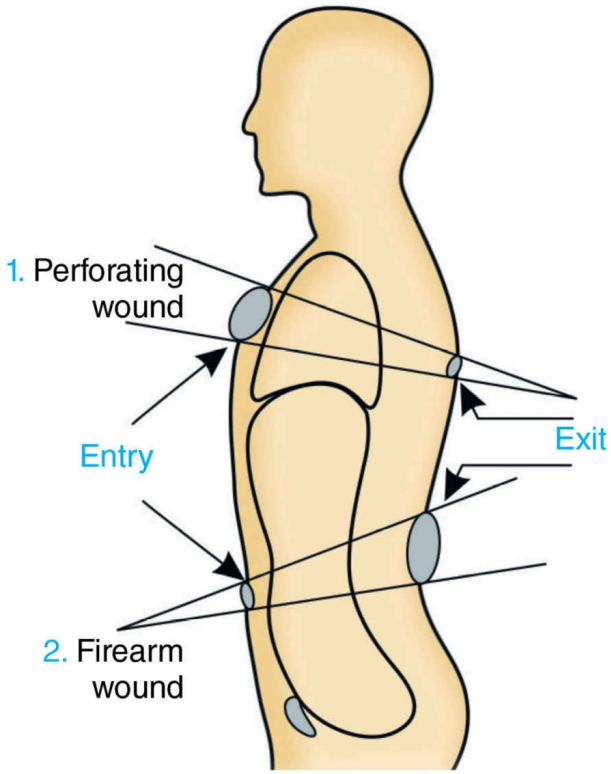


Fig. 12.21: Entry wound is larger than exit in perforating wounds. In firearm wounds, exit is larger due to more wobble at exit

A. Characteristics

1. Length

Length of stab wound 'l' is *normally less* than width 'w' of weapon. To predict the correct width 'w' of the weapon, the edges of the wound must be brought together [by fingers, or by an adhesive tape. This releases tension in the elastic fibres, so the wound displays its original length 'l', which should then be measured [**Fig 12.22**].

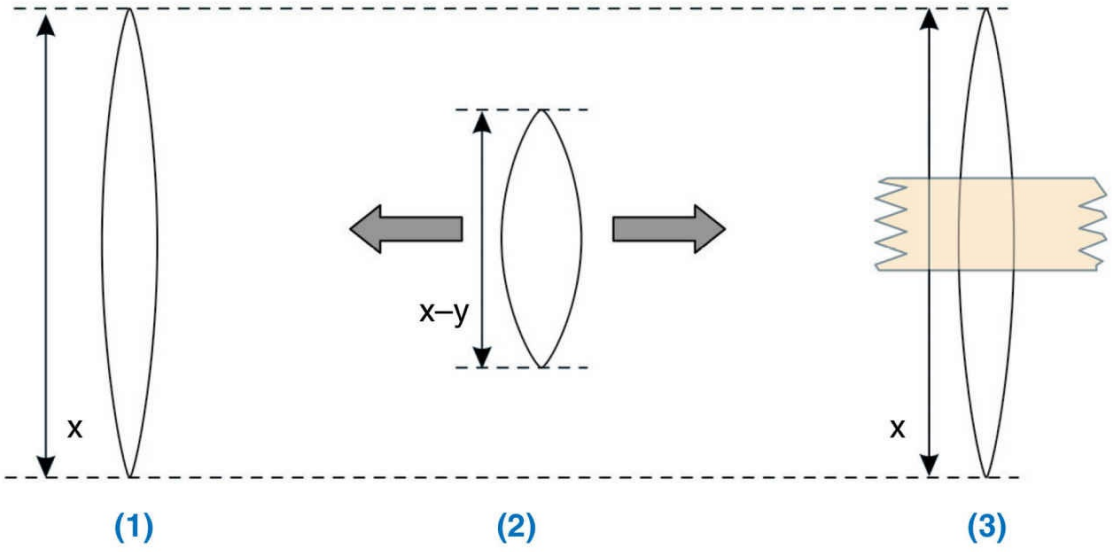


Fig. 12.22: Dimensions of a weapon may be wrongly reported, if gaping is not accounted for **(1)** Original wound when weapon is within the wound accurately reflects the width of weapon (x) **(2)**. As weapon is withdrawn, the skin gapes, and the length may get reduced to "x-y". An inexperienced pathologist may report this as the width of weapon **(3)** Restoring the wound with an adhesive tape restores the wound and allows pathologist to correctly assess the width of weapon.

2. Width

Width of stab may not indicate the true thickness of blade, because of retraction of wound edges, after knife is withdrawn.

3. Margins

(1) **If weapon has sharp edges** [eg knife, dagger]– Margins are clean cut [incised stab wound]

(2) **If weapon has rounded edges** [eg cricket stump, ice-pick, pencil, screw driver etc]– Margins are contused and lacerated [lacerated stab wound].

4. Depth

(1) **Greatest dimension** - Depth is the greatest dimension in stab wounds.

(2) **Assessing depth** – In the living, a stab wound must never be probed in order to assess depth, as it may disturb a loose clot and may lead to fatal hemorrhage, and produce false tracks. It should always be done in the OT, when the wound is being repaired. In the dead - Radiopaque dye must be injected in the track and X-ray taken Alternatively incision is made at the side

of stab wound, and tissues dissected parallelly. Reveals depth, without disturbing the actual wound A pliable rubber tubing may be introduced gently, then taken out and measured. Once inside, the tube may be made more rigid by inserting a probe.

5. Shape

(1) **Cleavage lines of Langer:**

(i) These are the lines within the skin along which the dense feltwork of intimately intermingled dermal collagen and elastic fibres is arranged [**Fig 12.23**]. Named after Austrian anatomist **Karl Langer** (1819-1887), professor of anatomy at Budapest and Vienna, who first described them in **1861**.

(ii) They are arranged in parallel rows, which are same in all persons

(iii) In the extremities – they run longitudinally; In the neck and trunk - circumferentially

(iv) Stab wound which runs across the wound causes gaping; a parallel cut does not and an oblique cut produces an oval or sometimes a crescentic or semicircular wound.

(2) **Shape of stab wound –** Normally it is slit shaped with two acute angles. But it would depend upon -

(i) Cleavage lines of Langer. Same weapon may produce different sized wounds on different parts of body, depending upon the course of these lines

(ii) Depth of penetration

(iii) Direction of thrust

(iv) Movement of blade within wound

(v) Movement of victim

(vi) Shape of weapon

(vii) Tenseness or wrinkling of skin.

(3) **Shapes of weapon influencing shapes of stab wounds:**

(i) Single edged weapon -produces tear drop [or rain drop], triangular or wedge shaped [**Fig 12.24**]. The angle caused by sharp edge is sharp and the one caused by blunt edge is rounded, blunt or squared off. Sometimes the blunt edge causes a split, such that the wound appears like the tail of a fish [**fishtailing**, **fishtail wound**].

(ii) Double edged weapon – produces elliptical, slit like or spindle shaped wound. Both ends are sharp.

(iii) Single edged weapon producing spindle shaped wounds – (a) If some length of knife near the tip was double edged, and the skin was too elastic. The angle corresponding to blunt edge would reflect the shape of initial few cm. As weapon is thrust deeper, excessive elasticity would “let in” the weapon without corresponding change in shape (b) If weapon penetrated at an oblique angle. As the knife perforates at an angle, the blunt edge does not impart its shape as it does not contact it.

(iv) Single or double edged weapons producing a wound with both ends blunt – if weapon was thrust up to the level of ricasso [short, unsharpened section of blade between the cutting edge and guard]. In both the above cases a correct shape of weapon can be given by examining the serosal planes [pleural, pericardial, peritoneal surfaces, liver capsule] and muscle fasciae, which clearly show the wedge shape. Thus in multiple wounds examination of all wounds is necessary before opining upon the nature of weapon. Possibility of multiple weapons [both single and double edged] must also be kept in mind.

(v) A round object with a pointed end like an ice pick or a spear – produces a circular wound. May resemble a small caliber bullet wound. **Leon Trotsky** [1879 – 1940], the famous Russian revolutionary was assassinated by a mountaineering ice axe much like an ice pick.

(vi) A round object with a blunt end [eg pointed stick, ball point pen, metal rod] – (a) produces a circular wound with bruised, inverted and ragged ends. (b) Blunter the tip, the coarser or more stellate would be the whole it makes (c) as such weapons are likely to be rusted and dirty, foreign material eg dirt, splinters, rust etc would be found within the wound

(vii) Pointed square weapon eg a screw driver – produces a cross-shaped injury, each of 4 edges producing its own “cut”.

(viii) Fall on a pointed article – would produce circular wound

(ix) Stabbing with a fork – produces a cluster of punctured wounds depending upon the number of prongs. A two edged fork would produce two separate wounds, their distance depending upon the angle at which it is struck. An open scissors would produce a similar wound.

(x) Thick relatively blunt-edged weapons eg bayonets – produce penetrating wounds with excessive laceration around

(xi) Stabbing followed by rocking, twisting or victim moving around – L or V

shaped wounds, with one limb corresponding to main or primary wound and the other an extension of it as it exits

(xii) Knife with a serrated edge – produced a ragged wound.

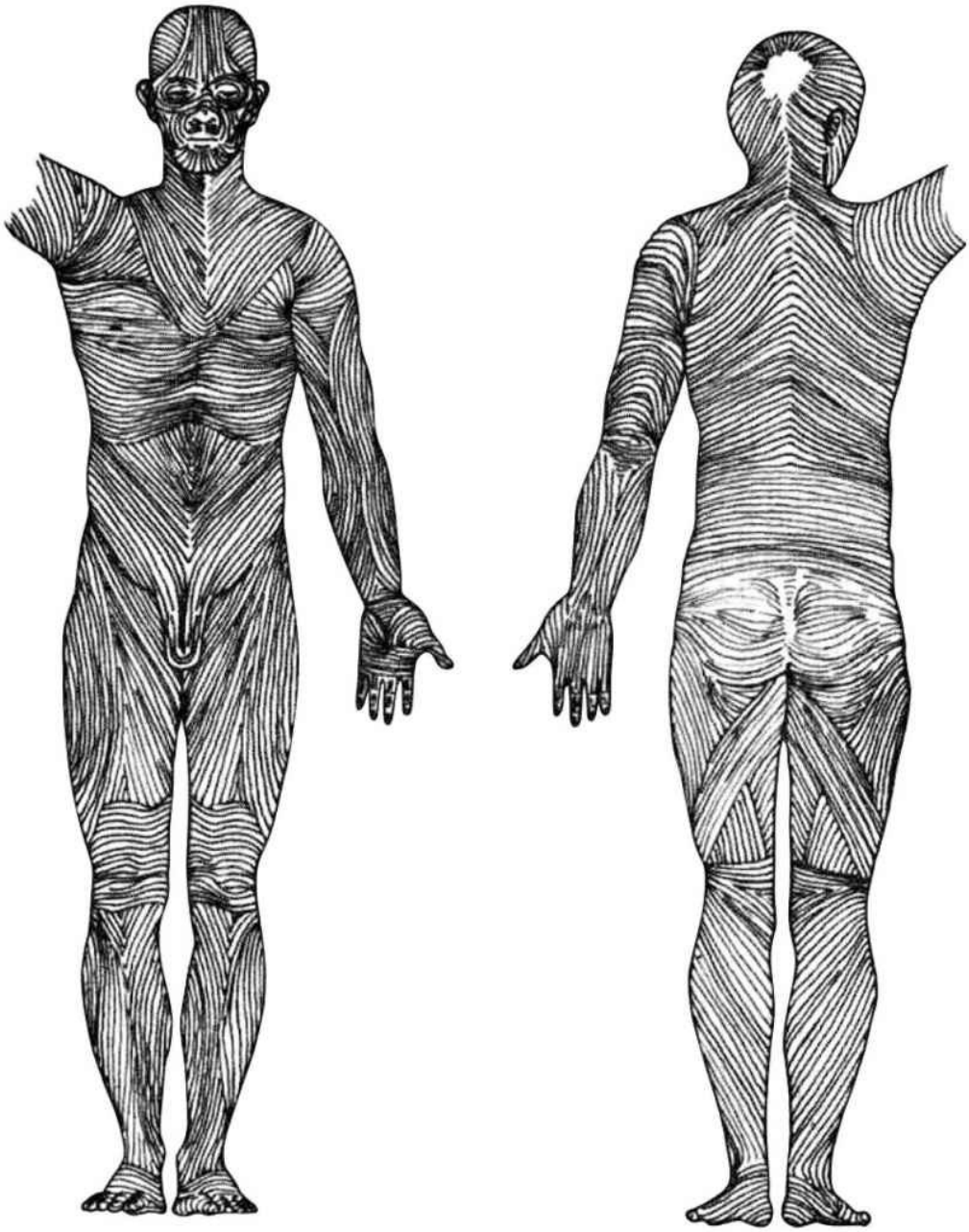


Fig. 12.23: Cleavage lines of Langer

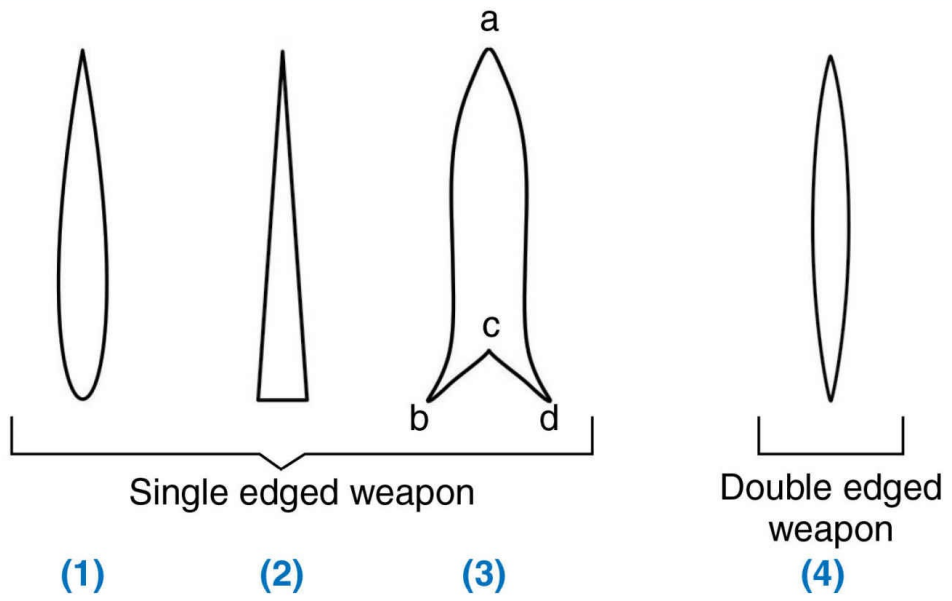


Fig. 12.24: Stab wounds produced by single edged weapon [**(1)** Tear drop or rain drop; **(2)** Triangular or wedge shaped and **(3)** Fishtail wounds] and double edged weapon [**(4)** Spindle shaped].

6. Direction

(1) **Penetration at an angle** – bevelled margin on one side and undermining [undercut] on the other [**Fig 12.18**],

(2) **Weapon withdrawn partially and thrust again** – externally only one entry wound would be visible. But two or more punctures seen in soft parts.

7. Opinion

Proper examination of stab wound would enable the pathologist to opine upon

(1) Amount of force used

(2) Dimensions of the weapon

(3) Direction in which weapon hit the victim

(4) Movement of knife within the wound

(5) Taper of blade

(6) Type of weapon.

Fig. 12.25: Homicidal wounds of the neck. Please note multiplicity of wounds without any parallel pattern as is seen in suicidal wounds. Pathologist must test for air embolism in all such cases.

B. Complications

(1) Air embolism – especially in stabs of neck vessels
(2) Choking due to inhalation of blood
(3) Hemorrhage
(4) Infection
(5) Pneumothorax.

C. MLI of Stab Wounds

(1) **Concealed puncture wounds** – used homicidally. Please see above.
(2) **Depth of wound** – indicates force used
(3) **Direction and dimension of wounds** – Indicate relative positions of assailant and victim
(4) **Manner of production** – Suicide, accident or homicide may be determined [**Table 10**]. (i) **Hara-kiri** [syn **Seppuku**, "cutting the belly"] – (a) Unusual form of suicidal stab wound on the abdomen. (b) At one time used as a capital punishment. (c) Procedure - The victim inflicts a single large wound on the abdomen with a tanto or wakizashi [ceremonial Japanese swords], while in a

sitting position; or falls forwards upon it, and pulls out intestines (d) Cause of death - (I) Excessive bleeding (II) Sudden . in intra-abdominal pressure ".ed cardiac return" Sudden cardiac collapse.

(5) **Multiplicity of wounds** – Indicates overkill [more wounds inflicted than were necessary to kill]. This happens in sexual and revenge murders

(6) **Shape of wound** – Indicates class and type of weapon [eg single edge, double edged]

(7) **Time of attack** – Can be determined by assessing the age of stab wound

(8) Must be differentiated from incised and lacerated wounds [**Table 11**].

Table 10: Differences between suicidal, homicidal and accidental stab wounds

S.No	Feature	Suicidal stab wounds	Homicidal stab wounds	Accidental stab wounds
1.	Cause	Self-inflicted	Inflicted by others	1. Falling against sharp objects [glass, nails] 2. Gored by horns of an animal [bull, buffalo]
2.	Position of wound	Accessible parts	Generally vital parts, but can be anywhere	On front or back
3.	Grouping	Well arranged in parallel lines [hesitation cuts]	No definite grouping	May be a single large wound
4.	Number of wounds	Generally one large wound accompanied by several superficial hesitation cuts	Multiple	Generally one
5.	Direction	In right handed persons, from left to right	Any direction	Generally straight inside
6.	Severity	Hesitation cuts are superficial; final fatal cut is deep. More than one deep stabs are generally against suicide	Severe, extensive	Severe
7.	Defense wounds	Absent	Present if attacked during fully conscious stage	May be there, if tried to fend off attack by animals

Table 11: Differences between incised, lacerated and stab wounds

S.No	Feature	Incised wound	Lacerated wound	Stab wound
1.	Manner of production	By sharp objects or weapons	By blunt objects or weapons	By either
2.	Site	Anywhere	Usually over bony prominences	Usually over chest and abdomen, but can be anywhere
3.	Margins	Clean cut, Even, everted, Smooth	Irregular, undermined	Clean cut, parallel edges. If weapon is blunt pointed, margins may be lacerated
4.	Abrasion on edges	Absent	Usually present	Absent
5.	Bruising of adjacent tissues	Absent	Present	Present if weapon was blunt pointed
6.	Shape	Linear or spindle shaped	Irregular	Linear or irregular
7.	Dimensions	Length is the greatest dimensions [longer than deep]	Usually longer than deep	Depth is the greatest dimension [deeper than long]
8.	Anatomical structures at the base of wound	Cleanly cut	Bridging strands seen	Cleanly cut
9.	Hair bulbs	Cleanly cut	Crushed or torn	Clean cut if weapon was sharp
10.	Hemorrhage	Usually profuse and external. Spurting of blood present if artery cut	Slight, except in scalp. Occurs externally	Profuse. May occur both internal and external
11.	Underlying bones	May show cuts	May show fractures	May show cuts
12.	Foreign bodies	Absent	Usually present	Usually absent

VII. DEFENSE WOUNDS

Defense wounds are caused by immediate and instinctive reaction of the victim to save himself, either by raising the arm to prevent the attack, or by grasping the weapon [**Fig 12.26**].

Salient features:

(1) **Incidence** – Seen in about 40% of all homicides.

(2) **Nature of Defense wounds** – (a) When attacking weapon is sharp - (i) Incised wounds (ii) perforations of forearms or hands [rarely] (b) When attacking weapon is blunt - (i) abrasions and bruises (ii) rarely fractures of carpals, metacarpals and phalanges (c) Defense wounds "due to attack" – When the victim "attacks" but the basic purpose is to defend himself. This may cause wounds (i) on the victim [eg on knuckles of hand or fingers, when the attacker was struck in the mouth] (ii) on the attacker [teeth bite marks on the hands and forearms of the attacker]. This is a rare case where Defense wounds are on the attacker and not on the victim.

(3) **Location** – (a) Palm, when the victim tries to seize the weapon [***"active"***

defense wounds] (i) If single edged weapon is used - incised wound is either on the palm or on the bends of fingers or thumb [**Fig 12.27**] (ii) If double edged weapon is used – incised wounds are found both on the palm and fingers (b) Back and ulnar aspects of forearm, when the victim raises the hands or arms for protection [***"passive" defense wounds***] (c) interdigital spaces Knuckles rarely thighs and feet, if the victim is thrown on ground, and she curls up and kicks the assailant.

(4) **Defense wounds are absent** - (a) when it is a case of suicide or accident (b) in cases of homicide when (i) victim is unconscious (ii) taken by surprise (iii) attacked from back (iv) weak, debilitated, old, very small infant (v) drunk or under influence of drugs

(5) **Injuries simulating Defense injuries** - Offensive injuries may sometimes simulate Defense injuries Victim punches assailant on his anterior teeth with his fist"may sustain knuckle lacerations which may look like Defense injuries Victim punches assailant on his head or chin"may sustain fractures of metacarpals

(6) **Medico legal importance** - Homicide - Defense wounds indicate homicide Sexual assault - If they are present in females, especially on thighs.

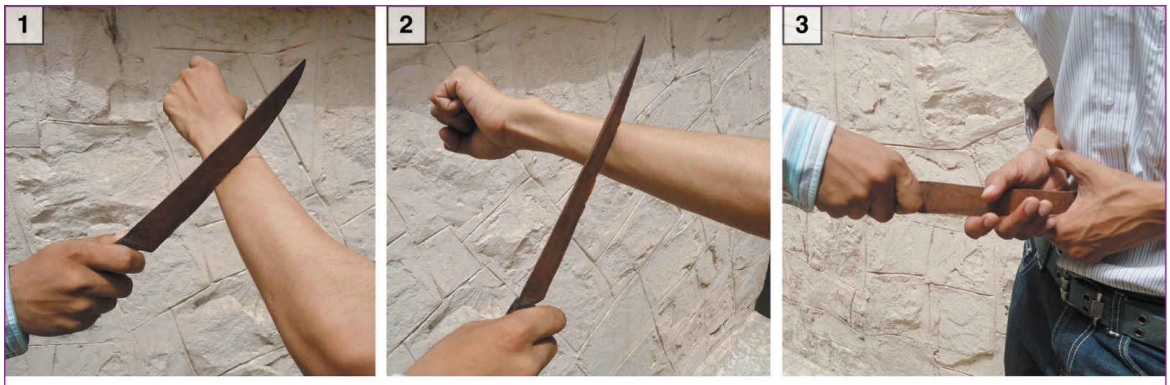


Fig. 12.26: When a person tries to defend himself, the defense wounds appear on **(1)** back and **(2)** ulnar aspect of the forearm and **(3)** on palms. First two are "passive defense wounds"; 3rd is active defense wound.

Fig. 12.27: Active defense wounds in a decomposed body. Medicolegal value of these wounds increases in decomposition [or in disposed off severed arms] where they may be the only remaining sign of homicide. This figure shows multiple active defense wounds in a decomposed body. The weapon used is a double edge weapon, as cuts are there both in fingers and at the base of thumb.

VIII. OFFENSE WOUNDS

Offense wounds [syn, protective wounds] are reverse of defense wounds and are caused by offense rather than defense.

Salient features:

(1) **Injuries** - (i) Abrasions, (ii) contusions, (iii) lacerations over knuckles, (iv) boxer's fractures [**ch 17**]

(2) **Lacerations** - may be caused if a fist was planted on the victim's open mouth with exposed teeth. The injury should be matched with dislocated or fractured teeth. Saliva of victim would be detected in such lacerations. Teeth of victim would reveal blood of assailant [Locard's exchange principle].

IX. SELF-INFLICTED WOUNDS

Self inflicted [syn, fabricated, fictitious, forged or invented wounds] are those

inflicted by a person on his own body or by another with his consent.

Salient features:

(1) They may be created ab initio or an existing injury may be deepened or enhanced

(2) **Reasons** - (i) By an assailant - To change appearance of wound so he may not be connected with the crime or so that he may pretend self defense (ii) By a victim - (a) To blackmail an enemy (b) To charge an enemy with assault or attempted murder (c) To make a simple injury appear more serious (iii) By military men - To escape service or to get leave (iv) By policemen, servants, watchmen acting in collusion with thieves and robbers - To show they were defending property (v) By prisoners - To bring a false charge of attack against police officers (vi) By women - To bring a false charge of rape against an enemy. (vii) by mentally ill – eg paranoid schizophrenics may inflict hundreds of small wounds upon themselves (viii) Religious mendicants – may cut away their penis, scrotum and testes in order to achieve celibacy.

(3) **Nature** - (i) Incised – mostly (ii) burns, Contusions, Stabs – next most common (iii) lacerated, firearm wounds – rarely. Author has dealt with a case of self inflicted firearm wound in the right thigh. The allegation was that his enemy had shot him. However it was a near discharge phenomenon. The track of wound was virtually vertical, entering just below the inguinal ligament and exiting from just above the knee.

(4) **Characteristics** - (i) General – (a) Multiple, (b) parallel, (c) superficial (d) generally on accessible areas, but rarely may be found on back. Author has dealt with several cases, where the perpetrator fixes a razor blade at the end of a cleft-stick and makes cuts on back with the help of a mirror. (e) An injury covered by clothing and unaccompanied by any damage to it is self inflicted, as the perpetrator lifts cloth to inflict injury. It cuts are seen on cloth, they are inconsistent with the injury (f) Old linear scars in nearby areas or other parts of body suggest similar occurrences earlier. However they are often seen in mental patients too. (g) Rarely a person may inject local anesthetic before inflicted such wounds. Needle puncture mark must be looked for around the wounds. (h) history is inconsistent with injuries, eg an injury may be old, and history given may be of recent assault and vice versa; history may be of attack with lathis, but examination may reveal incised wounds etc. Additional characteristics of specific wounds are (ii) Incised wounds are (a) They are of

equal depth at origin and termination. (b) avoid sensitive and vital areas eg face [eyes, ears, nose, lips] (c) direction – on top of head, behind forwards [tailing in front]; from above downwards on outer side of upper arms; horizontal or from below upwards on forearms and abdomen; variable on chest, and from below upwards on abdomen and legs. (iii) Stab wounds (a) Site – fleshy areas eg upper arms; virtually never on chest. (iv) Burns – superficial; mostly on left upper arm. Usually produced by lighted cigarette tips, but unusual objects may be used.

X. THERAPEUTIC WOUNDS

Therapeutic or ***iatrogenic wounds*** are wounds produced by doctors during treatment (eg. surgical wounds of chest and abdomen for drainage tubes, tracheostomy, thoracotomy, laparotomy, cut-open wounds for IV catheters etc).

Salient features:

(1) Sometimes these wounds may be mistaken for traumatic wounds (**Fig 12.28**). It is thus best to study original MLC and operation notes before starting pm.

(2) Sometimes an original traumatic wound may be enlarged and used for therapeutic purposes (i) drainage tube may be inserted in a homicidal stab wound (ii) gunshot wound of the abdomen may be used for colostomy.

Fig. 12.28: Iatrogenic injuries. These circular abrasions look like a bite mark, and may be confusing. They are due to electrocardiographic leads placed on the chest for long periods. The person was in the ICU for long.

XI. MEDICOLEGAL IMPORTANCE OF WOUNDS

It must be determined if the wound was suicidal, homicidal or accidental in nature (**Table 12**).

Table 12: Differences between suicidal, homicidal and accidental wounds

S.No	Feature	Suicide	Homicide	Accident
1.	Type of wounds	Usually incised or stab	Usually lacerations and stab	Usually abrasions, contusions and lacerations
2.	Number of wounds	Multiple	Usually single. May be multiple in revenge murders, "overkill"	Multiple
3.	Severity	Mostly superficial (hesitation wounds) wounds which are not fatal. There is one final fatal wound	Severe and extensive	**Extensive** if involved in a serious accident (eg run over by a truck or railway, fall from a great height). Less extensive in less severe accidents
4.	Target area	Accessible parts only (eg front and sides of the body, neck, chest, abdomen, wrists, groin)	Vital parts (head, chest, abdomen)	Usually on exposed parts and bony prominences. If body did not roll over, most wounds would be on one side only
5.	Grouping of wounds	Well arranged in parallel lines	Irregular	Exposed parts and bony prominences
6.	Hesitation marks	Usually present	Absent	Absent
7.	Defense wounds	Absent	Present if victim was awake and resisted	Absent
8.	Direction	From left to right (In right handed persons); and from above downwards	No particular direction	In the direction of vehicle movement (in dragging by vehicles)
9.	Associated findings	(i) Hands never tied or gagged (ii) Signs of struggle not present (iii) no signs of sexual assault	(i) Hands may be tied behind. Person may be gagged (ii) signs of struggle may be present (broken bangles, scratch marks over face and arms etc (iii) In case of lust murders, there may be signs of sexual assault	(i) Never tied or gagged (ii) There may be several minor injuries on bony prominences, besides main fatal injuries (iii) no signs of sexual assault
10.	Weapon/injury causing object	Lying by the side of body. May be grasped firmly due to cadaveric spasm	Absent (killer takes it away)	Absent (if hit and run accident). Present in other instances (eg fall from height)
11.	Clothes	Not damaged, as they are usually removed. Stabs etc on abdomen would be made by lifting the clothes to visualize the area	Usually damaged and torn due to struggle. Stab etc are through the clothes	Torn and damaged. Soiled extensively with dirt, grass, grease, mud, oil, sand, soil etc. May contain glass particles, if glass was shattered from vehicle (window panes, head lights etc)
12.	Scene of crime	(i) Usually room locked from inside (ii) no disturbance of surroundings (iii) Suicide note may be found	(i) Room never locked from inside (ii) surroundings disturbed (eg overturned flowerpots, displaced tables and chairs, wrinkles on carpets etc) (iii) suicide note not found	(i) very rarely in a closed room. Generally outside (ii) Car tire marks going through the body may suggest run over (iii) suicide note not found
13.	Motive	Present on the part of suicide (chronic disease, failure in love, debt, worries, failure in exam etc)	Present on the part of a killer (revenge, robbery, sexual assault, domestic quarrels, financial or land disputes)	Absent

13. Firearm Injuries

I. INTRODUCTION

A. Firearm

A ***firearm*** is a **thermodynamic machine** in which the **potential energy** of the gun-powder is transformed into the **kinetic energy** of the projectile.

B. Ballistics

Ballistics is the science which deals with the motion of projectiles.

1. Internal ballistics

Internal ballistics deals with what happens to a cartridge and its bullet from the time the trigger of the gun is pulled until the bullet exits from the gun barrel. Also known as ***initial***, ***interior*** or ***proximal ballistics***.

a. Muzzle velocity

Muzzle velocity is the speed at which a projectile leaves the muzzle of the gun (cf striking velocity). Muzzle velocities range from **subsonic** (i.e. less than the speed of sound 340 m/s or 1200 ft/s) for some air guns to **supersonic** [about 1,800 m/s (5900 ft/s)] for tank guns.

2. External ballistics

External ballistics - deals with what happens to a bullet or projectile from the moment it leaves the gun barrel to the moment of impact on a target or object. Also known as ***exterior ballistics*** or ***intermediate ballistics***.

3. Terminal ballistics

Terminal ballistics – deals with study of behavior of missiles after they penetrate their target.

a. Wound ballistics

Wound ballistics is a subset of ***terminal ballistics*** when the target is living tissue (human or animal)**.**

b. Striking velocity

It is the velocity of the projectile at the point of impact (cf muzzle velocity).

4. Forensic ballistics

Forensic ballistics is scientific investigation of firearms in a criminal or civil matter.

II. CLASSIFICATION OF FIREARMS

Table 1 gives classification of firearms based on **five** criteria, of which **first** is **most popular**.

Table 1: Classification of firearms

[A]	**According to condition of barrel:** (1) Rifled firearms (2) Smooth bore firearms
[B]	**According to Manufacturing process:** (1) Factory made (2) Country made (katta) (3) Unorthodox guns (pen guns, zip guns, stud guns)
[C]	**According to muzzle velocity of projectile:** (1) Low velocity firearms (<340 m/s; 1200 ft/sec) (2) Medium velocity firearms (340 – 850 m/s; 1200-3000 ft/sec) (3) High velocity firearms (>850 m/s; 3000 ft/sec)
[D]	**According to use:** (1) Used by civilians [pistol, revolver etc] (2) Used by police under specialized circumstances [Riot guns] (3) Used in war [Artillery] eg Field guns, Anti-aircraft guns
[E]	**Military Classification:** (1) **Small arms** - Denotes infantry weapons an individual soldier can carry. Revolvers, pistols, rifles, shot gun etc are small arms. (2) **Crew-served weapon** [syn, infantry support weapons]– Any weapon system that requires a crew of >1 person due to weight and bulk. **Ex** - Medium and heavy machine guns, automatic grenade launchers, mortars with calibers less than 120 mm (4.72 inches), anti-tank guns, anti-aircraft guns, recoilless rifles, shoulder-launched missile weapons and static anti-tank and anti-aircraft missiles.

A. According to Condition of Barrel

1. Rifled firearms

A ***rifled firearm*** is one which has spiral grooves (riflings) inside its barrel, which impart spin to bullets. Just as the grooves inside a bolt make the nut turn around, grooves inside rifled weapons make the bullet revolve (**Fig 13.1**).

When bullet exits the rifled firearm, it is revolving around its own axis at 3000 rev/sec (**Fig 13.2**). This stabilizes the bullet, and gives it better penetrating power. If no rifling were present inside the firearm, the bullet would topple over very soon. Rifled firearm is more lethal. It is used to shoot large animals. It fires **bullets** (cf smooth bore firearms which fire pellets). **Ex. Pistol, Revolver, Rifle, Carbine, Air rifle.**

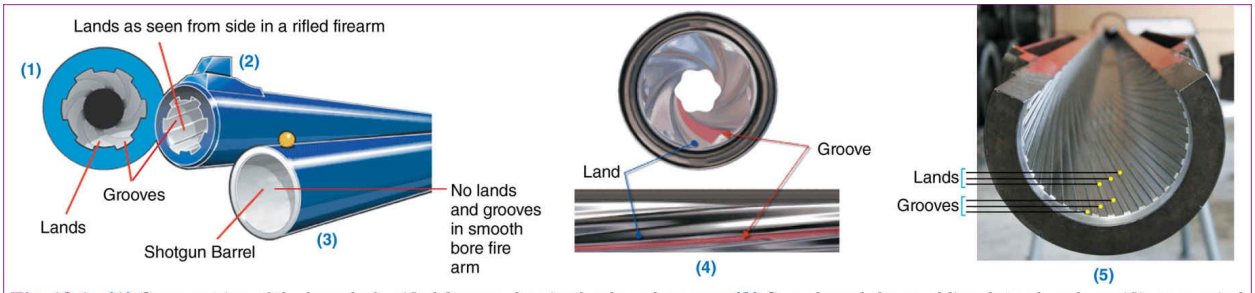


Fig. 13.1: **(1)** Cross section of the barrel of a rifled firearm showing lands and grooves **(2)** Same barrel shown obliquely to show how riflings or spiral grooves etched inside the barrel are responsible for creation of lands and grooves **(3)** A smooth bore firearm (shotgun) shows no such riflings, and hence no lands and grooves **(4)** Barrel of a rifled firearm cut open both transversely and longitudinally to show correspondence of lands and grooves in both views **(5)** A detail from inside of a rifled firearm. Pl note the riflings inside the barrel.

Fig. 13.2: Spin imparted to a bullet by rifled firearms

a. Lands and Grooves

A rifled firearm has ***spiral grooves*** inside its barrel. They range from **2 to more than 20** [**6 grooves** are **commonest**. They may be clockwise or anticlockwise (depending on manufacturing company). The raised portions inside the barrel are

known as **lands**, and the grooved portions, the **grooves** (Fig 13.3). The land width is generally in the range of .069-.090”; groove depth is usually between 0.002” and 0.005” (lead bullets require deeper grooves than copper jacketed bullets).

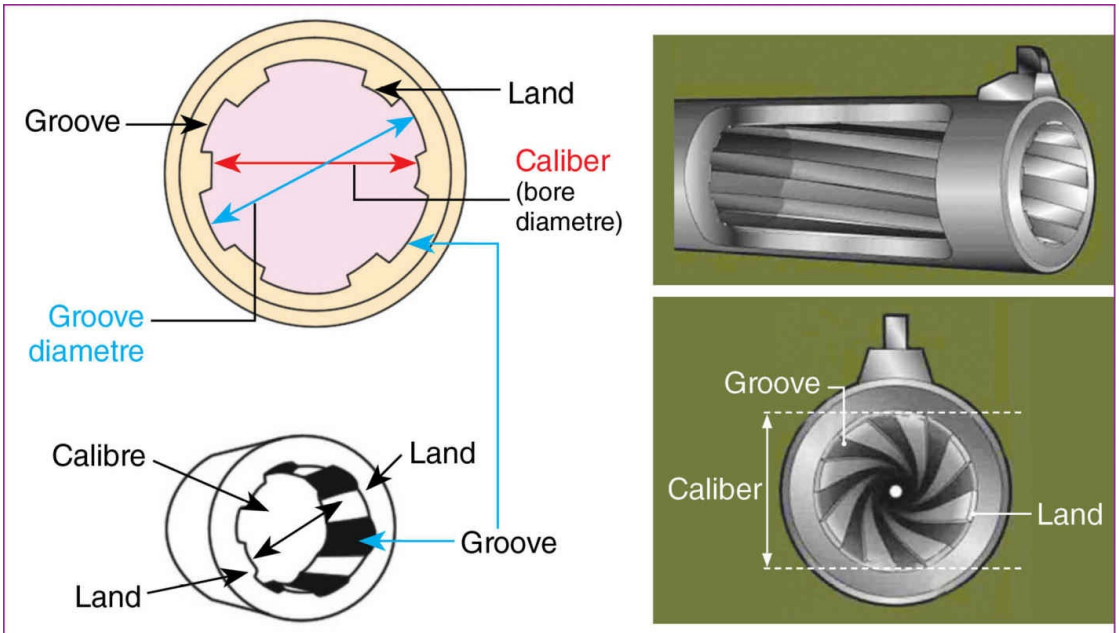


Fig. 13.3: Lands and grooves inside a rifled firearm. **Caliber** is the distance between two opposite lands. **Groove diameter** is the distance between two opposite grooves.

Primary and secondary markings

In all rifled firearms, the bullet is slightly larger than the barrel [so that it can provide perfect obturation]. Thus as it passes through the barrel, not only do the lands and grooves leave their mirror image on it (**primary markings,** Fig 13.4), but also the unique linear markings which are specific to each gun (**secondary markings**) [**ch 30**"class evidence and individualizing evidence]. The sides of the barrel in fact act as a “machine die” to reproduce all markings on the bullet, which comes out carrying a faithful reproduction of all markings inside the barrel. [A] **Primary markings** can reveal the manufacturer of the gun (since each company has different number of riflings, their widths and depths, the direction of their rotation and twist etc). They are part of **class characteristics** of a fired bullet [please see below] [B] **Secondary markings** [Fig 13.4] can pinpoint the individual gun, and are due to individual variations in each gun. These can in turn be due to (i) unavoidable individual variations in minor details

of rifling at the time of manufacturing itself (ii) repeated wear and tear of each gun (iii) gun cleaning activities by the user (iv) metallic fouling – each time a shot is fired, the bullet leaves some metal inside the barrel, which hardens. Because of the infinite possible variations, each gun leaves its own characteristic secondary markings on the bullet, which are often called the "fingerprint of the gun". The phrase is however not entirely correct; while fingerprints do not change during the life an individual, markings made by guns change gradually over the life of a gun – as it wears out more and more. They can also altered by the gun owner very easily by cleaning it repeatedly. They are synonymous with **individual** or **accidental characteristics** of a fired bullet [please see below]. [C] Primary, secondary and other markings described below are not seen if bullet was discharged without the agency of a firearm [eg heat-induced firing; please see above].

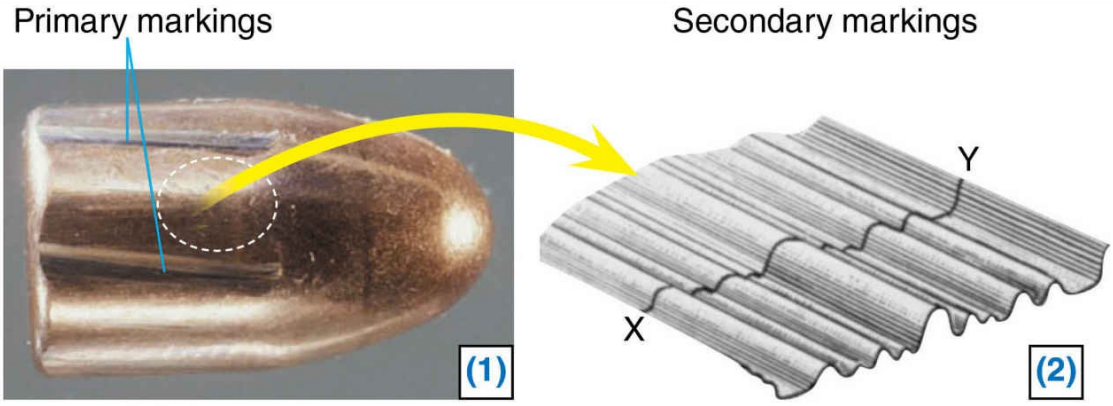


Fig. 13.4: (1) Primary and secondary markings on the bullet. While primary markings are visible to the naked eye, secondary markings are often not. **(2)** These can be seen on microscopic examination as minuscule mountains and valleys. Line XY shows the general contour of secondary markings. While primary markings identify the manufacturer, secondary markings identify the individual gun.

b. Caliber or Gauge

Caliber (***Gauge***) is the internal diameter of a rifled firearm.

Salient features:

(1) Traditionally it is measured between **two opposite lands** (Fig 13.5) [**Groove or rifling diameter** is the distance between two opposite grooves].

(2) **Units** - (i) measured either in caliber units [one caliber equals 1/100 inch] or mm.

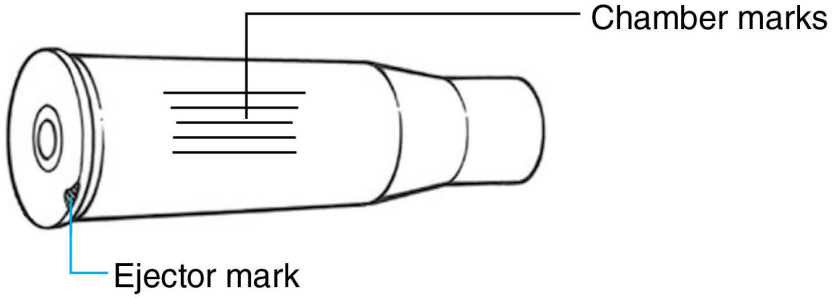


Fig. 13.5: Chamber marks and ejector marks

c. Examples of rifled firearms

i. Revolver

A ***revolver*** is a rifled firearm that has a cylinder containing multiple chambers (usually 6) where cartridges are placed for firing.

ii. Pistol

Pistol is a hand arm which uses cartridges contained in a vertical ***magazine*** [syn, clip], placed within the butt.

iii. Rifle

A ***rifle*** is a firearm with a long barrel, the bore of which is rifled.

(1) **Muzzle velocity** – 450-1500 m/s [average – 800 m/s]

(2) **Pressure in firing chamber** – 20 ton/sq inch

(3) **Range** – 1000-3000 meters [**Table 2**].

iv. Carbine

A ***carbine*** [German: Karabiner] is a shortened version of rifle (having a shorter barrel) firing the same ammunition at a lower velocity, used especially by mounted troops.

Salient features:

(1) **Barrel** – is <55 cm in length

(2) **Muzzle velocity** – varies according to the make. A typical carbine [The M1 carbine] has a muzzle velocity of 600 m/s. There are carbines with lesser and higher muzzle velocities, eg Standard .30 carbine"(580 m/s) and M4 carbine"880 m/s.

(3) **Range** – 300 meters.

v. Other firearms

Gyrojet – A family of unique firearms developed in the 1960s. Named for the

method of gyroscopically stabilizing its projectiles. Firing small rockets rather than inert bullets, they had little recoil and didn't require a heavy barrel to resist the pressure of the combustion gases. Velocity on leaving the tube was very low, but increased to around 1,250 feet per second at 30 feet. The result was a very lightweight weapon with excellent ballistics. Now they are out of production. Fuel is kept at the base of projectile, which keeps burning during its flight. Because of this, the entrance wound shows a singeing effect even at very large distances.

2. Smooth bore firearms

A ***smooth bore firearm*** is one whose barrel is smooth from inside. It fires **multiple pellets** instead of single bullets. The pellets do not rotate on their axis as does the bullet. A smooth bore firearm [**Ex**. **Shotgun, *M*usket (s*m*ooth)**] is less lethal, less accurate and has a lesser range than a rifled firearm. It is used to shoot small, moving targets (birds, hare etc), which change their direction unpredictably. Multiple pellets ensure that at least one pellet hits the "moving" target.

a. Bore

Bore is the internal diameter of a smooth bore firearm. What is caliber (please see above) to a rifled firearm is bore to a smooth bore firearm. Traditionally (and for historical reasons), it is **not expressed in inches or mm,** but in a very different way – the bore. The ***bore*** is defined as the ***number of equal sized spherical balls*** made from ***1 pound (454 g) of lead***, each of which exactly fits the inside of the barrel. Thus the **12 bore gun** is one whose diameter is that of a ball of lead of such a size that 12 such balls could be made from 454 g of lead. The smaller the bore, the larger the diameter of firearm. A **20 bore gun** has a **smaller diameter** than that of a **12 bore gun** (**Fig 13.10**).

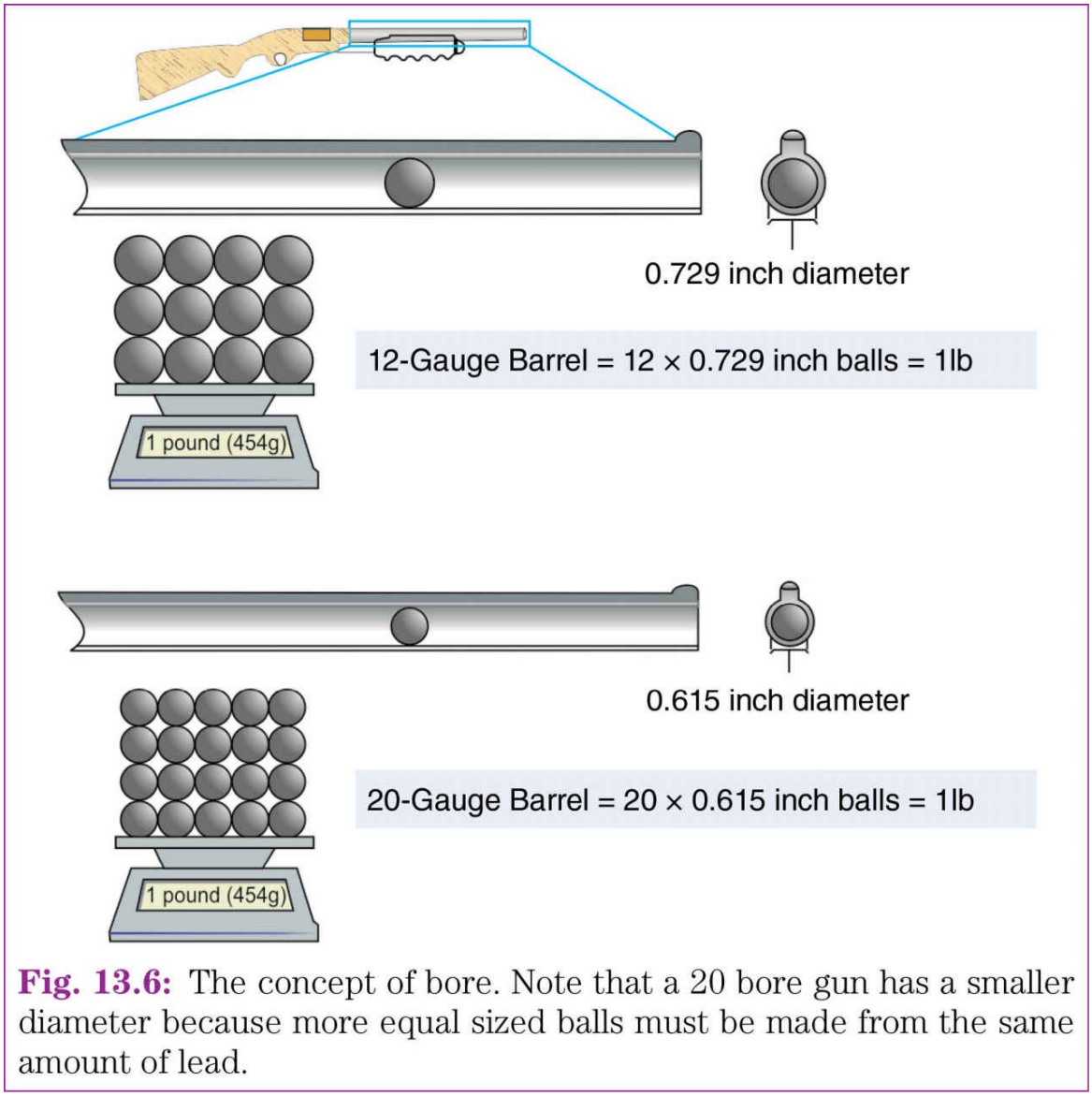

0.729 inch diameter
12-Gauge Barrel = 12 × 0.729 inch balls = 1lb
1 pound (454g)
0.615 inch diameter
20-Gauge Barrel = 20 × 0.615 inch balls = 1lb
1 pound (454g)

Fig. 13.6: The concept of bore. Note that a 20 bore gun has a smaller diameter because more equal sized balls must be made from the same amount of lead.

b. Examples of smooth bore firearms

i. Shot gun

A ***shotgun*** (also known as a scattergun or peppergun) is a firearm that fires several spherical pellets (shots) simultaneously. Sometimes a single projectile (slug) is also fired.

Salient features:

(1) Shoulder arm

(2) **Barrels:** (i) Like rifles, they may be single barreled or double barreled. (ii) The barrels may lie side by side or one above the other [Over-and-under shotgun]. Single barrels and side-by-side are more common. (iii) Length -

varies from 18–36” (45–90 cm), with 26 and 28” the most common. [**U.S. federal law** requires a minimal barrel length of **18”** for a gun to be classified as shotgun]. The length of the barrel makes handling and hiding difficult; barrels are thus often shortened [**sawn off**] for criminal activities.

(3) **Range** - Effective, 30–40 m; terminal, 50 m.

(4) **Muzzle velocity** – 300 m/s.

(5) **Bore** – (for definition, please see above) May vary from **4 to 40**. (i) **.410** - This is the only bore, which is referred to by the size in inches [spoken as “four-ten”]. If converted to bores, it would be 68 bore. So called because it was originally made from worn out .303 rifles. It fires a single lead shot of 6.2 g with a muzzle velocity of 1700±100 ft/sec. In India, it was originally used by **Railway Protection Force**, but its use is now obsolete.

(6) **Insertion of cartridges** - Shotgun is made to “break” or open on a hinge for insertion and extraction of cartridge cases

(7) **Loading** – can be done in two ways just as in rifles. Muzzle loading shotguns are commoner than muzzle loading rifles

(8) **Choke** – If the entire barrel from breech to muzzle end is of same diameter, it is called a **cylinder bore, true cylinder** or **unchoked** gun. If a shot gun has a cylinder barrel, the individual pellets tend to scatter, decreasing the probability that the target will be hit. To keep the pellets together, the terminal 7-10 cm of the barrel is constricted. This constriction is referred to as choke.

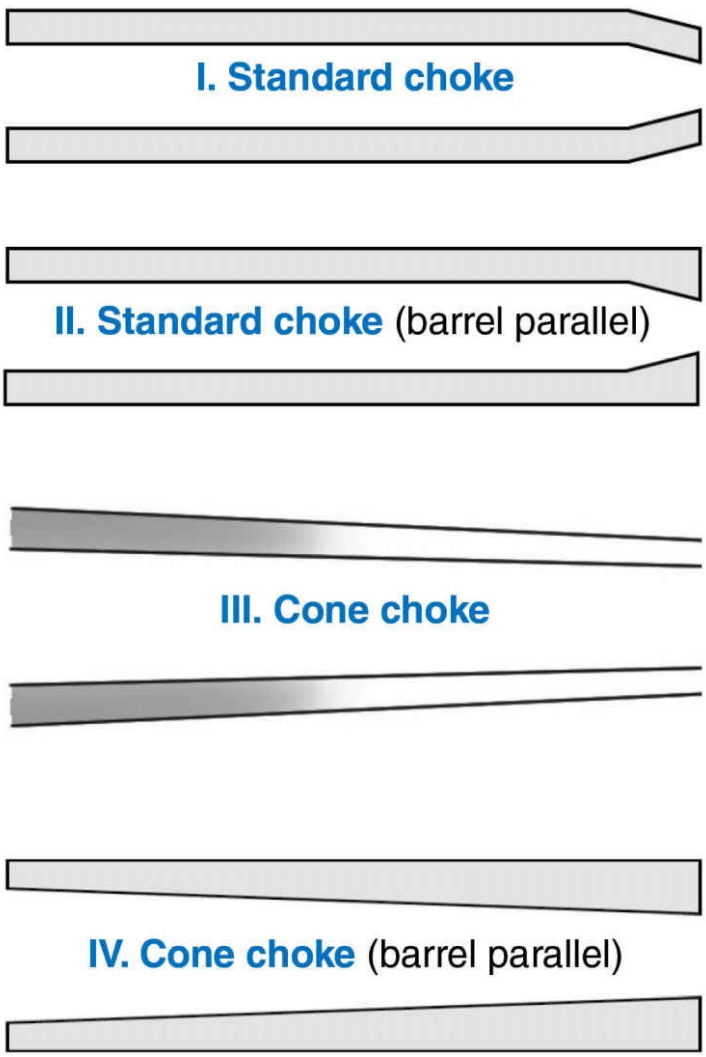


Fig. 13.7: Choke types according to shape. (I) and (II) Standard choke. (III) and (IV) Cone Choke. Note that in (II) and (IV), outer aspects of barrel are parallel. In (I) and (III), the outer aspects of the barrel make the choking obvious even on a casual examination of the weapon from the outside.

ii. Musket

A ***musket*** is a muzzle loading, smooth bore firearm designed for use by infantry.

A soldier armed with a musket is called a musketeer.

Salient features:

(1) It is a shoulder arm

(2) **muzzle velocity** – 350-600 m/s.

3. Air guns

An ***air gun*** can be rifled (air rifle, air pistol), or smooth bore. It fires projectiles by means of ***compressed air*** or ***other gas (CO_2)***, in contrast to a firearm which burns a propellant.

Salient features:

(1) Most air guns use **metallic projectiles** as ammunition. Air guns that only use **plastic projectiles** are classified as **airsoft guns**. It is not really a separate class of firearms.

(2) **Muzzle velocity** – 80-105 m/s [average – 100 m/s]

(3) **Range** – Effective, 30 m; Terminal, 40 m

(4) **Injuries** – Usually minor. Death can occur from injury to head, heart and abdomen.

(5) **Exit wounds** – usually not seen, because of low kinetic energy.

Table 2: Some important figures regarding firearms

S.No	Firearm	Breech pressure [chamber pressure]	Muzzle velocity	Revolution of projectile	Effective range [distance up to which serious wounding can occur]	Terminal range [distance up to which ammunition will travel]
1.	Rifle	20 ton/sq inch	800 m/s	3000/s	1000-3000 m	1000-3000 m
2.	Pistol	6 ton/sq inch	400 m/s	Variable	200 m	400 m
3.	Revolver	5 ton/sq inch	200 m/s	Variable	150 m	200 m
4.	Shotgun	1 ton/sq inch	300 m/s	-	30-40 m	50 m
5.	Air rifles	Very little	100 m/s	-	30 m	40 m

III. CARTRIDGE

A ***cartridge***, (called a "***round***", when it is loaded within the gun), is a single unit, comprising of four components (i) the case (shell) (ii) the projectile (bullet in case of rifled firearm, and pellets in case of smoothbore firearms), (iii) propellant (gunpowder) and (iv) primer. The latter three are enclosed within the case, which is made to fit the firing chamber of a firearm precisely. **Fig 13.8** depicts a rifled firearm cartridge; **Fig 13.9**, a smooth bore firearm cartridge. Cartridges are

always used in ***breech loaders***. In muzzle loading guns each component of the cartridge, eg gunpowder, wads, ammunition etc is introduced separately from the muzzle end [please see “loading mechanisms” above].

A. The Rifled Firearm Cartridge

The rifled firearm cartridge (**Fig 13.8**) - It consists of a **metal case** (1) in which is filled the **propellant** [gunpowder] (2). The metal provides strength and hardness to the case; it must withstand pressures up to 40,000 pounds per square inch, generated by the gases produced. The base is rimless in case of pistol, but has a **rim** (3) in revolver and rifles both (in the figure it shows a rim). The **primer** (4) is filled in the rim (in rim fire ammunition, as in the picture). The **bullet** (5) fits into the upper end of the case, the **case mouth** (6) the edges of which are “pressed” into the body of the bullet, leaving an impression on the bullet - the **cannelure** (7). It serves to hold the bullet in place. The bullet may have **knurls** (8), which hold lubricant. Lubricant serves two purposes (i) it lubricates the interior of the barrel as the bullet grazes past it. This makes it easier to clean it later. (ii) provides for a better seal for gases coming from behind. **Knurls** may be visible on the outside (as in the diagram), or beneath the neck of the cartridge case and not visible.

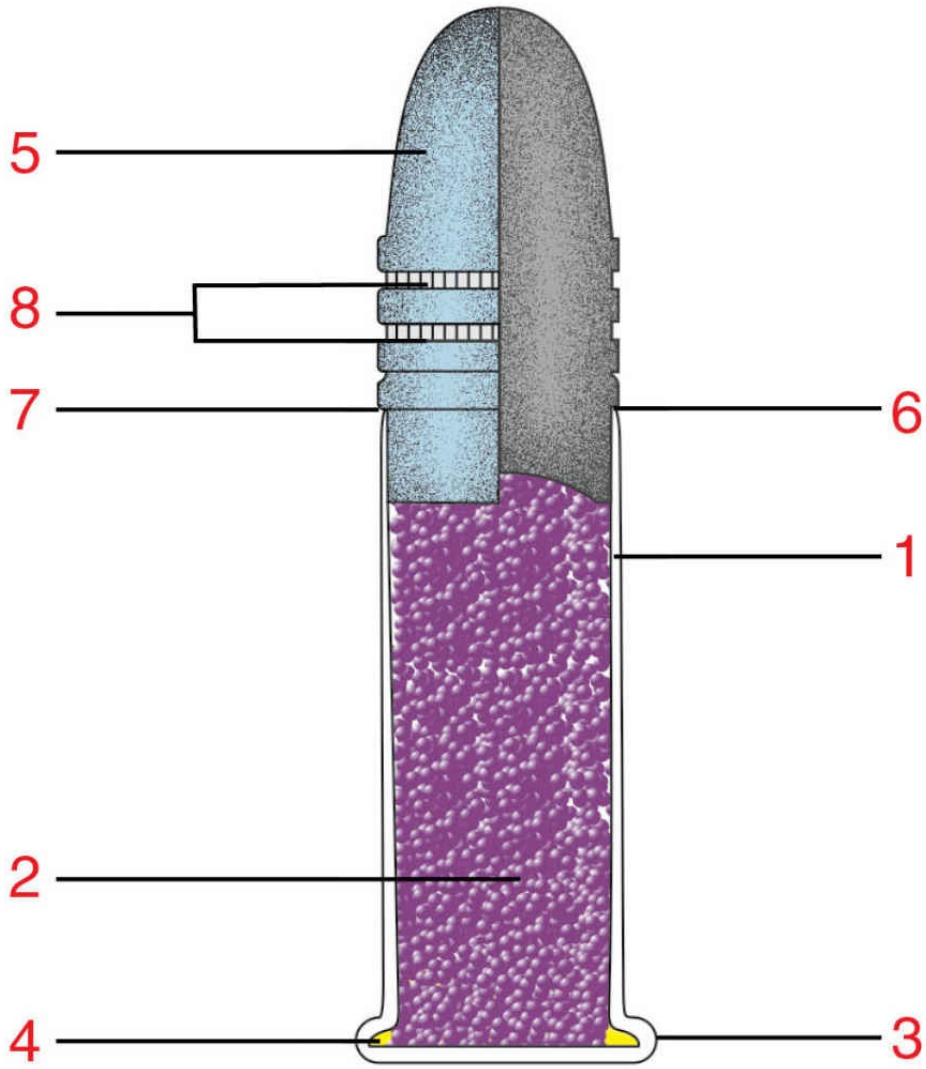


Fig. 13.8: Rimfire Cartridge of a rifled firearm, showing various parts. Please correlate nos (given in red). with text.

B. The Smoothbore Firearm Cartridge

The smoothbore firearm cartridge [**Fig 13.9**] – It consists of a paper or plastic **case** (1), which keeps all components in place, and provides a waterproof container for the propellant (2) (gunpowder). The base is covered in a thin brass

covering, (12) to provide it with strength; it must be hard enough to withstand ramming (pushing in) and extraction (pulling out). In “high brass” cartridges (more powerful) the brass extends further up along the sides of the shell, while in “low brass” cartridges (less powerful) the brass does not extend so far up. The brass not only provides strength to the base of cartridge, but the difference in appearance also provides shooters with a way to quickly differentiate between high and low powered ammunition. Sometimes entire cartridge may be made up of solid brass. At the bottom is the **propellant** (2) and the **primer** (4). There is a **rim** (3) at the base to hold the cartridge securely in chamber; it also makes extraction easy. The powder is covered by an **overpowder card disc** (5). It acts as the gas seal (known as **obturation**), and is placed firmly over the powder; it may be made of paper or plastic. Without the overpowder card disc (5) [and two components above it (6) and (7)] the gas would just blow through the shot rather than propelling it. Over it is the **wad** (6) [also known as **card wad** or **cushion**]. It is designed to compress under pressure, to act as a shock absorber and minimize the deformation of the shot, to take up as much space as is needed between the powder and the shot and to act as a piston for the shot above (for this purpose the diameter of wad is kept greater than that of the bore of the gun). Wad also contains grease, which **lubricates** the bore, each time a shot is fired. Functions of card wad thus are (I) Obturation (II) shock absorber (III) to act as piston for shot (IV) lubrication (V) They also act as spacers, setting the correct volume for the propellant and shot charges. One of the functions of overpowder card disc (5) is to protect the powder from the “grease soaked wad”. Wads are made of cork, felt, glazed board, plastic or straw board. Above this is **undershot card disc** (7). It prevents the shots from getting lodged in the felt wad. Also serves to hold the shot together as it moves down the barrel. Both wad and undershot card disc play a part in obturation too. Above this are the **pellets** (8), and over it is the **overshot card disc** (9). Overshot cards give a stable platform for the **crimp** (10) - one of the most critical factors of a proper shotgun cartridge. At the moment of fire the firing pin strikes the base of the primer cup (11) igniting the primer, which in turn sets fire to the gunpowder. Cartridges are numbered according to the shot they contain. The total length of smoothbore cartridge varies from **5-7 cm**.

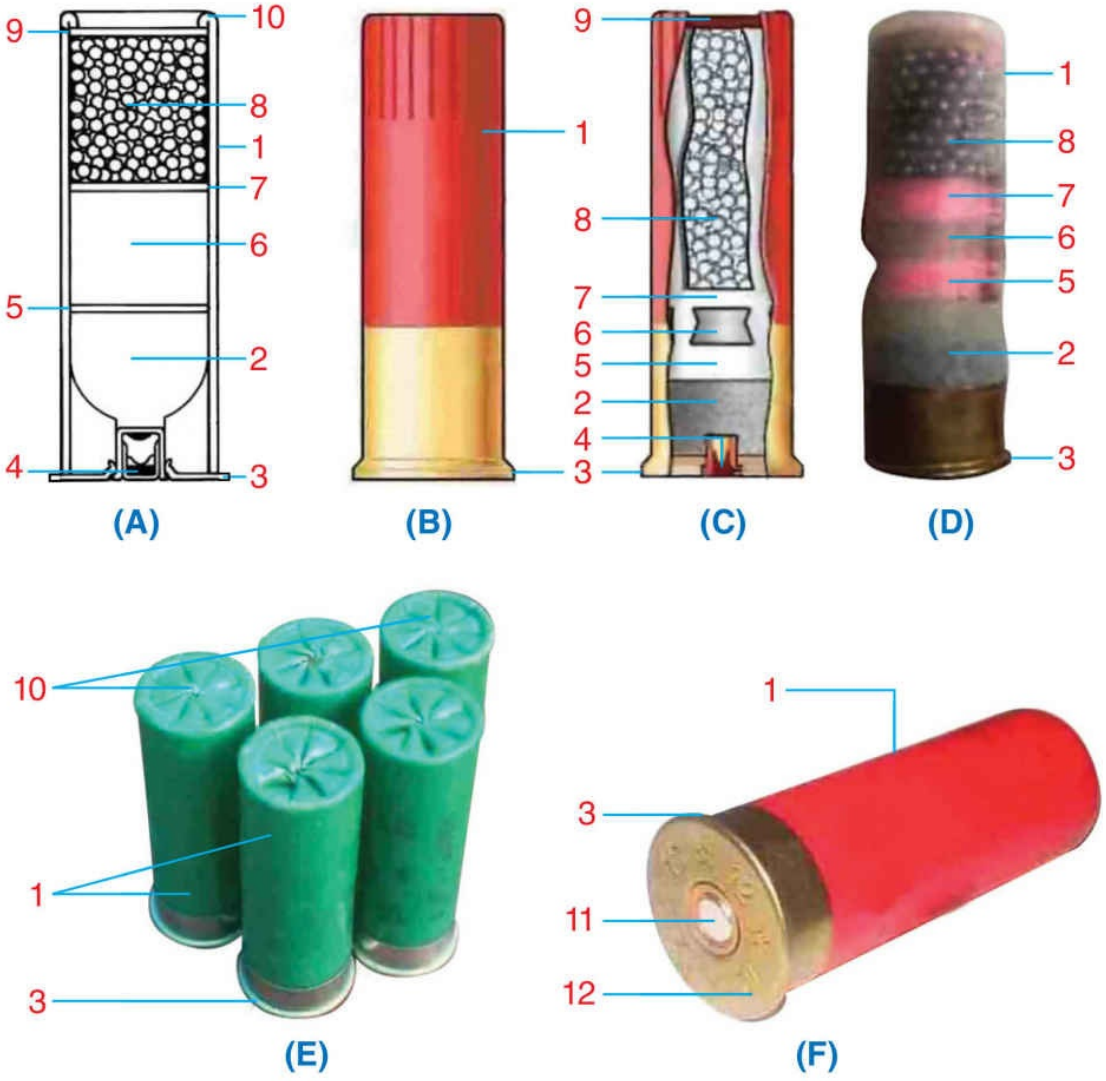


Fig. 13.9: Several different views of the cartridge of a smooth bore firearm showing various part from different angles. (A) Cross section of a cartridge. (B) An upright cartridge from the outside. (C) Another cross section of a cartridge. (D) A cartridge with a transparent case. (E) Top view of a cartridge. (F) A reclining cartridge showing the base. Please correlate nos (given in red). with text.

C. Components of a Cartridge

1. Primer

Primer is shock-sensitive explosive mixture that ignites when struck mechanically. It is a p**rimary explosive** [**ch 15**][hence the name primer]. Primer is kept in a small metal cup that is inserted into a recess in the center of the base

of the cartridge. When the trigger is pressed, the firing pin strikes the cup and crushes the primer between the cup and an anvil to produce a shower of sparks [incandescent particles] and hot gas that pass through a flash hole to ignite the powder charge.

a. Composition of Primer

Memory Aid 1: Composition of primer

B L^2 A S T –

1. **B**arium nitrate
2. **L**ead peroxide
3. **L**ead styphnate
4. **A**ntimony **S**ulfide
5. **T**etracene

Others:

(1) Diazodinitrophenol [DDNP]
(2) Mercury fulminate
(3) Pentaerythritol tetranitrate [PETN]
(4) Potassium perchlorate. Mercury fulminate is not used now in most countries because mercury tends to corrode brass walls of cartridge on long storage. It is being used in India though.

2. Propellant

A ***propellant*** is a material that is burnt to propel ammunition. It may be black powder, smokeless powder or a combination of the two.

Salient features:

(1) Propellants are **secondary explosives** [**ch 15**]
(2) **Primer** merely provides a spark to set the propellant on fire. To put it figuratively, while primer is the spark plug, propellant is the petrol.

a. Black powder

Black powder (also called ***gunpowder***) is a mixture of potassium nitrate (75%), charcoal (15%) and sulfur (10%) by weight. Black powder was first produced by **Roger Bacon** in **1250**.

Salient features:

(1) **Functions of each component:** (i) Charcoal - fuel (ii) Sulfur - (a) binding agent (b) fuel (c) .es the temp of ignition (d) -es the speed of combustion (e)

improves shelf life by reducing water absorption (iii) KNO_3 - Oxidizer.

(2) **Size of grains** - Black powder is essentially a surface burning powder, larger the surface area, the quicker it will burn. Larger surface area is achieved by grinding the black powder very fine. The four most commonly used granulations are graded as **FFFFg**, **FFFg**, **FFg**, and **Fg** (also known as **4Fg**, **3Fg**, **3Fg** and **Fg** respectively). More the F's, finer the grain, with FFFFg being the finest and Fg the coarsest. Thus 4Fg would burn fastest and Fg, slowest.

(3) **Production of gas** - One **gram** of black powder on burning produces **280** cc of gas. The chemical formula involved is: **$10KNO_3 + 3S + 8C$"$2K_2CO_3 + 3K_2SO_4 + 6CO_2 + 5N_2$.** A typical cartridge may contain between 1-20 grams of black powder, depending on the size of projectile, it is expected to propel. P53 Enfield rifle introduced by the British in 1853 contained 4.4 g [68 grains] of black powder.

(4) Gunpowder is a **low explosive** having a **low brisance** [please see chapter 15 – Explosion injuries].

(5) **Obsolete now** - Today black powder is used only in muzzle-loading firearms, blank cartridges, refilled shotgun cartridges and country made cartridges. It is rarely if ever used in standard, factory-made cartridges.

b. Smokeless powder

Smokeless powder is a misnomer – it is not entirely smokeless; it only produces less smoke than the black powder. Thus blackening is produced even with smokeless powders. Smoke is however somewhat grayish white, and it produces blackening of a much lighter color.

Salient features:

(1) **Composition** - Nitrocellulose alone (single-base powder); nitrocellulose+ nitroglycerin (double-base powder); nitrocellulose+ nitroglycerin + nitroguanidine (triple-base powder). Triple base powders are usually restricted to large caliber ammunition.

(2) **Burning** - Produces much less flame and smoke. They are more completely burnt than black powder

(3) **Color** – Varies from bright orange to bluish black

(4) **Shape** – minute globules, flakes, square, rectangles, irregular discs, cylinders and long threads.

(5) One **gram** of nitrocellulose on burning produces **950 cc** of gas.
(6) Smokeless powder is not used in muzzleloading firearms; it can damage the firearm and might injure shooters and bystanders.

c. Semismokeless powder

Combinations of black powder [80%] and smokeless powder [20%]. Though some advantages have been claimed for the powder, the mixing procedure is very dangerous. This has prevented its extensive production and use.

3. Projectile [bullets and pellets]

a. Bullets (used in rifled firearms)

i. Nomenclature of bullet

Traditionally the bullet is made of soft metal (eg lead) with varying amounts of antimony and/or tin added to it to provide hardness. Its various parts are (i) **Bourrelet** - Finely machined ring of metal just behind the ogive (ii) **Caliber** – its cross sectional diameter (iii) **Heel** – base (iv) **Meplat** - The flat area at the tip of bullet [in flat nosed bullets only] (v) **Nose** - Tip of bullet (vi) **Ogive** - The curved area extending backwards from the nose to the main cylindrical portion.

ii. Types of bullets

Bullets may be **classified** according to (a) shape of nose (b) shape of heel (c) their material (d) their covering (e) special purpose they serve (f) Forensic scientist's point of view.

(a) According to shape of nose

(1) ***Hollow point*** [**Fig 13.10(1)**]- is an expanding bullet that has a pit (hollow) in its tip so that it expands upon entering a target. It serves two purposes
(i) maximizing tissue damage and blood loss
(ii) decrease penetration (controlled penetration is necessary where over-penetration could cause collateral damage, as on an airplane).
(2) **Round nose** [**Fig 13.10(2)**]- has a semiblunt, conical shape
(3) **Spire point** [**Fig 13.10(3)**]- Made with two straight lines, joined to the shank at a specified angle [typically 9-12 degrees]. The spire shape is like a pencil tip or "church spire" in shape.
(4) **wadcutter** [**Fig 13.10(4)**]- Bullet specially designed for shooting paper targets, usually at close range and at subsonic velocities typically under 800

ft/s. Used in handgun and airgun competitions. Has a flat front (with a relatively sharp edge along the perimeter) that cuts a very clean hole through the paper target, making it easier to score. Because the flat nosed bullet is not well suited for feeding out of a magazine (used in pistols), wadcutters are normally used only in revolvers. To address this problem, semiwadcutter is used

(5) **semiwadcutter** [**Fig 13.10(5)**]- is designed to be fired from pistols. It consists of a conical section (**a**) that ends in a smaller flat surface (**b**), and a thin sharp shoulder (**c**) at the base of the cone. The flat point punches a clean hole, and the shoulder opens the hole up cleanly.

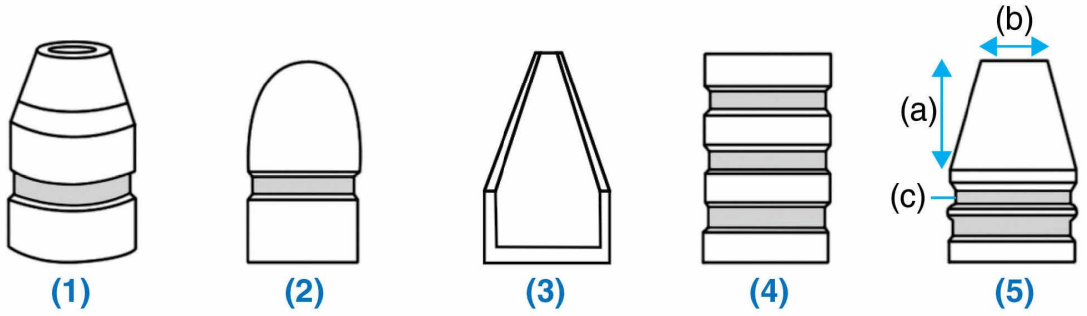


Fig. 13.10: Bullet types according to shape of nose (1) Hollow point (2) Round nose (3) Spire point (4) Wadcutter (5) Semiwadcutter

(b) According to their covering

(1) **Non-jacketed** [syn, unjacketed]– Made up of lead. As they travel down the barrel, lead is scratched off, and sticks to the inside of barrel (**lead fouling** of the barrel). To prevent lead fouling, these bullets are lubricated with grease or some other lubricating compound. This is contained in **knurls** (**Fig 13.8**). Copper gilding is used extensively in .22 high-velocity rimfire ammunition.

(2) **Jacketed:**

(i) Two types (a) **F**ull **M**etal **J**acket (FMJ); **T**otal **M**etal **J**acket (TMJ) bullets - Bullet nose is fully encased by a jacket; base is base (b) Full Metal Case - Both the base and the nose of a bullet are fully encased by the jacket; typical of military bullet where expansion is not desired

(ii) used in semi-automatic pistols.

(iii) Have a lead or steel core covered by an outside jacket. It is made up of gilding metal, gilding metal-clad steel, cupro-nickel (copper and nickel), or

aluminum.

(iv) Jackets generally range from **0.0165 to 0.030 in.** in thickness.

(v) Advantages – (1) Imparts additional strength to the bullet. Help to prevent the bullet from breaking apart (2) higher muzzle velocities are achieved (3) Prevent lead fouling as well as jamming of the gun that occurs if a large number of lead bullets are fired [due to lead deposition on barrel and other areas of the gun] (4) prevents damage to bores from steel or armor-piercing core materials.

(vi) Deformation – Full metal jacketed bullets exit undeformed; unjacketed bullets show deformations

(vii) Armor-piercing bullet [APB] - (a) APB is a special jacketed bullet fired from small arms [Armor-piercing rifle and pistol]. They are meant to penetrate bullet-resistant vests [commonly known as bulletproof jackets] and other tough targets like automobile doors.

(3) **Partially jacketed** [syn - **S**emi-**J**acketed, SJ bullet, soft-nosed bullet, Soft-point bullet – Jacket is left open at the tip, exposing some of the lead inside. Exposed lead expands upon impact.

(c) According to special purpose they serve

(1) **Dum-dum bullet** (**Fig 13.11**):

(i) A special type of expanding bullet [please see below]

(ii) A British military bullet developed for use in India during the late 1890s.

(iii) Jacketed .303 bullet with the jacket nose open to expose its lead core.

(iv) Immediately balloons out on impact with the target thus increasing the damage.

(v) The phrase ‘Dum-Dum’ is now taken to include any soft-nosed or hollow pointed bullet.

(2) **Expanding bullet:**

(i) Expands on impact, increasing in diameter to produce a more serious wound.

(ii) Also known as **mushroom bullet**, because of the mushrooming that occurs on impact.

(iii) The two typical designs are the (a) **hollow point bullet** [please see above] and the (b) **soft point bullet**. (c) Dum-dum bullet is a special type of expanding bullet

(3) **Frangible bullet**:

(i) Made of bonded powdered iron [or lead], which fragments on impact.
(ii) Caliber – mostly .22
(iii) Normal use – target practice by police, shooting galleries, to stun cattle
(iv) Recovery and matching - of fragments is difficult because of fragmentation
(v) They do not ricochet [please see below]
(vi) If bone is penetrated, they are usually recovered in an eroded state.
(vii) D/d - An x-ray would reveal several fragments which look like small pellets. One may wrongly conclude that the victim was hit by a shot gun

(4) **Tracer bullet** - Special kind of bullets that contain a powder in their base that burns very brightly during their flight. This enables the shooter to follow the bullets' trajectories, so he can adjust his aim accordingly. It is used both in ground as well as aircraft guns.

Fig. 13.11: Dum-dum bullet after firing and recovery from target. Note expansion (mushrooming).

Fig. 13.12: Tracer bullets from .50 caliber machine gun. Pl note the bright trajectories of bullets.

(d) According to Forensic scientist's point of view

(1) **Crime bullet** - Bullet with which crime is committed. This bullet is recovered from the body of the victim, and is produced in court as evidence; hence also known as **evidence bullet**. Crime bullet is fired by the criminal

(2) **Test bullet** - Bullet that is fired from a suspect weapon by the forensic scientist, so he can compare the markings on the bullet with that of the crime bullet. If the markings match perfectly, the suspect weapon is the actual weapon used for crime. Both crime and test bullet when presented in the court as evidence are known as **exhibit bullet**.

b. Pellets and slugs (used in smooth bore firearms)

i. Types

Pellets are of four **types**

(1) **Soft** or **drop** shot [made of soft lead] and

(2) **Hard** or **chilled** shot [made of lead; hardened by antimony]

(3) **Steel shot** [made of steel. Individual steel pellets weigh less than comparatively sized lead pellets, and thus travel less] and

(4) **Plated shot** [lead shot plated with copper or nickel or both.

ii. Shot size and form

In any one given cartridge, the number of pellets may vary from 1 to several thousand.

(1) **Dust shot** is the smallest size of shot. Up to 2600 tiny particle like shots in a 12 bore cartridge. Used to shoot house sparrows

(2) **Bird shot** - 200-400 shots (.05-.18" in diameter) in a 12 bore cartridge. Mainly used to hunt birds in flight (hence the name). Birdshot sizes are numbered similar to the shotgun gauges; the smaller the number, the larger the shot. Generally birdshot is just called "shot", such as "**number 9 shot**" or "**BB shot**".

IV. FIREARM WOUNDS

A ***firearm wound*** is an injury produced by a firearm.

Salient features:

(1) **Types:**

(i) Entrance [syn, entry, in-shot] wounds

(ii) Exit [syn, out-shot] wounds

(2) **Confusing appearances** - Firearm wounds are normally recognized easily. However they may sometimes simulate other injuries

(i) Bullet strikes glass before striking body – simulate lacerated wounds

(ii) Glancing wounds – simulate incised or lacerated wounds

(iii) Wounds by rifled weapons – simulate wounds inflicted by burning pointed stick or red-hot poker

(3) Differentiation:

(i) Firearm wound causes crushing of tissues [produce an actual hole]

(ii) Stab wounds tend to seal after the knife is withdrawn.

A. Wounding from Firearms

1. Effects of distance on Firearm wounds

[**Fig 13.13**] shows the ejection of flame, smoke and unburnt powder particles from the gun just as it is fired. Flame travels the shortest distance; smoke goes farther; and unburnt powder particles go farthest.

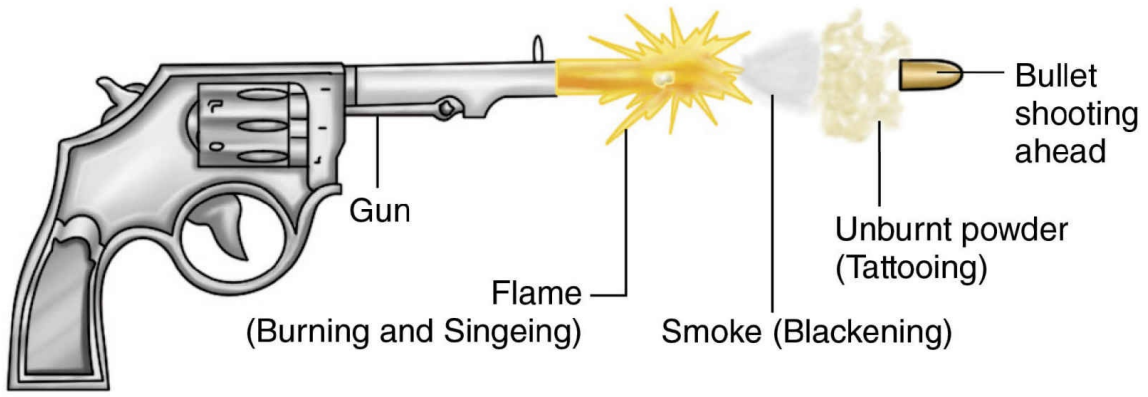


Fig. 13.13: Figure illustrating the effects of distance on firearm wounds. Note that burning, singeing, blackening and tattooing are caused at different distances

a. Flame

(1) Flames causes **burning**, **searing**, **scorching** and **singeing**.

(2) Burning is used in relation to skin and singeing in relation to hair.

(3) **In the hair** – Keratin melts due to excessive heat and then resolidifies on cooling, producing a clubbed, rounded appearance at the tips

(4) Searing and scorching are areas of intense burning; typically seen in near contact wounds.

(5) Darkening and parchmentization of burnt area – occurs after death due to drying.

b. Smoke

(1) Produced by complete combustion of gun powder.

(2) Responsible for **blackening** [syn, **fouling**, **smudging**]. Primer residues also do not enter the skin, and produce blackening

(3) Smoke simply deposits on the skin, without actually entering inside the dermis [**Fig 13.14**].

(4) **Appearance** – Normally black, but if powder is smokeless, grayish or white deposit.

(5) **How to detect:**

(i) Take a wet cotton gauge, sponge or towel and try cleaning the wound; the

blackening would vanish. Cotton would be blackened.

(ii) IR photography – detects blackening on both skin and clothing.

(iii) In bone - Lead may be rubbed off from bullet, which may look like soot. Test for metals would be negative in true blackening.

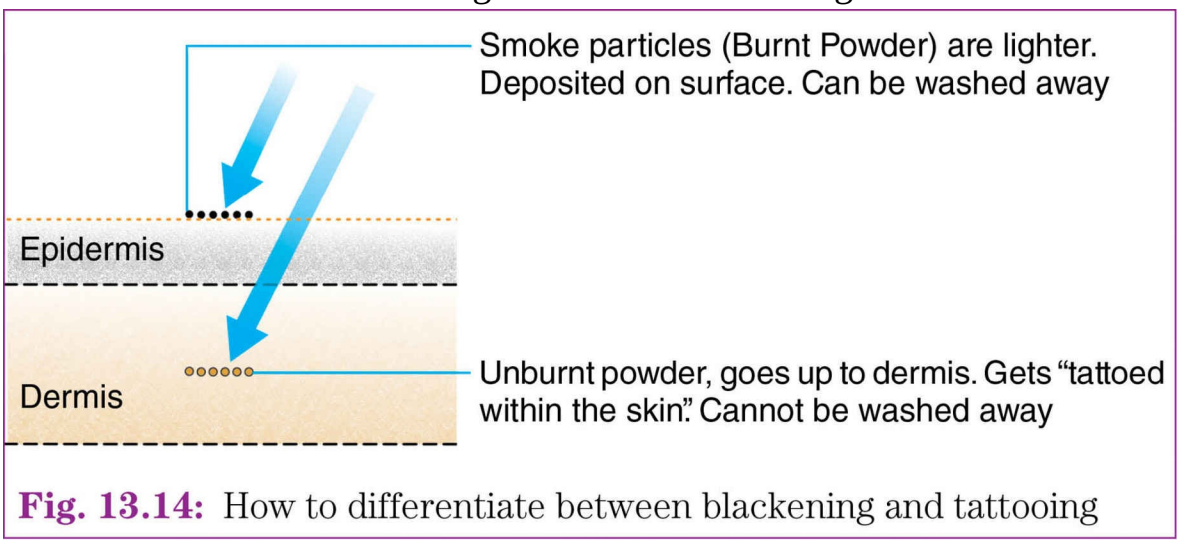

Smoke particles (Burnt Powder) are lighter.
Deposited on surface. Can be washed away
Epidermis
Dermis
Unburnt powder, goes up to dermis. Gets "tattoed within the skin". Cannot be washed away

Fig. 13.14: How to differentiate between blackening and tattooing

c. Unburnt powder

(1) Produced by incomplete combustion of gun powder, which has the consistency of very fine sand.

(2) **Appearance:**

(i) Multiple punctate abrasions - seen as small dark brown dots [individual tattoo point] surrounding the wound.

(ii) Individual tattoo points - are surrounded by a small magenta colored zone of micro-contusion. Caused by the trauma of high speed impaction of powder grains with rupture of small bv.

(3) **How to detect:**

(i) Take a wet cotton gauge, sponge or towel and try cleaning the wound; tattooing would not vanish. Cotton would not be blackened.

(ii) Can be identified under a hand lens or dissecting microscope, picked up for analysis [by a little probing] and even ignited with a red hot wire for confirmation.

(iii) IR photography – detects tattooing on both skin and clothing.

(4) **Measurement** - Tattooing must be measured in all directions around the wound. Tangential wounds produce unequal tattooing [also burning, singeing and blackening] around the wound [**Fig 13.15**].

(5) **Range** - If tattooing is visible, the wound is termed an intermediate-range gunshot wound [please see below].

(6) **Miscellaneous points:**

(i) Palms and soles are very resistant to powder tattooing [thick skin resists entry of powder].

(7) **Table 4** gives important ranges.

Fig. 13.15: This entry wound on left side front of abdomen is caused by a close fire from the left side. Please note heavy concentration of tattooing on the left side. The entry wound is also showing an eccentric abrasion collar which is heavily directed towards the left side. Please also note virtual absence of powder from a broad band. The victim was wearing a belt here. Pl compare with **Fig 13.24** which shows similar distribution with respect to abrasion collar.

Table 3: Table showing how burning, blackening and tattooing are produced and how best they can be detected

Product of firing a gun	Responsible for	Detected by
Flame	Burning, scorching and singeing	Looking at singed hair around the wound
Smoke	Blackening	Wiping the wound with a wet sponge. Blackening vanishes. Sponge becomes dirty.
Unburnt powder particles	Tattooing	Wiping the wound with a wet sponge. Tattooing does not vanish. Sponge remains clear.

Table 4: (with **Memory Aid: 2**)–A simple mnemonic to remember commonly accepted ranges of burning, blackening and tattooing in various firearms. Only one numeral – 3″ – needs to be remembered. Rest are multiples of that as we move across cells (shown by blue arrows).

Effect	Burning and singeing	Blackening (×2)	Tattooing (×2 - ×3)
Pistols and revolvers	3″ [7.5 cm] →	6″ [15cm] →	12″-18″ [30-45 cm]
Rifles (×2)	6″ [15 cm] →	12″ [30cm] →	24″-36″ [60-90 cm]
shotguns (×2)	12″ [30 cm]	24″ → [60cm]	48″-72″ [120-180 cm]

2. Ranges

Please see **Table 5**.

Table 5: Various conventional ranges and associated effects

S.No	Ranges	Effects
1.	Contact	Gases, smoke, unburnt powder enter inside the wound
2.	Near contact	Burning, singeing [flame effects] present
3.	Intermediate	Tattooing present. Blackening may or may not be present depending on distance.
4.	Distant	Burning, singeing, blackening, tattooing all absent.

Memory Aid 3: Relative distances of various effects of firearms

1. **Burn Black Train** - Burning, blackening, tattooing [from inside to outside]. Simple, but with lesser detail.

2. **T**u**B**^{2}erculo**S**i**S** – **T**attooing, **B**lackening, **B**urning, **S**ingeing and **s**corching [from outside to inside]. This memory aid ***adds details to burning effects*** too.

a. Contact

(1) **Characteristics:**

(i) Range - The term is applied when the muzzle of the weapon is held against the surface of the body at the time of discharge.

(ii) In all contact wounds, soot, powder, carbon monoxide [carboxyhemoglobin, COHb], and vaporized metals from the bullet, primer, and cartridge case are deposited in and along the wound tract. Their concentration .es with -ing depth.

(2) **Classification** - Contact wounds may be

(i) hard

(ii) loose

(iii) angled or

(iv) incomplete [a variation of angled].

i. Hard contact

Characteristics:

(1) Muzzle of the weapon is jammed "hard" against the skin, indenting it, so that the skin envelops the muzzle; contact is so hard that gases cannot escape out

(2) immediate edges of the entrance are seared by the hot gases of combustion and blackened by the soot

(3) This soot [along with unburnt powder] is embedded in the seared skin and cannot be completely removed either by washing or by vigorous scrubbing of the wound [**Fig 13.16**]. This is thus a special case, where burnt powder [smoke] also produces tattooing.

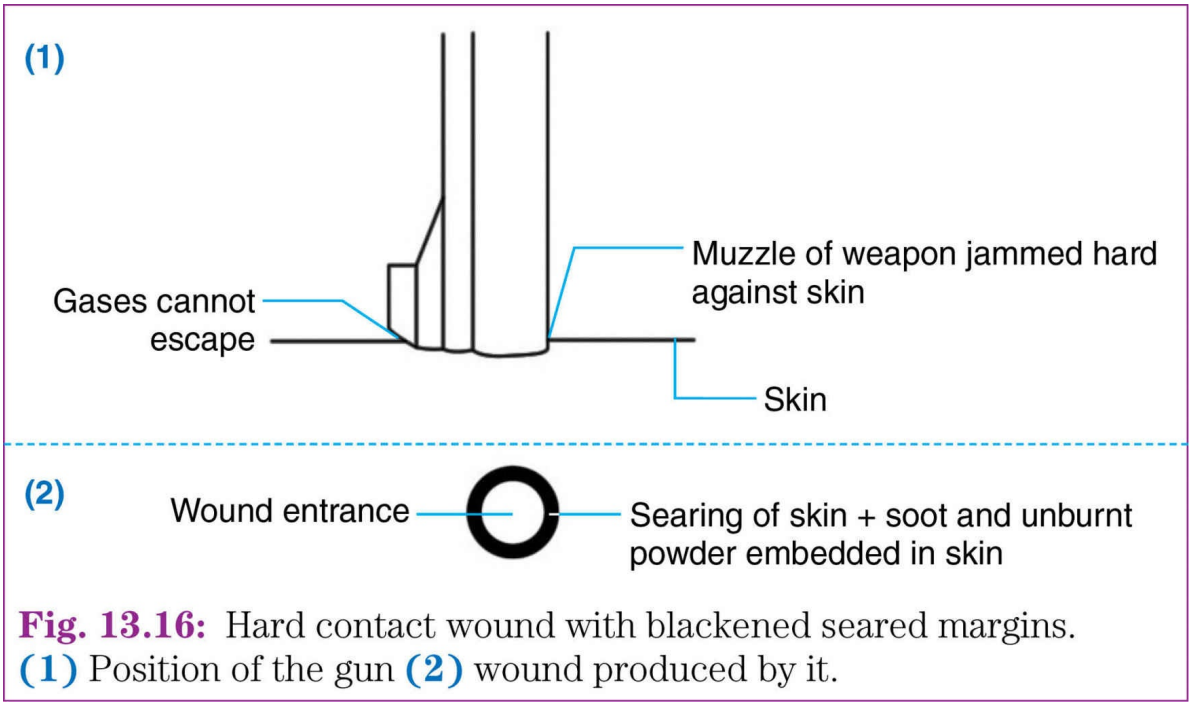


Fig. 13.16: Hard contact wound with blackened seared margins. **(1)** Position of the gun **(2)** wound produced by it.

ii. Loose contact

Characteristics:

(1) The muzzle, while in complete contact with the skin, is held lightly against it.

(2) Gas preceding the bullet, as well as the bullet itself, indents the skin, creating a temporary gap between the skin and the muzzle through which gas can escape. This temporary gap cannot be produced in hard contact wounds.

(3) Soot carried by the gas is deposited in a zone around the entrance

(4) This soot can be easily wiped away.

(5) A few unburnt grains of powder may also escape out this gap and be deposited on the skin around the zone of soot [**Fig 13.17**].

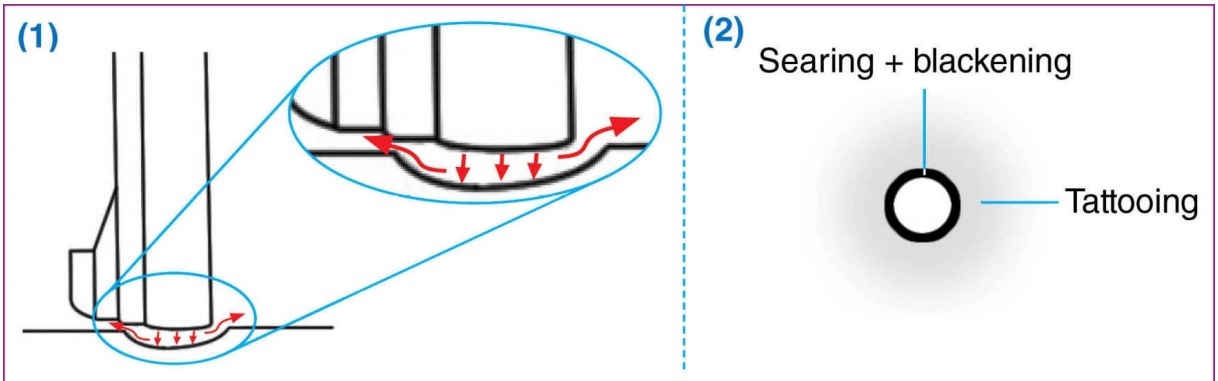


Fig. 13.17: loose contact wound with soot deposited in zone around entrance. **(1)** Position of the gun. Note that there is some gap through which gases can escape. **(2)** wound produced by it. Please compare with **Fig 13.16**.

iii. Angled contact

Characteristics:

(1) The barrel is held at an acute angle to the skin so that the complete circumference of the muzzle is not in contact with it.

(2) Gas and soot escaping from the gap, where contact is not complete, radiate outward from the muzzle, producing an eccentrically arranged pattern of soot.

(3) The soot is arranged in two different zones. (a) More noticeable zone – [often the only one seen] is a smaller blackened seared area of skin or cloth having a pear, circular, or oval configuration [**Fig 13.18**] (b) Less noticeable zone - is a larger fan-shaped zone of light-gray soot that radiates outward from the gap. On the skin (the less noticeable zone with respect to soot) this light zone is usually washed away, or obscured by bleeding or removed in cleaning the wound for examination.

(4) A few unburnt grains of powder may also be deposited in these zones.

(5) The entrance wound is normally present at the base of the seared blackened zone. All or at least the majority of the seared blackened zone will be on the opposite site of where the muzzle was in contact with skin, and thus “points” the way the gun was directed.

(6) As the angle between the barrel and the skin -es, i.e., the barrel moves toward a perpendicular position to the skin, the entrance hole will be found more toward the center of the zone.

(7) If the angle between the barrel and the skin .es, the gap between the muzzle

and skin becomes larger, and more material can escape through the gap.

(8) At some point, the gap becomes sufficiently large that unburnt grains of powder escaping through the gap will skim over the zone of seared skin, fanning out from the entrance, impacting distal to the entrance wound in a fan shaped pattern of powder tattooing.

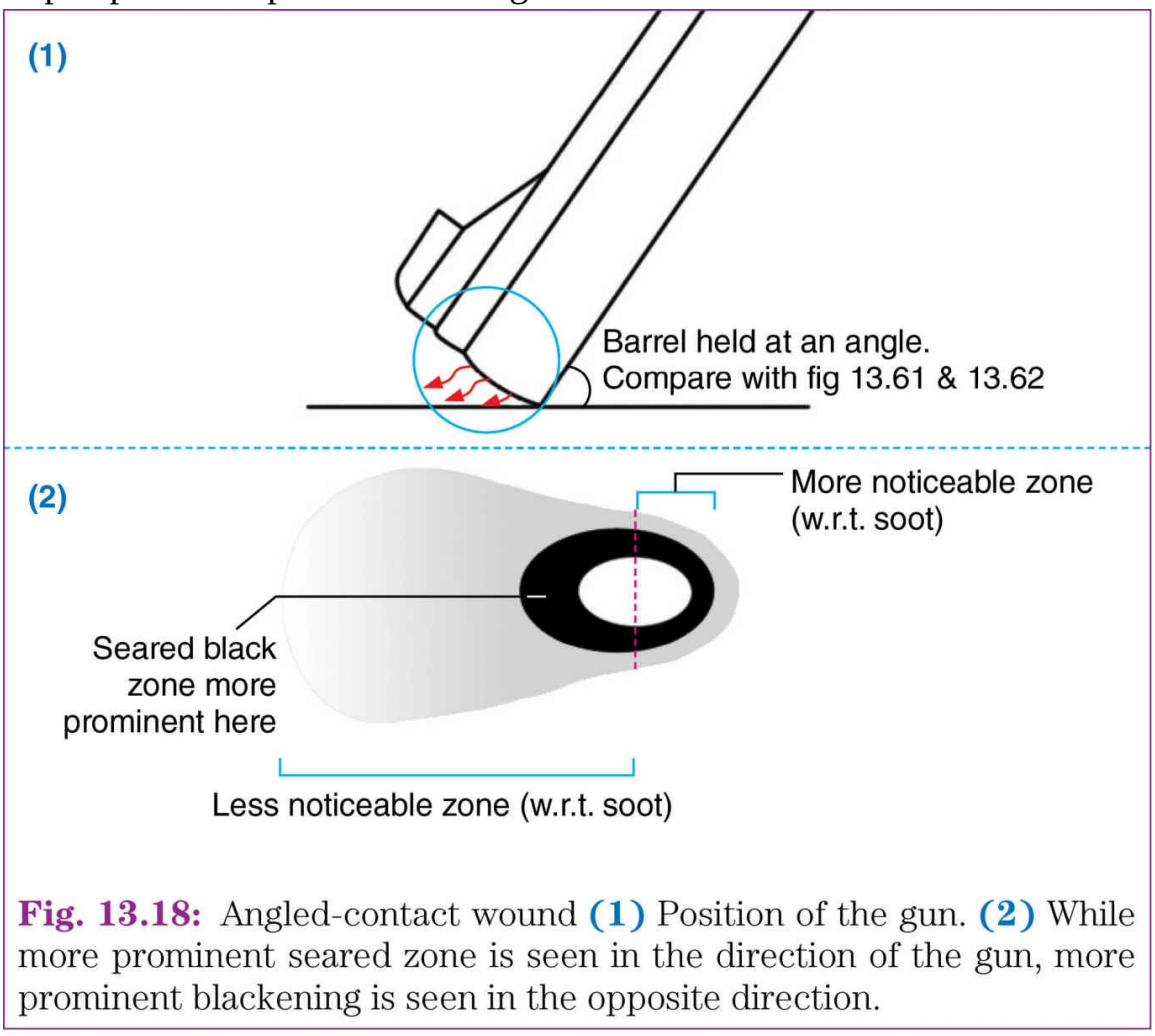


Fig. 13.18: Angled-contact wound **(1)** Position of the gun. **(2)** While more prominent seared zone is seen in the direction of the gun, more prominent blackening is seen in the opposite direction.

iv. Incomplete contact

Characteristics:

(1) This is a variation of angled-contact wound.

(2) Muzzle of the weapon is held against the skin, but because the body surface is not completely flat, there is a gap between the muzzle and the skin [**Fig 13.19**].

(3) Difference with angled contact – In angled contact, the barrel is held intentionally at an angle. In incomplete contact there is no intention to hold it

an angle; angle is produced because of curvature of surface.

(4) A jet of soot-laden gas escapes from this gap producing an area of seared, blackened skin. Powder grains may also be deposited on the skin.

(5) Location of this seared, blackened zone can be anywhere in relationship to the muzzle circumference, depending on where the gap is.

(6) Incomplete contact wounds are most often seen in self-inflicted contact wounds of the head due to long arms, i.e., rifles and shotguns.

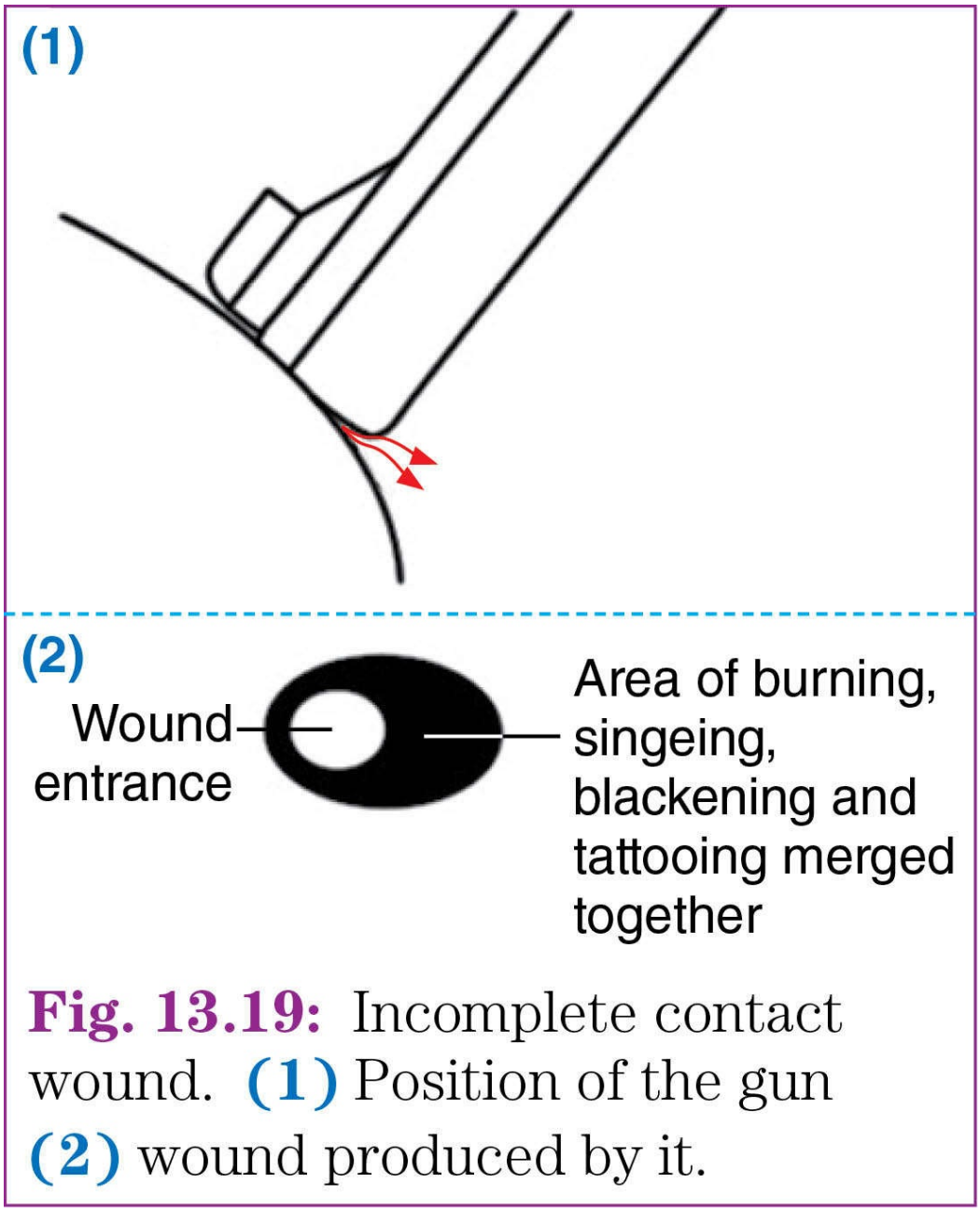


Fig. 13.19: Incomplete contact wound. **(1)** Position of the gun **(2)** wound produced by it.

b. Near contact

(1) **Characteristics:**

(i) Range - The term is applied when the victim is within the range of flame

(ii) Muzzle of the weapon is not in contact with the skin, being held a short distance away.

(iii) The distance, however, is so small that the powder grains emerging from the muzzle do not have a chance to disperse

(iv) Flame effects - Present. There is an entrance wound, surrounded by seared, blackened skin. The zone of searing is wider than that seen in a loose contact wound.

(v) Overlying it is a wide zone of soot

(vi) The soot in the seared zone is baked into the skin and cannot be completely wiped away.

(vii) Small clumps of unburned powder may be present in the seared zones.

(viii) Up to this distance the shot is also called "**point blank**".

(2) **Classification** - Contact wounds may be

(i) straight near contact

(ii) angled near contact.

c. Intermediate

Characteristics:

(1) Range - The term is applied when the victim is within the range of tattooing.

(2) Tattooing - Present. Powder grains expelled from the muzzle produce **tattooing**. These markings are the sine qua non [essential requisite, hallmark] of intermediate-range gunshot wounds.

(3) Blackening – May or may not be present.

d. Distant

Characteristics:

(1) Range - The victim is outside the range of flame, blackening and tattooing

(2) No burning, singeing, blackening or tattooing are seen. The only marks on the body are those produced by the mechanical action of the bullet [entry wound].

B. Wounds from Shotguns

1. Ranges

(1) **Table 4** gives general ranges.

(2) Some provisos are:

(i) Larger the caliber, the greater the distance to which the powder is discharged.

(ii) The cards travel for 2 m

(iii) Wads travel for 2-5 m

(iv) Wads produce minor injury up to a distance of 3 m.

2. Entry wounds

Entry wounds from shotguns depend upon

(1) Distance from which weapon is discharged

(2) Size of the shot

(3) Nature of the explosive

(4) The firearm.

a. Distance

At close range, the shotgun is the most formidable and destructive of all small arms. Discharge from a shotgun produces a long shallow cone of shots. Its apex is at the muzzle of the gun. Farther along the victim is, larger the wound is [**Fig 13.20**].

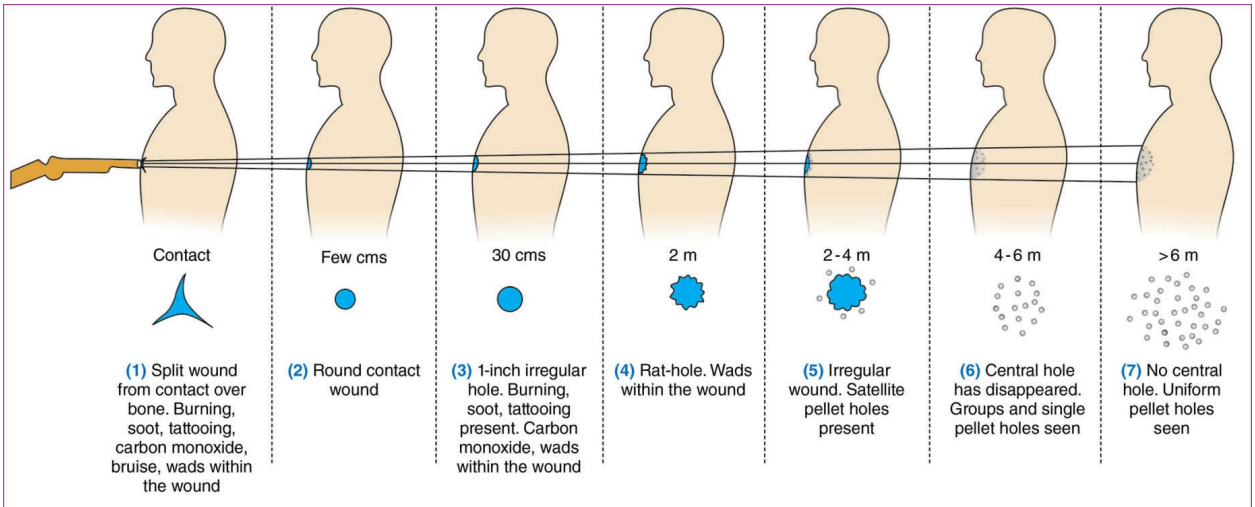

Contact
Few cms
30 cms
2 m
2-4 m
4-6 m
>6 m
(1) Split wound from contact over bone. Burning, soot, tattooing, carbon monoxide, bruise, wads within the wound
(2) Round contact wound
(3) 1-inch irregular hole. Burning, soot, tattooing present. Carbon monoxide, wads within the wound
(4) Rat-hole. Wads within the wound
(5) Irregular wound. Satellite pellet holes present
(6) Central hole has disappeared. Groups and single pellet holes seen
(7) No central hole. Uniform pellet holes seen

Fig. 13.20: Wounds produced by a shotgun at different ranges

i. Hard Contact

Characteristics

(1) **Cruciate laceration** [syn, cruciate wounds, stellate laceration]:
(i) Laceration in the shape of a cross [+ or x].
(ii) Seen especially if there is bone immediately underneath [eg skull].
(iii) Caused by gases entering beneath the skin, and coming out through the same wound [**blowback phenomenon**].
(iv) Sometimes there may be subsidiary linear tears extending from the cruciate wound.
(2) **Bone underneath** – shows
(i) a large irregular hole with fissured fractures running from the margins
(ii) **Crazy-paving fracture** - is sometimes seen in skull. Involves base of skull, roof of orbits and middle ears [**bursting fractures**, **burst head**].
(iii) Burning, blackening and tattooing of bone – In skull wounds frequently soot may enter the inner surface of skull and may blacken it from inside. Dura may also be blackened.
(3) **Soiling and burning** – of the wound minimal or absent, because all material discharged from gun is driven within the wound.
(4) **Track of wound:**
(i) Seared and charred by flame
(ii) Blackened by powder and smoke
(5) **Blast effects** - shattering and destruction of tissue are maximal. Effect .es and range -es.
(6) **Backspatter** [syn, German - Rückschleuderspuren]– It is the ejection of blood, cloth fibres, hair and tissue fragments from the entry wound towards the firearm.

Salient features: (i) **Occurrence and degree** - depends on (a) anatomical location of wound (b) range and (c) caliber of weapon. (ii) **Etiology** [of backspatter]- (a) Relevant for contact wounds - (i) negative pressure in the barrel created immediately after firing (ii) Expansion of gas trapped s/c. (b) Relevant for all ranges -(i) intra-cranial pressure generated by the temporary cavity and (ii) tail splashing - backward streaming of blood and tissue along the lateral surfaces of the bullet. This may represent an early stage of the temporary cavity effect. (iii) Seen in all firearms, but more common with shotguns. (iv) Seen at all ranges, but blood and tissues etc are found within the muzzle of gun only in contact and close ranges. (v) **MLI** - Crime scene reconstruction. It can be found inside the barrel, outside the weapon, on the

person shooting and on persons or objects nearby. Assists in differentiation between suicide and homicide.

(7) **If entry is through a cloth:**

(i) Ammunition hole in the cloth touching the muzzle - may be surrounded by a flat ring corresponding to the outline of the muzzle

(ii) Loose fibres of the cloth - in the center of hole may be turned in and stuck in the wound. Sometimes they may be turned outwards due to pressure of gases returning from the entry wound

(iii) Smoke - (a) may enter within clothing, and may blacken each layer of clothing within, and even skin. (b) If cloth is bloodstained, soot may not be recognizable. IR and UV photography may be helpful. Also examine the cloth from inside, where soot may be recognizable

(iv) Entry hole in cloth - shows singeing. Synthetic clothes may show melting of fibre tips

(v) A ring of burning - may be seen around the wound also.

(8) **Other features** - same as those of loose contact and near contact.

ii. Loose Contact and near contact

(1) **Blast effects:**

(i) Lesser than those seen in hard contact;

(ii) Cruciate wounds are less commonly seen.

(iii) Shotgun wounds up to this range cause much more destruction than rifled weapon due to larger amount of gunpowder producing greater amount of gas.

(2) **Dimensions:**

(i) Single

(ii) Usually round, but may be oval in angled shots

(iii) Equal to the bore in size

(3) **Margins:**

(i) Often ragged [because of individual shots at the periphery] and torn [because of gases]

(ii) Usually inverted, but may be everted due to rebounding gases

(iii) Charring, blackening and tattooing - (a) around the wound and also within the wound. (b) Corona– is seen sometimes. (I) It is a circular zone of soot deposited around the hole with a clean area between it and the hole. (II)

Cause - Due to faster-moving gases near the hole not allowing soot to deposit there. As the effect of gases lessens at greater distances, it is seen only up to near contact range. (III) Seen in all firearms up to near contact range

(iv) Contused [caused by impact of heavier particles of unburnt gun powder]

(v) Color – (a) of entry wound as well as track is pinkish due to CO within the gases combining with Hb to form COHb. (b) Intensity - gradually .es along the track, till it almost disappears near exit. But very rarely it may be present in exit wounds also. (c) COHb may be demonstrable in blood also.

(vi) Muzzle impression [muzzle imprint] – seen around the wound. (a) Causes - It is an impact or imprint abrasion caused by (I) firm mechanical pressure of impact of metal rim against the skin (II) s/c expansion of gases lifting the skin forcibly up against the muzzle from within.[**copy abrasion**, **imprint abrasion**, **recoil abrasion**]. (b) Appearance - Rarely it presents a perfect imprint of the muzzle end, but mostly it is an incomplete, indistinct bruise. (c) Double barreled shotgun – Leaves a tell-tale impression, which may help in identifying the firearm. The impression caused by the unused barrel is often more distinct [**Fig. 13.21**]. (d) If muzzle impression is present, other collars may be absent as all material is driven inside the wound. (e) May be absent due to - (I) explosive damage associated with discharge [because gases have restricted space for expansion; blast effect]. In head wounds, the entire contour of head and face may be destroyed, location of actual entry being virtually impossible. (II) Recoil"Immediate removal of weapon away from wound

(4) **Track of wound** – as in hard contact

(5) **Underlying tissues** – Entry of gases has following effects

(i) s/c tissues - show severe disruption. A pocket may sometimes form containing blood mixed with soot and gunpowder

(ii) Chest wounds – Skin and s/c tissues may be peeled away from the rib cage to form a pocket, which collapses after sometime.

(iii) Abdominal wounds – Coils of intestine may lie outside

(iv) extrusion of soft tissues [eg fat] through the wound [due to a “tail vacuum” produced by rapid entry of gases in the wound].

(6) **Depth of the wound** – Contains

(i) pellets

(ii) **Brightly colored plastic granules** – if they were present as a filler between the shots [please see above]

(iii) felt, wads or plastic cups.

(7) **Bone underneath** – may show fractures as in hard contact, but since gases do not enter wound, blackening and tattooing of bone absent.

(8) **Entry within mouth:**

(i) abrasion or bruising of both lips

(ii) splitting of angle of mouth [due to blast effect]

(9) **If entry is through a cloth** – Same as in hard contact above.

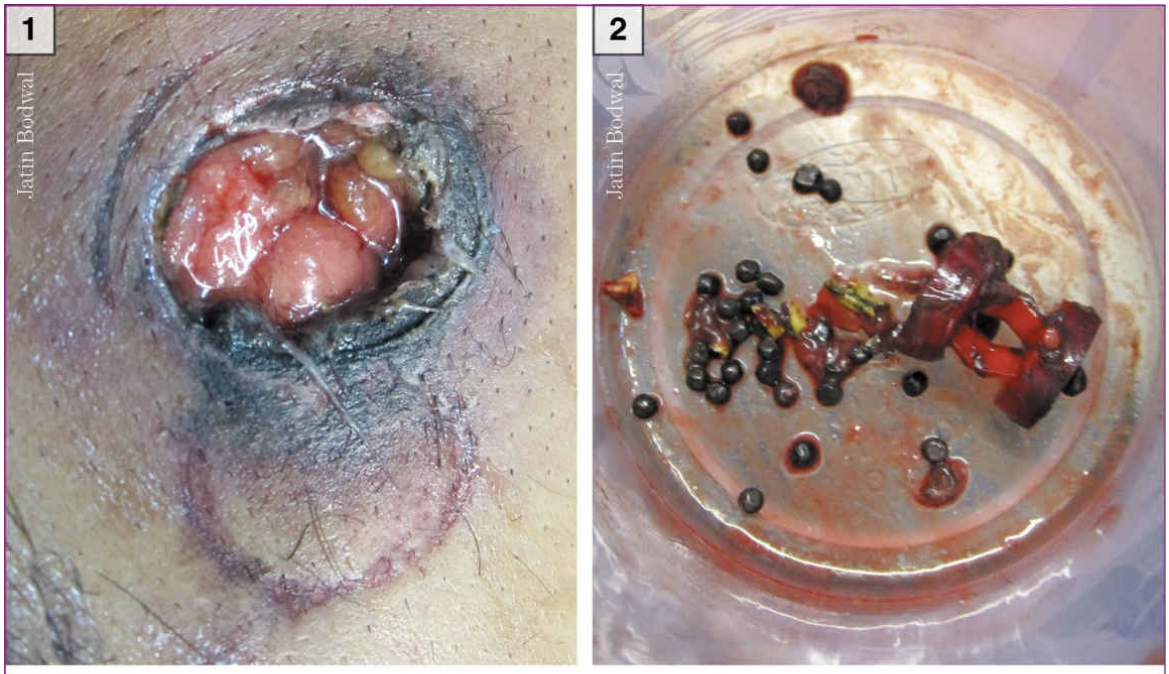


Fig. 13.21: (1) Double barrel muzzle impression seen in a loose contact entry wounds. Please note blackening of margins and of tissues inside. A mild inflammatory ring [contusion collar] is also visible. **(2)** Shotgun pellets, and cushion [card wad] recovered from the depths of wound.

iii. Up to 1 meter

(1) **Wound** – Appearance same as in loose contact and near contact wounds

(2) **Burning, singeing, blackening and tattooing** – around the wound present.

(i) Burning – (a) Wide flare of narrow rim of hyperemia or even blistering caused by flame (b) Unequal burning in angled wounds [**Fig 13.15**]

(ii) Blackening and tattooing – More extensive than in near contact wounds. Intensity .es as distance -es.

(3) **Color** – pinkish, as above. Seen up to a distance of 60 cm.

(4) **If power piston is used:**

(i) It opens up between 30-60 cm. If victim is standing beyond this range, there would be a circular entrance wound, with a typical "flower petal" or "Maltese cross" pattern abrasion around it.

(ii) These petal marks can occur even if the entrance site is covered with clothing.

(iii) By 90 cm, the petals fold back; beyond this range this characteristic abrasion would be absent.

(5) **Depth of the wound** – Same as above. Plastic granules [if present within the shot] may produce very fine punctate abrasions around the pellet holes **[Pseudo-tattooing]**.

iv. 1-2 meters

(1) **Central aperture** – up to 2 m, all shots travel together [en masse] and enter through a single hole. There is a single central aperture of diameter 3-4 cm. As distance -es, the size of hole also -es.

(2) **Distance at which outer shots just begin to separate:**

(i) variable (a) 1 m – with sawed-off gun (b) 2 m – with unchoked guns (c) 4 m – with full choke guns.

(ii) Appearance - Central aperture has jagged margins [scalloping]. The wound appears as if a rat has gnawed at the margins [**rat hole**].

(3) **Up to 2 m** – Cards and wads are found deep within the wound. If found within the wound, bore of gun may be estimated.

v. 2-4 meters

(1) **Just after 2 m:**

(i) Central aperture - irregular

(ii) Individual pellet holes - are seen at the periphery of central aperture. Their entry wound is round and shows a rim of abrasion around their margins

(iii) Cards and wads – strike the body below the shotgun wound and fall off, producing bruises

(2) **3 m** – Central aperture is surrounded by separate openings, in an area of about 8-10 cm in diameter

(3) **As distance -es:**

(i) Diameter of central aperture progressively .es

(ii) Individual pellet wounds -in number

(iii) Occasionally few individual pellet holes may coalesce producing larger entrances than those produced by individual pellets

(4) Examination of individual pellets – recovered from wound would reveal deformation. Cause - lead is softer"rubs against the inside of barrel"deformation. Intense heat inside barrel may cause melting and fusion of several pellets.

vi. >4 meters

(1) **Shots spread widely** - All pellets enter the body separately, producing its own track

(2) **In head shots:**

(i) pellets may not have sufficient energy to enter skull. If at all they enter, they do not enter the entire thickness of the brain

(ii) **Cavitation** – minimal or does not occur

(iii) Infarcts – may be produced by lacerations of blood vessels by pellets or bone fragments.

b. Size of the shot

(1) At close range wounds produced by shots of different sizes are similar

(2) Penetrating power of large shot > small shot

(3) Smaller the shot, more minutely irregular are the edges.

c. Nature of the explosive

Smokeless powder causes less blackening and tattooing than black powder, and it is somewhat grayish white rather than black.

d. The firearm

Appearance of wound depends upon

(1) Bore

(2) Length of barrel – shorter barrels usually produce deposits over larger areas

(3) Presence or absence of choke, and in case of presence, its degree.

3. Exit wounds

(1) **Syn** – In all firearms, they are also known as **outshoot wounds**.

(2) **Generally not present** – due to low kinetic energy [because of .mass and .velocity of pellets]. They exit in following conditions:

(i) Nature of projectile – balling or welding of shot [please see below], large caliber buckshot, rifled slug

(ii) Part of body struck - Thin [neck, extremities]. Occasionally head. However entrance wounds in mouth or nose are not accompanied by exit wounds because of resistance offered by hard palate. In such cases, shots may be seen to lie between the skull and scalp at the back or side of head.

(iii) Manner in which struck - (a) Contact wounds (b) Tangential wounds [where some pellets have a very short track through the body]

(3) **Appearance of exit wounds** [when present]:

(i) Disruption - Greater than in entrance wounds

(ii) Margins – everted [because the unsupported skin is struck from within]

(iii) No burning, singeing, blackening and tattooing

(iv) If the skin is well supported – may show "shored" exit wounds. Please see below.

(4) **In contact wounds of head** – following features are seen:

(i) **Krönlein shot** [syn, Kr***ö***nleinschuss]- (a) In contact wounds, occasionally a large exit wound is produced, and the gases entering through the wound cause the expulsion of entire brain out of the skull, which may be found relatively intact at some distance from the body [evisceratio cerebri sclopetaria totalis]. (b) This phenomenon is only seen in contact wounds, where gases enter inside the wound, and is much more common in high velocity missiles [muzzle velocity [**600–800 m/s**]. (c) In high velocity missiles, temporary cavitation produces an enormous force within the brain, which may also cause expulsion of brain [please see "**cavitation of brain**" above]. (d) May be seen in exit wounds of other firearms also eg pistols, revolvers and rifles. (e) Named after German surgeon **Rudolf Ulrich Krönlein** (1847-1910) who first described it in **1899**.

(ii) **Eyeball** – may be blown out of socket.

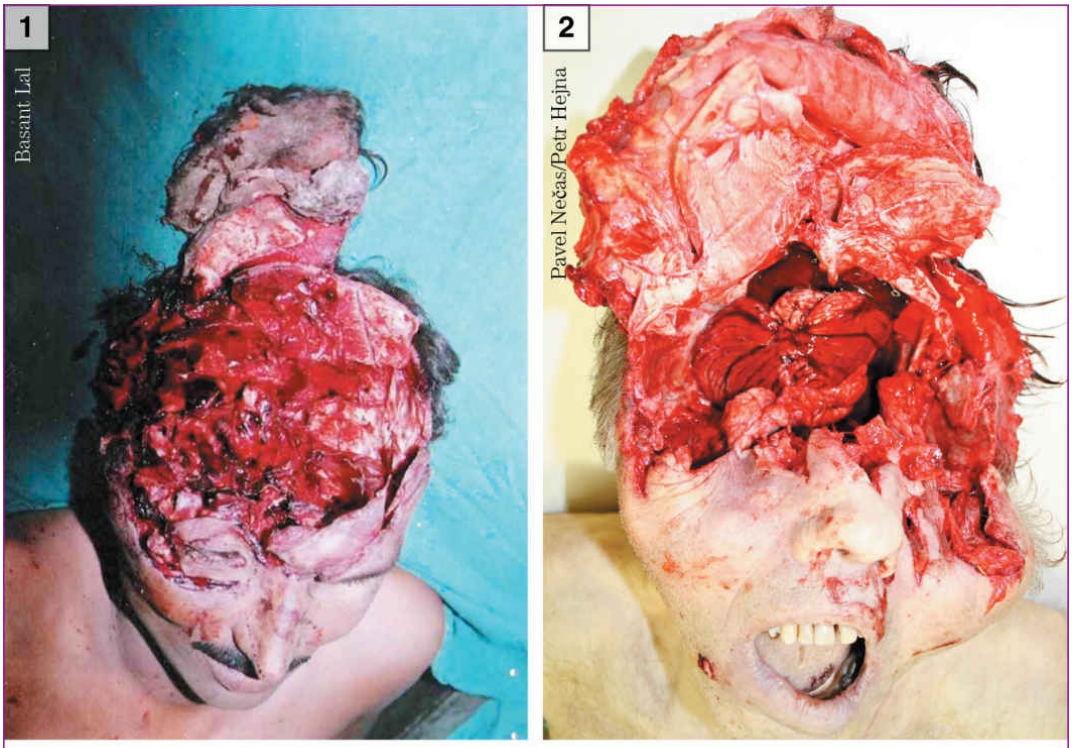


Fig. 13.22: Krönlein shots. Note virtually complete expulsion of brain from both skulls, which have exploded outwards. In 2nd pic, cerebellum may be seen lying within the skull cavity.

4. Special effects

a. Billiard ball ricochet effect

Billiard ball ricochet effect is excessive spreading of shot pellets due to passage through an intermediate target such as a glass window.

b. Balling or welding of shot

Balling [syn, welding] ***of the shot*** is exactly the reverse of billiard ball ricochet effect.

Salient features:

(1) Here the shots stick together and travel for large distances as a single mass. Spread is thus less than what it should be and causes the forensic pathologist to estimate less distance than actual.

C. Wounds from Automatic Pistols and Revolvers

1. Entrance wounds

a. General

(1) **Components of an entrance wound** - from center to the periphery are as follows [**Fig 13.23**].

Memory Aid 4: Zones of rifle firearm entry wound

Mnemonic 1: **W**ild **G**oat **A**te **C**hara - **W**ound defect, **G**rease collar, **A**brasion collar, **C**ontusion collar [from within outwards]

Mnemonic 2: CAGE [from without inwards]. **E** represents **E**ntry wound. Either may be remembered according to comfort level.

(2) **Revolvers and automatic pistols** - cause similar wounds [both entry and exit]. However penetrating power of pistol bullets is much greater because of greater velocity. Pistol bullets are also more often coated with hard metal.

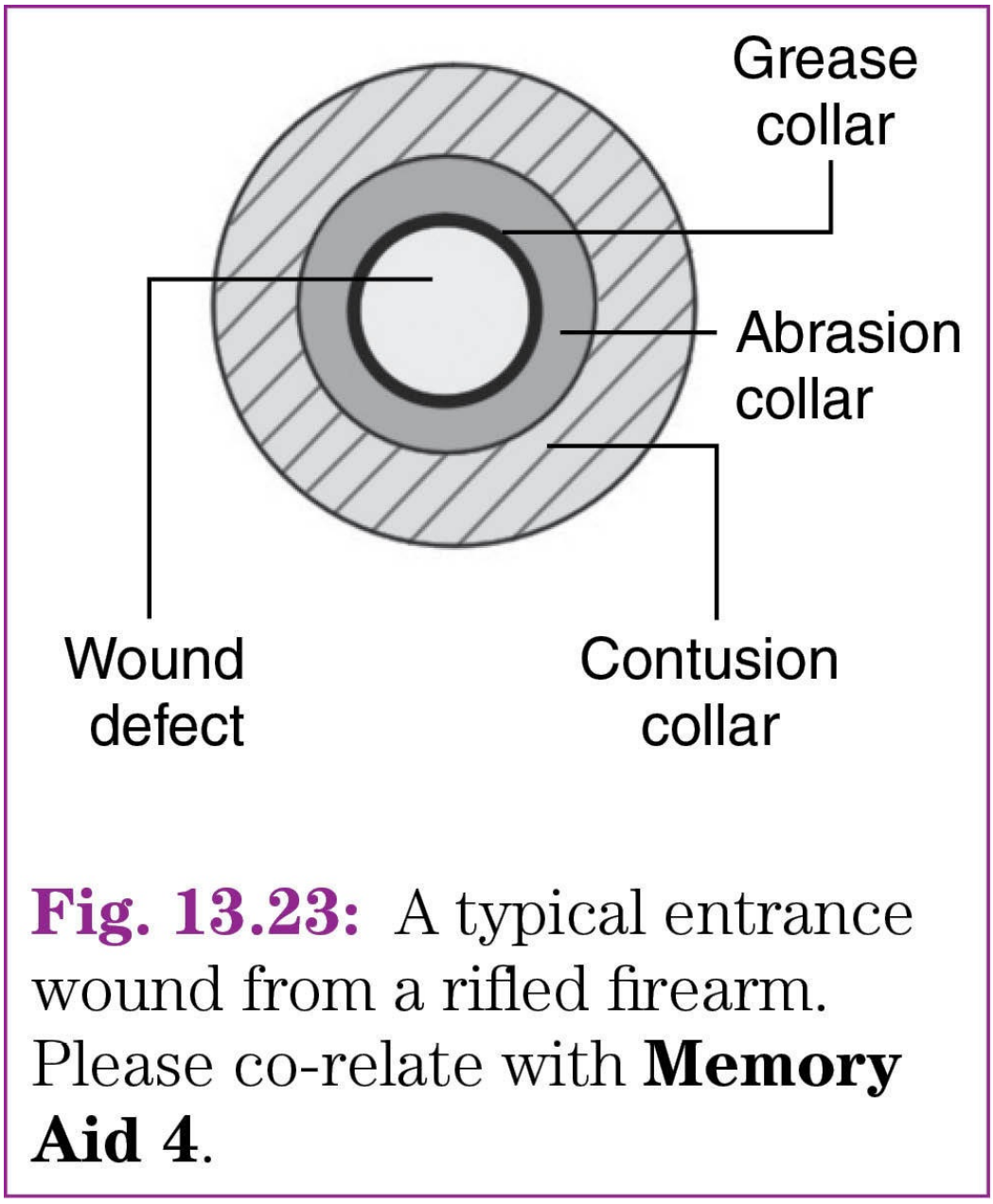


Fig. 13.23: A typical entrance wound from a rifled firearm. Please co-relate with **Memory Aid 4**.

i. Wound defect

This is the actual entrance wound of the bullet

ii. Grease collar

Grease collar [***syn*** – bullet wipe, bullet wipe soiling, burnishing, dirt collar, grease ring, lead ring, leaded edge, metal fouling, metal ring] is a ring of grease seen around the entry wound in the form of a gray coloration. It is present at all distances.

Salient features:

(1) **Cause** – As bullet moves down the barrel, it collects debris and metal from barrel, dirt, gun oil, powder and primer residue, soot etc over it. This is deposited on to the skin, as the bullet penetrates the body. [**Fig 13.8**].

(2) **Less common** – in jacketed bullets

(3) If wounded area is covered by clothing, seen on clothing only [importance of examining clothings]

(4) **Detection:**

(i) Naked eye

(ii) IR photography

(5) **Wiping** – collar produced by grease can be wiped, but not that produced by metal, which enters inside the skin. The term "fouling" is used in two contexts:

(i) fouling of the barrel [please see above], and

(ii) fouling of the entrance wound [as mentioned here].

(6) **MLI** - may link the entrance wound to a weapon because metallic elements from the grease collar may be linked to those from the weapon.

iii. Abrasion collar

Abrasion collar [***syn*** **-** Abrasion ring, marginal abrasion] is a ring of abrasion around the entry wound which is present at all distances.

Salient features:

(1) **Cause:** (i) friction - between the bullet and the epithelium, which occurs as the bullet indents and perforates the skin. (ii) massive temporary overstretching - of the skin adjacent to the bullet perforation (iii) rotational movement of bullet [opinion differs on this cause].

(2) **Width of abrasion ring** - varies with the caliber and type of the firearm

(3) **Appearance:** (i) Color - Reddish at first; becomes reddish brown or dark brown as it dries (ii) concentric or eccentric – depends on the angle of bullet entry [**Fig 13.24**]. (iii) Patterned abrasion collar – Sometimes pattern of fabric may be imprinted on skin if fabric is interposed in between.

(4) **Peeled keratin** – is often seen outer to the abrasion collar. The stratum corneum of skin is raised to form a slightly frayed edge.

(5) **Absent** – when tissues are soft and yielding, eg abdomen, buttocks.

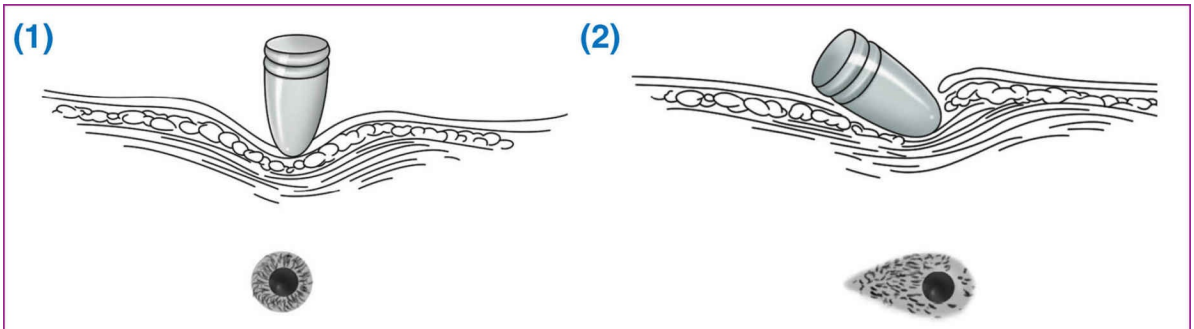


Fig. 13.24: (1) Abrasion collar in a perpendicular shot is concentric; **(2)** that in an oblique shot is eccentric. Pl compare with **Fig 13.15** which shows similar distribution with respect to tattooing.

iv. Contusion collar

Contusion collar [syn, Contusion ring] is a ring of contusion around the entry wound which is present at all distances.

Salient features:

(1) ***Cause*** - damaged blood vessels in the dermis.

(2) Abrasion and contusion collar may often overlap.

b. Hard contact

(1) ***Muzzle impression*** [syn, recoil impression] is seen only in hard contact wounds.

(2) All features same as those seen in shotgun hard contact, eg backspatter, blast effects, fractures etc [please see above].

(3) **If entry is through a cloth** – same features as in shot gun above at this range.

c. Contact shot

The description is true for all 4 variants of contact wounds [please see above]

(1) Discharge from muzzle eg flame, gases, metallic particles, powder and smoke are blown inside the track. Because of this blackening and tattooing are absent or minimal. These features begin when muzzle to target distance >1 cm. **Corona** may be present in loose contact wounds.

(2) **Burning and singeing** - due to flame and hot gases. Can be seen both macroscopically and microscopically.

(3) **Size** - Wound is large; tissues within lacerated; shows cavitation due to gases

entering within

(4) **Shape** – Cruciate, elliptical, stellate or triangular,

(5) **Edges** – Normally inverted, but may be everted if there is bone underneath. Due to gases coming out after entering the wound.

(6) **Abrasion and contusion collar** - present

(7) **Bone underneath, entry through cloth**, **track of wound, underlying tissues** – same as in shotgun wounds at this range.

d. Near contact

(1) All features present in shotguns at this range are also present here, eg backspatter, corona etc

(2) **Entrance wound:**

(i) Shape - circular.

(ii) Edges - inverted. However gases rebounding from within the wound may level them up or even evert the margins.

(iii) Burning and singeing - present

(iv) Surrounding skin - hyperemic. Shows bruising, burning, blackening, tattooing. Abrasion collar, dirt collar and grease collar present.

(v) Surrounding hair - Burnt and singed. Ends clubbed due to intense heat.

(3) **Bone underneath, margins**, t**rack of wound, underlying tissues** – same as in shotgun wounds at this range.

e. Intermediate

(1) Intermediate range in case of pistols and revolvers is up to **45 cm** [**Table 4**, **Table 5**].

(2) **up to 15 cm:**

(i) Burning and lacerating effects of gases present. Lost beyond this range due to dispersion

(ii) Small fragments of metal derived from the interior of barrel or bullet itself are embedded in the skin surrounding the entry wound.

(3) **Entry wound:**

(i) Shape - rounded; slightly smaller than the diameter of the bullet [retraction due to elasticity of skin]. Due to this, caliber of bullet cannot be determined.

(ii) Margins - bruises, inverted, zone of abrasion and grease collar, blackening and tattooing.

f. Distant

(1) **Entry wound:**

(i) Shape - same as in intermediate range.

(ii) Margins - inverted. No burning, blackening or tattooing.

(2) **Special appearances:**

(i) Distant entry wounds of palms and soles - irregular, often having a stellate appearance. There is usually no abrasion ring. Thus they may look like exit wounds.

(ii) Deformity and fragmentation - (a) Presence of a full or partial metal jacket has a major effect on deformity. (b) Bullets with full metal jackets - often remain in one piece and usually do not deform much. (c) Trail - These projectiles typically do not leave a trail of lead fragments along their path. (d) Semi jacketed, hollow-point, nonjacketed, and soft-point bullets - tend to deform on impact or break apart, leaving a telltale trail of metal fragments through the soft tissues. (e) Mushrooming - Hollow-point handgun bullets usually deform by simple **mushrooming** with minimal fragmentation, whereas high-velocity soft-point rifle bullets usually undergo marked fragmentation. (f) ”Lead snowstorm” appearance - This fragmentation of high-velocity bullets creates a **“lead snowstorm” appearance** on radiographs. The area over which the lead snowstorm fragments are deposited in the soft tissues widens as the distance from the entry site -es. Thus, a conical distribution of lead fragments is seen on radiographs, with the apex of the cone pointing toward the entry site. (g) Deformation, fragmentation, cavitation, dissipation of KE and stretching are the major causes of lethal effects of bullets (h) Such deformities and fragmentation may be seen in closer shots also. However as closer shots are more likely to exit, these are described here in distant shots.

g. Skull

i. Beveling

Beveling is the scraping away of bone chips from the unsupported table of skull caused due to intense vibration set up by the bullet.

Salient features:

(1) **Mechanism:** (i) Outer table - As bullet strikes the skull, it sets up intense

vibrations in the skull. Outer table is supported by the inner table, and thus shows a clean punched-in hole. However the inner table is unsupported and bone chips scrape away from it, causing bone chips to come away from it in the shape of a cone. It produces a sloping surface on the inner table [beveling] (ii) Inner table – Situation gets reversed; now outer table is unsupported and shows beveling.

(2) **Bone chips** – from the inner table at the entry may produce lacerations in the dura, and get driven inside the brain forming their own accessory tracks.

(3) **Asymmetry of beveling** – helpful in determining angle of fire. Also corroborated by different levels of entry and exit wounds.

(4) **Exit wound** – is larger due to (i) deformity and (ii) tumbling of bullet

(5) **Radiography** - can reveal beveling of the bone, and thus direction of bullet travel may be determined even in the living by X-raying the skull.

ii. Puppe's rule

Puppe's rule is used to determine sequence of shots when more than one shot have been fired at the cranium. First enunciated by **Georg Puppe** (1867-1925) of Germany.

Salient features:

(1) **Principle** - First bullet will cause certain fracture lines to radiate from the hole it causes [**Fig 13.25**]. The fracture lines emanating from the second bullet hole (b) will stop at the lines caused by the first hole. The fracture lines emanating from the third bullet (c) would stop at the lines caused by first as well as second and so on.

(2) Rule is applicable to multiple instances of blunt force caused by any object, or even multiple objects, eg a bullet first and then an iron rod etc. Also applicable to other flat bones eg sternum, hip bone

(3) Also determines the sequence of shots in a glass window or some other similar object.

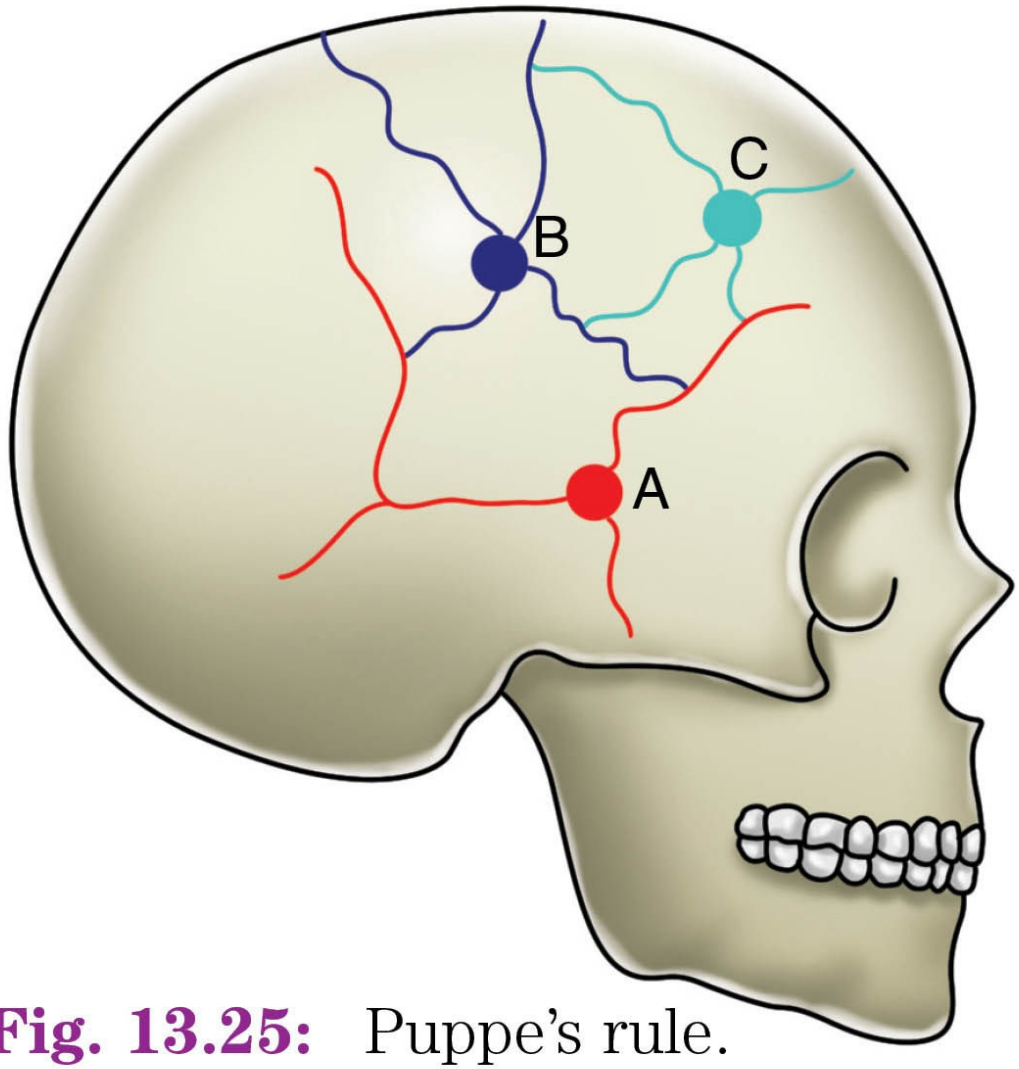


Fig. 13.25: Puppe's rule.
Pl see text for details.

h. Shot through clothing

(1) If entry occurs through clothing, an examination of clothing can reveal range of fire.

(2) **Contact shot:**

(i) clothing shows cross-shaped perforation

(ii) Deeper layers of cloth and skin around bullet hole are blackened

(iii) Pieces of cloth may be carried inside the wound along with the bullet

(3) **Close shot** - Clothing may absorb or filter out all discharge products except the bullet.

(4) **At all distances** - if a lead of full metal jacketed bullet is used, a gray to black rim is produced on the clothing [**bullet wipe**]. It is the equivalent of **grease collar** seen in unclothed entry wounds [please see above], and caused due to same reasons.

i. MLI [of entrance wounds from pistols and revolvers]

(1) **Caliber Determination from Entrance Wounds:**

(i) in skin – Not possible due to elasticity [small wound than bullet], tumbling and explosive effects [larger wound than bullet].

(ii) in bone – possible to some extent. A small bullet may produce a larger wound [explosive effect, tumbling], but not vice-versa. Bone does have some elasticity though; thus a 9-mm bullet may produce a 8.5-mm defect, but not much smaller.

2. Exit wounds

(1) **Shape** – Extremely variable. They may be crescentic, cruciate, elliptical, round, slit like or stellate [star shaped].

(i) Slit like wounds - are produced by (a) Body factors - (I) folding skin [axilla, umbilical area, groin, buttock crease] (b) Bullet factors - (I) a bullet exiting sideways or a (II) tumbling bullet. May resemble a stab, incised or lacerated wound

(ii) Stellate wounds – seen in exit wounds of head. May resemble contact entrance wounds.

(2) **Size:**

(i) Usually bigger - than entry

(ii) Both entry and exit may be of same size - with high velocity bullets

(iii) Exit is smaller than entry in some exception cases - (a) breaking of bullet - within the wound, with only a portion exiting (b) bullet entering through folded or creased skin (c) contact wounds [due to tearing of soft tissues by inrushing gases] (d) Tangential entry - with focal avulsion of tissues (e) Tumbling or yawing of bullet

(3) **Large variation in shape and size is due to:**

(i) Deformation of bullet

(ii) Tumbling of bullet

(iii) Breaking up of bullet in the tissues and exiting as several pieces. If jacketed,

the jacket may separate completely or in part.

(iv) The bullet may contact bone and/or cartilage, and their fragments may be blown out of the body with the bullet.

(v) unsupported skin at exit tends to shatter and tear. The resulting defect may bear little relationship in shape to the missile that caused that defect.

(4) **Edges:**

(i) Everted, puckered, torn. Eversion of wound often differentiates exit from entry wound. However both entry and exit wounds may be everted in (a) **Fatty persons** [due to protrusion of fat] (b) **Putrefaction** [due to protrusion of gases].

(ii) No burning, singeing, blackening, tattooing; no abrasion or contusion collar [exception - shored exit]

(iii) Pieces of contused hemorrhagic s/c fat may protrude through the defect.

(5) **Thermal artifacts** – A bullet is hot when it enters the body and still so at the time of exit. Rarely burning as a thermal artifact may be seen at the site of bullet exit as at the site of entrance. However blast effect of muzzle gas would be absent.

(6) **Exit wounds may not be seen:**

(i) in distant shots

(ii) If bullet fragments within body [Frangible bullets; please see above].

(7) **Differentiation from entrance wounds:**

(i) May not be easy always, and may look alike.

(ii) Reliance must not be made on information from police, victims, or onsite witnesses as it may be false or inaccurate.

(iii) Helps in determining (a) direction of fire (b) posture of victim at the time of fire (c) number of bullets in the body – Can be determined by the "**Odd and even rule**". The rule states that if the total number of bullet holes (both entrance and exit) is odd (or even), then the number of bullets remaining in the patient is odd (or even) as well [**Table 6**].

(iv) **Table 7** lists some imp differences.

Table 6: Odd and even rule

Bullet holes	Bullets in the body
Odd	Odd
Even	Even

(8) **Shored [syn, supported] exit wounds:** Exit wounds shored or supported by a firm material or object pressed against the skin.

Table 7: Difference between entry and exit wounds of a rifled firearm

S.No	Feature	Entry wound	Exit wound
1.	Size	Smaller than the diameter of bullet [except contact shot]	Bigger
2.	Edges	Inverted	Everted, puckered, torn
3.	Burning, blackening, tattooing	May be seen around the wound	Never seen
4.	Grease, abrasion and contusion collar	Present	Absent
5.	Bleeding	Less	More
6.	Protrusion of fat	No protrusion	May be protruded
7.	Wound track	May be cherry red [due to CO in explosive gases]	No such color
8.	Fibres of clothing	Turned in and may be carried into the wound. Blackening may be seen	Turned out. No blackening
9.	Radiological/ microchemical examination of wound	Materials present in grease collar [metal from barrel etc] may be detected. In case of unjacketed bullets, lead on outside of wound.	No such material detected. In unjacketed bullets, Pb may be detected on the inside of the wound rather than outside.
10.	Spectrography	Same as above, but detected by spectrography	Same as above
11.	Skull	Punched out clean cut hole on the outer table; beveling on inner table	Punched out clean cut hole on the inner table; beveling on outer table
12.	Histopathology	(i) Near discharge shows signs of burning and singeing, deposition of soot and unburnt powder (ii) swelling and homogenization of dermal collagen caused by heat of bullet is more marked	(i) No such deposition (ii) Swelling and homogenization less marked

D. Wounds from Rifles

(1) **Appearance** - Wounds are similar to those produced by revolvers and

pistols, but produce more damage

(2) **Burning, blackening, tattooing** - **Table 4**.

1. Contact wounds

a. Contact wounds of head

Extent of damage - These are most devastating wounds, producing a bursting rupture of the head.

b. Rifle discharge in mouth

1. Massive wounds from the gas and the temporary cavity occur.
2. Lacerations of the corners of mouth, nasolabial folds, medial to the eyes, at the bridge of the nose and along the nasal ridge.

c. Contact wounds of the chest and abdomen

External appearances

The wound of entrance is typically circular in shape and usually larger in diameter than those due to pistol bullets. The wounds do not have the peculiar explosive appearance of similar wounds in the head. There is almost never tearing of the skin due to gas.

2. Intermediate-range wounds

Severity of injuries: Both intermediate range and distant head wounds, show a wide range in the degree of severity, depending on the type of bullet and the entrance site in the head.

3. Distant wounds

Entry wound is usually smaller than the diameter of bullet; appears as if it was made by forcing a lead pencil into skin.

4. Exit wounds

(1) At all ranges, exit wounds of the chest and abdomen have the same appearance. They are larger and more irregular than the entrance wounds, with the majority of exit wounds ≤25 mm in diameter.

(2) If the bullet strikes a bone, extensive shattering and communion takes place. In such cases, exit may be as large as the palm of the hand.

(3) Sometimes several small holes are found around the large exit. These are caused by fragments of bone being driven out.

E. X-ray Examination of Gunshot Wound Victims

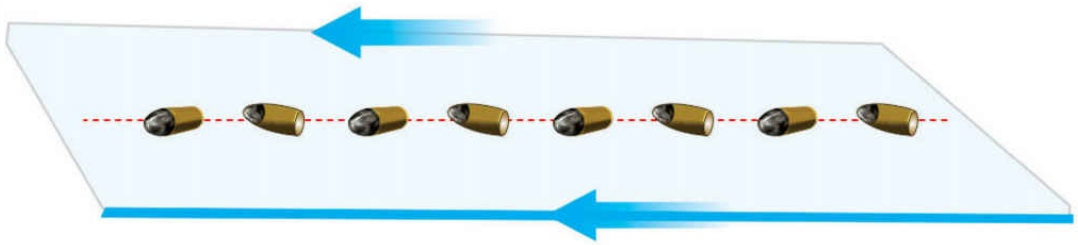
X-ray helps to

(1) determine defects, fractures etc
(2) determine the break-up pattern of the bullet
(3) locate air embolism accompanying large vessel damage by the missile
(4) locate the bullet, bullet fragments, jackets, pellets etc
(5) show the track of bullet, demonstration of ricochet etc [a radiopaque dye may be instilled in the track before taking X-ray]
(6) type of ammunition used [caliber etc].

F. Peculiar Effects of Firearms

1. Artifacts

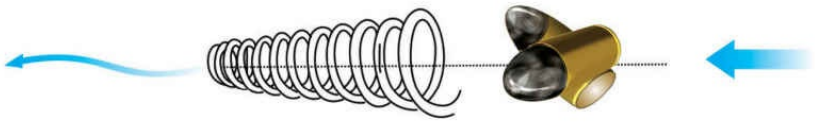
(1) **Kennedy phenomenon** - Name given to problems in interpretation of wounds caused by surgical alteration or suturing of gunshot wounds.
(2) **Rayalaseema phenomenon** – Victim first stabbed " Then to mislead the police, a bullet is stuffed into the stab wound. Once common in Rayalaseema district of AP.



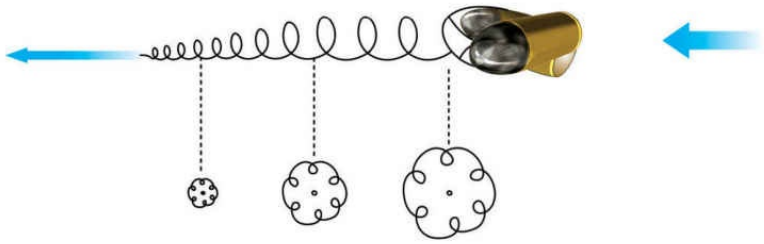
(1) Yaw is motion of bullet sideways

(2) Pitch is motion of bullet up and down

(3) Tumbling is the action of forward rotation around the center of mass. It results in tumbling over of the bullet



(4) Precession is a circular yawing around the center of mass in a spiral fashion



(5) Nutation is the rotational movement in small circles forward in a rosette pattern

Fig. 13.26: (1) yaw (2) pitch (3) tumble (4) precession and (5) nutation. Notice that both precession and nutation decrease as distance from the barrel increases.

2. Atypical entrance wounds

a. Ricochet bullet

A ***ricochet bullet*** is one which before striking an object strikes some intervening

object first, and then after ricocheting and rebounding from the intervening object, hits the object.

Salient features:

(1) **Types of ricochet** – (a) **External ricochet** – Bullet rebounds off some external object (eg roof) (b) **Internal ricochet** – Bullet rebounds off some internal body surface (eg inside of skull, chest)

(2) **Occurs rarely** – Most of the times the bullet either breaks up on striking intervening object, or penetrate it.

(3) Occurs with **inferior firearms** and **low velocity bullets**

(4) **Critical angle** – 10°-30°.

b. Bullet graze or slap

Bullet strikes at such an acute angle, that instead of entering the body, it simply causes an abrasion. It may appear as an elongated, elliptical or triangular reddish brown abrasion. Underlying dermis may or may not be involved. Adjacent skin should be examined; it may show ecchymosis.

c. Peculiar flight characteristics of bullet

i. Yaw, pitch, tumble, precession, and nutation

ii. Tail wobble or tail wag

(1) The tail of the bullet wobbles for a few microseconds after it leaves the muzzle [50 meters for pistol; 150 meters for rifle]

(2) Produces atypical entrance wounds and causes greater tissue damage at these ranges.

3. Bullet embolism

If bullets or pellets enter a large vessel, they move along the blood flow and become embolized at a distant part.

4. Concealed firearm wounds

Entrance wounds may be hard to locate if present inside the ear, mouth, nostrils, axilla, anus or vagina. If bullet is found inside the body and no entry wound may be located, search should be made of such areas.

5. Entrance-exit anomalies

a. Single entrance and multiple exits

Occurs in following circumstances:

(1) Bone acting as secondary missile – bullet strikes bone" chips off a piece or more of bone"Bone pieces act as secondary missiles and exit through separate tracks. Such a bullet may also split producing further exits.

(2) Jacket acting as missile – Jacketed bullet strikes victim"jacket separates within body and exits separately from bullet. Part of the jacket may sometime remain within the body. In such cases despite multiple exits, radiologically jacket would be visible inside, albeit part of it only.

(3) Splitting - Bullets splits up within the body in 3-4 pieces and each piece comes out through a separate track. Happens especially when it strikes bone.

b. Multiple entrances and exits from a single shot

(1) **Situations when this occurs:**

(i) the victim was running or sitting in an unusual position

(ii) Bullet hit irregular surface of the body eg breast, buttocks

(iii) bullet struck victim in such an orientation that several parts of the body came in its line successively.

(2) **Investigation:** Examination of clothing [or skin, if an unclothed portion was hit] for burning, blackening and tattooing. These phenomena, if present, would be seen only in the 1st entrance wound, only wound 1 would show these phenomena. Other entry wounds, eg 3 and 5 would resemble wounds caused by distant fire].

(3) **Number of bullets present in the body** - Please see “odd and even rule” above.

c. Entrance wound is present; no exit wound but bullet not in the body

(1) This occurs when bullets exit through natural orifices. **Ex** (i) Bullet enters mouth [eg through a cheek wound]"spit out (ii) Bullet enters nose [eg through a face wound]"sneezed out (iii) Bullet enters stomach"vomited (iv) Bullet enters trachea"coughed out (v) Bullet enters intestine or colon"defecated (vi) Bullet enters uterus"passed out through vagina (vii) Bullet strikes a bone at right angle"ricochets back and comes out through the same wound through which it entered.

(2) Sometimes such bullets are found unaccountably on the stretcher. This should warn the surgeon [or pathologist] of the possibility of bullet’s exit through one of the natural orifices.

(3) This is an exception to “Odd and even rule” [please see above].

d. One entrance, one exit; bullet within the body

May occur occasionally when the area around exit was heavily clothed, or victim’s body was juxtaposed against a hard object [eg cement floor]. Bullet enters "attempts to exit"makes an exit wound, but returns to the body, as it

encounters the hard object. Causes a shored exit wound. The hard object at the exit site must be examined for damage.

6. Fetal firearm wounds

Gunshot wounds of the pregnant uterus are relatively uncommon. Maternal death in such cases is rare. The gunshot injury to the fetus or placenta usually results in IUD or premature delivery with or without evidence of injury to the child. If fetus was "quick" and death occurs, assailant would be tried **u/s316, IPC** [**ch 26**].

7. Firearm going off by itself

If there is some defect in the mechanism, the firearm may discharge by any mechanical pressure applied anywhere on the gun. Country made firearms and old worn out guns may discharge on being pushed, or thrown or by being dropped on the ground.

8. Souvenir bullets

A ***souvenir bullet*** is one which has been lodged in the body for a long time without causing ill effects.

Salient features:

(1) **Location** - Lies usually in an inaccessible place, eg, near or inside a vertebra and the surgeon considers it best to leave the bullet alone

(2) **Souvenir pellets** – from shotgun. Individual pellets may be tedious to extract, so the surgeon may decide against removing them

(3) **Scar** – indicates the original entrance wound

(4) **Dense fibrous tissue** – forms around the bullet [encapsulation]

(5) **Chronic lead poisoning** – may occur over the years, as lead gradually absorbs from the bullet or pellets [**ch 36**]

(6) **Synovial fluid** – in the joints around bullet location may display -Pb.

(7) **Souvenir bullet as an artifact** - It is not the cause of death. An inexperienced pathologist may however label it so [**ch 10**].

9. Unexplained bullets in the body

Major rules explaining number of bullets inside body:

(1) Rule 1 - Odd and even rule [please see above]

(2) Rule 2 - No. of bullets in body = no. of entry wounds – no. of exit wounds. If the number of bullets in the body are different [either more or less] than the number of “entrance-exit” wounds, the absence or presence of some bullets has to be explained. This happens mostly in case of **tandem bullet** or **piggyback bullet**.

Tandem bullet

Tandem bullet is a pair of bullets going off by a single fire due to a defective firearm.

Salient features:

(1) **Mechanism:** (i) When Old, rusty, unused for several years and worn out weapon is used – (a) It may not provide perfect obturation to the bullet. Bullet travels a little distance within the barrel and sticks somewhere within the barrel [**lodged bullet**] (b) When a second bullet is fired [**striking bullet**], the first bullet [lodged bullet], being firmly stuck within the barrel provides perfect obturation and both bullets move out together. The two bullets together are called the ***tandem bullet***. (ii) When a mismatched cartridge is used – Generally happens when a smaller cartridge is loaded in a larger gun.

(2) **Trajectory:** (i) Tandem bullets tend to follow the same trajectory, but only for a short distance because the two together form an unstable projectile. (ii) The bullets are thereafter separated and follow different trajectories. (iii) If the tandem bullets hit the target before separation, a single hole of entry may be observed. (iv) If the bullets separate before they hit the target two entry holes on the same or on different targets may be observed. (v) The phenomenon of two separate regular entry holes by a single effective firing can be established if the recovered bullets are identified as tandem bullets.

(3) **Peculiarities:** (i) 1 entrance; 2 exit – If the target is near, tandem bullets would enter through a single entry wound; then separate inside the body and come out of different exit wounds. (ii) 1 entrance, 1 exit, 1 bullet inside the body – Rule 2 above is flouted. Situation explained like this"Tandem bullets entered through single entrance wound"Separated inside body"one exited and one remained inside body.

(4) **Imp** – Tandem bullet is different than ***tandem*** or ***duplex cartridges***; in the latter, the cartridge itself contains 2 bullets – one behind the other. They come out in separate lines and enter through two different entry wounds.

V. MEDICOLEGAL QUESTIONS

A. Is the Injury Caused by Discharged of a Firearm?

(1) Around wound look for: (i) muzzle impression (ii) abrasion (iii) contusion and (iv) grease collars (v) blackening and (vi) tattooing.
(2) Presence of bullets and pellets in body.
(3) Corresponding cuts in clothes
(4) entrances and exits
(5) track
(6) presence of (i) cards (ii) wads (iii) shotgun fillers (iv) metal jackets and (v) other residual matter associated with firearms.

B. Can Firearms Produce Lacerated Wounds?

Yes, by pistol whipping [buffaloing]. Please see **ch 12** for details.

C. What Kind of Weapon Fired the Shot?

1. Examination of cartridge, wads, ammunition etc

Cartridges are retrieved from crime scene. Wads and ammunition may be retrieved either from crime scene or from the wound. They can reveal the type of gun.

(1) **Characteristic cartridges and ammunition** - reveal the type of gun they were fired from
(2) **If wad is found to be a plug of paper, cotton or cloth** - gun should be a muzzle loader
(3) **If wad is more formally shaped and circular discs of felt or cardboard are also found** - it should be a breech loader. From the diameter of discs and wads, bore of the gun can also be determined.
(4) **Characteristic stains around clothes or skin** – can reveal whether black or smokeless powder was used.
(5) **Chemical and microscopic examination of stain** – reveals the particular brand of gunpowder used.

2. Examination of bullet

(1) Class and individual characteristics of the **crime bullet** are compared with those of a **test bullet** [please see above].

(2) Class characteristics are same in all guns of a certain make and model. Thus if class characteristics of crime and test bullet are matched, one can tell the make and model of the gun.

(3) **Class characteristics** are:

(i) Caliber

(ii) Primary markings - (a) Number of lands and grooves (b) direction of twist of rifling [left or right] (c) rate of twist (d) width of lands and grooves

(4) **Individual characteristics** are characteristic of a particular gun. They are most important in pinpointing the exact weapon from which the bullet was fired. That is why they are also known as **fingerprints of a firearm**.

(5) **Comparison microscope:**

(i) A comparison microscope [**Fig 13.27**] is essentially two microscopes joined together with a single eyepiece. This permits simultaneous observation of half the view of each microscope, side by side in the eyepiece.

(ii) A line in the center of the field of view separates the views of the two microscopes.

(iii) If two bullets [or cartridge cases] can be brought into position under the respective objectives so that their individual markings appear to flow from one to the other at the dividing line, an identification is established.

(6) **Procedure of comparison:**

(i) When a suspect gun is presented to the ballistic expert along with the crime bullet, he test fires a bullet from the suspect gun into a bag of rags, a box filled with cotton waste [bullet traps], oiled sawdust, water tanks [bullet recovery tanks] or some other similar non abrasive material, which does not leave its own markings.

(ii) Test bullet is then compared with the crime bullet under a comparison microscope. If each striation matches faithfully, the suspect gun is the actual crime gun [**bullet fingerprinting**].

(7) **Extraction of bullet at autopsy** – Since comparison of each and every single striation is important, the surgeon or the autopsy pathologist must extract the bullet from body by cotton or rubber tipped forceps, or preferably with bare hands, but never with needle, toothed forceps or scissors, which can leave their own markings on the bullet, rendering the comparison useless.

(8) **Other marks on bullets** – A lead bullet may often carry weave pattern of the cloth on it. Such a bullet found at the scene of crime can give away the type of cloth victim was wearing, even if his body has been disposed of.

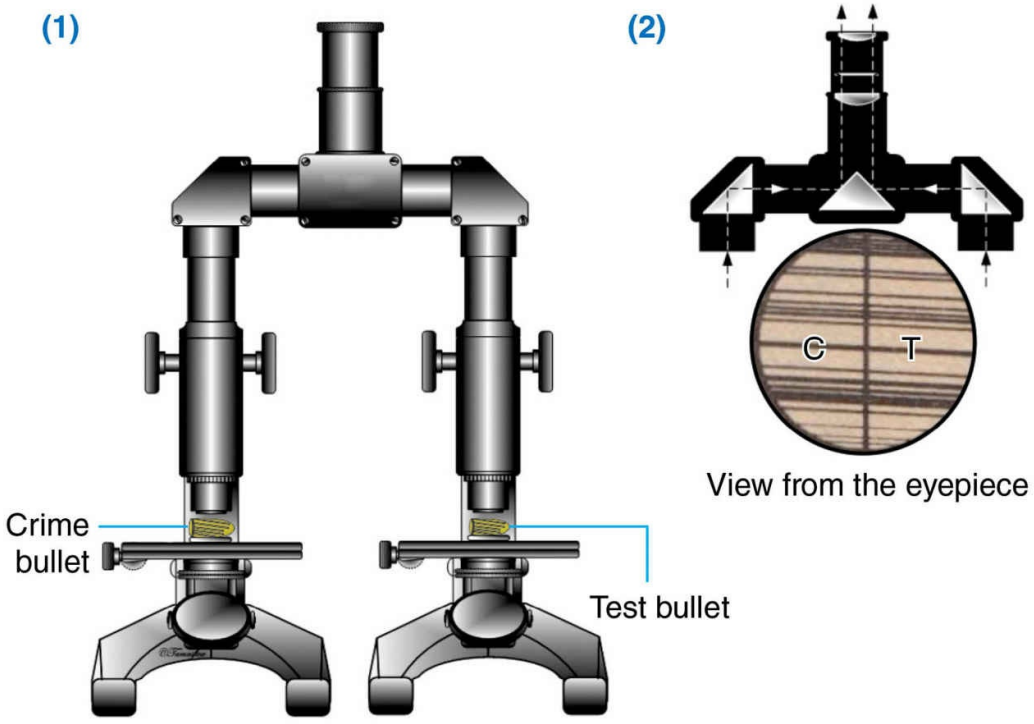


Fig. 13.27: (1) Comparison microscope to compare crime and test bullets **(2)** All striations in crime bullet 'C' match perfectly with those in the test bullet 'T' indicating that the suspect weapon was indeed used in the crime.

3. Examination of cartridges

(1) These may be found at the crime scene in cases of pistols and rifles.

(2) **Identification criteria:**

(i) Shape and size of case; manufacturer's stampings on the case head – reveal caliber, type and manufacturer of cartridge

(ii) Markings on cartridges – [please see above under the heading "other markings"] can reveal the make and model of gun.

(iii) Comparison of markings – between crime cartridge and test cartridge [as in the case of bullets] can identify the exact gun from which a particular cartridge was fired.

D. From what Distance and Direction was the Shot Fired?

(1) **Range of fire at short ranges** - determined by presence of burning, singeing, blackening, tattooing etc.

(2) **At greater ranges, when these signs are absent:**

(i) look for spread of pellets and

(ii) test fire in case of shotgun [quite accurate] and

(iii) nature of wounds and penetration in rifled firearms [only an inspired guess]

(3) **Direction** – Look for entry and exit wounds [**Table 7**] and the intervening track. Possibility of bullet deflection and movements of body parts must be kept in account.

E. Which Person Did the Bullet Hit?

A bullet found at a scene may be linked to the specific individual through which the bullet had passed by examining tissue deposited on the bullet. This is possible even if no tissue is visible with naked eye. The tissue can be removed by swabbing the bullet and a **DNA profile** prepared [**ch 29**]. This procedure is useful if the criminal has removed the body, but forgot to remove the bullet which was hidden in grass etc.

F. Did the Suspect Fire the Gun?

This is determined by detecting **gunshot residue** (GSR) [also known as firearm discharge residue (FDR) or cartridge discharge residue (CDR)] over the hands of the suspect. **GSR**

Salient features:

(1) **Distance traveled by GSR** - Can travel out from the gun to distances of 3–5 feet or even farther. At the farthest distance, only a few trace particles may be present. Thus even if a person was standing in the vicinity of firearm discharge, GSR can be detected on his clothes, in his nose, hair etc.

(2) **Composition** - Gunshot residues are composed of (a) unburned and partially burnt propellant, (b) particles from primer, (c) smoke, (d) grease, (e) lubricants and (f) metal particles from the cartridge as well as the weapon.

(3) **Organic and inorganic GSR** – [A] Organic GSR [OGSR] consists of nitrocellulose, nitroglycerine, nitroguanidine, hydrocarbons, methyl and ethyl

centralite and many other diverse compounds and originates from (a) propellant (b) firearm lubricants and (c) products of their combustion. [B]Inorganic GSR [IGSR] consists of nitrates, nitrites, and metallic particles and originates from (a) the primer, (b) propellant, (c) cartridge case, (d) projectile jacket, (e) its core and (f) weapon barrel.

(4) **Size** – Diameter of GSR particles ranges from **0.5µ to 10µ**.

(5) **Mechanism of formation** - GSR escapes from weapon openings in vaporized form and solidifies into particulate matter on coming in contact with environment (suspect's hands, face, hair, clothes, around wound of entrance etc).

(6) The detection is done by following techniques:

(i) **Color Tests** – (a) **Paraffin test** - Also known as the **dermal nitrate test**, the **diphenylamine test**, or the **Gonzales test** [developed in 1931 by Teodoro Gonzales]. [A] **Technique:** (1) Molten paraffin wax is applied to the back of suspect's hands with a brush (2) Wax solidifies on cooling. It is then peeled off the hands (3) The surface of this wax cast is treated with **diphenylamine/sulfuric acid** reagent by spraying. (4) **Blue color** is produced if nitrites and nitrates are present (important constituents of GSR). [B] **Fallacies** – False +ve results are obtained with several common substances eg certain paints, pharmaceuticals, fertilizers, fingernail polish, leguminous plants, tobacco, tobacco ash and urine. The test is obsolete now. (b) **Harrison and Gilroy test** - Detects metal-containing components of GSR, eg lead, antimony, and barium, originating from the primer and the bullet. [A] **Technique:** (1) Suspect's hands are swabbed with cotton cloth dampened with 0.1 M HCl. (2) The swab is allowed to dry and is then tested with 1-2 drops of a 10% alcohol solution of **triphenylmethylarsonium iodide**. (3) The appearance of an **orange ring** indicates the presence of **antimony**. (c) **Griess Test** [pronounced grease]– Detects nitrites in GSR. (ii) **Anodic Stripping Voltammetry** (**ASV**) (iii) **Atomic Absorption Spectroscopy (AAS) and Flameless Atomic Absorption Spectroscopy (FAAS)** (iv) **Scanning Electron Microscope-Energy Dispersive X-ray** (**SEM-EDX**) (v) **Laser-induced breakdown spectroscopy** (LIBS).

G. Is a Given Hole in a Cloth Produced by the Passage of a Bullet

Through it?

In criminal investigations it is often necessary to determine whether or not a given hole or mark [in car, cloth, door, skin, wall etc] has been produced by a bullet. If a bullet passes through an object some of its material [cobalt, copper, lead, nickel] is rubbed off at the periphery of the hole [**bullet hole peripheries** (BHP)]. This material can be detected by AAS, NAA, and SEM-EDX [already described above]. Additionally following color tests may be used [collection techniques same as for GSR].

(1) **Sodium Rhodizonate test** – in presence of lead"reddish violet color develops.

(2) **Dithiooxamide Test** [DTO, also known as **rubeanic acid**]- in presence of

(i) copper"green-black color develops.

(ii) Cobalt"brown

(iii) nickel"blue. The copper test is more sensitive than the test for cobalt and nickel.

H. How Long Did the Victim Survive?

Depends upon the extent of injuries

(1) **Vital organs involved** - If multiple wounds are there in vital organs"Death could be immediate

(2) **Vital organs not involved** - If vital organs [i.e. brain, heart and lungs] were not involved and death occurred due to hemorrhage and shock"Death may take place after few hours.

(3) **Infection** - If infection is present"Death must have occurred more than 36 h after infliction of injury.

I. Suicide, Accident or Homicide?

Table 8 gives major differences between suicidal, accidental and homicidal wounds. Other important points are:

a. Nature of the entrance wound

1. Victim characteristics

(1) **Contact wound:** likely to be suicidal, unless found on inaccessible parts eg back of neck, chest or abdomen

(2) **Distant shot** – homicidal or accidental, but almost never suicidal.

(3) **No corresponding hole in overlying clothing** – likely to be suicidal, as suicides normally pull clothes away to expose skin.

b. The position and direction of the wound

[A] Position - **Table 8** [B] **Direction** - (1) **vertically downwards** – as over the top of skull would indicate homicide (2) **Obliquely downwards and backwards** – over the chest may indicate suicide, because it is difficult for a suicide to maintain a completely horizontal line over the chest.

Table 8: Differences between suicidal, accidental and homicidal firearm wounds

S.No	Features	Suicide	Accident	homicide
1.	Sex	Usually males	Usually males	Any sex
2.	Profession	Likely to be familiar with guns. More common in professions who have access to firearms, eg police and military personnel, hobbyists	Likely to be familiar with guns [eg if accident was due to cleaning of guns].	Not necessary
3.	Site of entrance wound	Head (temple, Center of forehead, under the chin, inside mouth), left side of chest. Rarely epigastrium	Any area	Generally head, but may be any other vital area
4.	Distance	Contact or very close range. Distant range excludes suicide	Close range	Any range
5.	Direction	Upwards and backwards	Any direction	Any direction
6.	Number of wounds	Usually one	One	Any number
7.	Hands of victim	Will display powder residue	(i) Will display residue if gun was in victim's hands when it accidentally went off (ii) if gun went off due to other reasons, eg, by dropping a defective gun, there will be no powder residues over hands	Will not display powder residue
8.	Cadaveric spasm	May be Present	May be present	Absent
9.	Weapon	(i) May be firmly grasped in hands [virtually confirms suicide] (ii) If not in hands, will be present near the body.	Present at the scene	Absent
10.	Scene	(i) Usually own house (ii) room locked from inside (iii) suicide note may be present (iv) No evidence of struggle	May be indoors (eg if cleaning the gun) or outdoors (eg if hunting)	(i) Any place (ii) if own house, room will never be locked from inside (iii) Absence of suicide note (iv) evidence of struggle apparent [disturbed furniture, rumpled bed sheets, carpets, upturned vases etc]
11.	Hesitation shots	May be present	Absent	Absent
12.	Motive	May have been depressed for some reason (financial loss, failed love affair, death of a loved one, incurable illness). Rarely insane persons may commit suicide with guns	No motive	Revenge, robbery, gang wars
13.	Behavior just before death	Victim indifferent to family or may start showing excessive love towards them. May start giving away valuable possessions to relatives, friends	No such behavior	Victim may display signs of worry due to pressure from enemy (eg for returning loan etc)

c. The number of wounds

(1) **Single wound** – eg on temple may indicate suicide. Other factors need to be taken into account also. For instance if the weapon is missing from the crime scene, even a single wound may indicate homicide. Cases have been described when a suicide shot himself and then threw the weapon outside the window to make it appear like a homicide.

(2) **Multiple wounds** – Especially over vital regions such as brain and heart would strongly favor homicide. A projectile passing through the heart or brain would immediately incapacitate the victim, and if it was a suicidal wound, he would not be in a position to shoot again. A phenomenon **hesitation shots** (flinch shots) has been described, very similar to hesitation cuts. ***Hesitation shots*** are shots fired either away from the body or on non fatal parts of the body, just before the final fatal shot. The victim, when he points the gun at his head, subconsciously pulls away from it at the moment of discharge, with the result that the bullet misses him completely. This may be repeated 2-3 times before he finally gathers courage to kill himself. Very rarely he may point the gun at some non fatal parts of his body, eg foot and fire. On examining the room in such cases, an investigator may see a number of bullet holes on the walls, and may wrongly draw conclusion that there had been gunfight.

d. Other characteristics

Victims hands would show (a) **gunshot residue** [GSR] (b) Blood splashes [backspatter].

2. Disposal of the body

If attempts have been made to remove the body from the scene (presence of drag marks etc), it is a case of homicide.

3. Miscellaneous

Multiple methods of suicide (combined suicides), eg by poisoning, hanging and finally shooting indicate suicide.

Fig. 13.28: Blood spray on hands highly indicative of suicide

14. Thermal Injuries

I. BURNS

Burn is an injury which is caused by the application of heat, chemical substances to the external or internal surfaces of the body causing tissue destruction.

Salient features:

(1) **Exclusions** - The definition excludes heat applied by hot liquids, which is known as **scalds**.

(2) **Application of heat** - may be (i) direct or external e.g. flame, heated body or (ii) indirect e.g. corrosives, X-rays etc which produce heat within the body.

(3) T**emp for producing burn:** (i) **<44°C** – Burn cannot be produced as heat is conducted away via the capillaries of the skin. (ii) **44°C** – minimum temp required to cause burn. At this temp, heat must be applied for minimum **6 h** to produce a 2^{nd} -3^{rd} degree burn. **MLI** – forgotten hot water bottle applied against an unconscious or geriatric patient may cause burns inviting negligence charges. (iii) **Between 44°C–51°C -** - in temp by 1°**C** halves the duration of exposure necessary to cause same amount of burns (iv) **At 65°C** – only 2 s required for burns (v) **>70°C** – instantaneous.

II. CLASSIFICATION OF BURNS

Burns may be divided pathologically or clinically.

A. Pathological Classification

In 1832, **Guillaume Dupuytren** (1777–1835), a French military surgeon divided burns into 6 degrees. Later **Wilson** [as well as Austrian dermatologist **Hebra** (1816-1880)] merged them into 3 degrees (**Fig 14.1**). To some extent the degree of burns can be estimated by clinical assessment [i.e. naked eye examination]. More precise degree [depth] of burn can be measured by a high frequency ultrasound device.

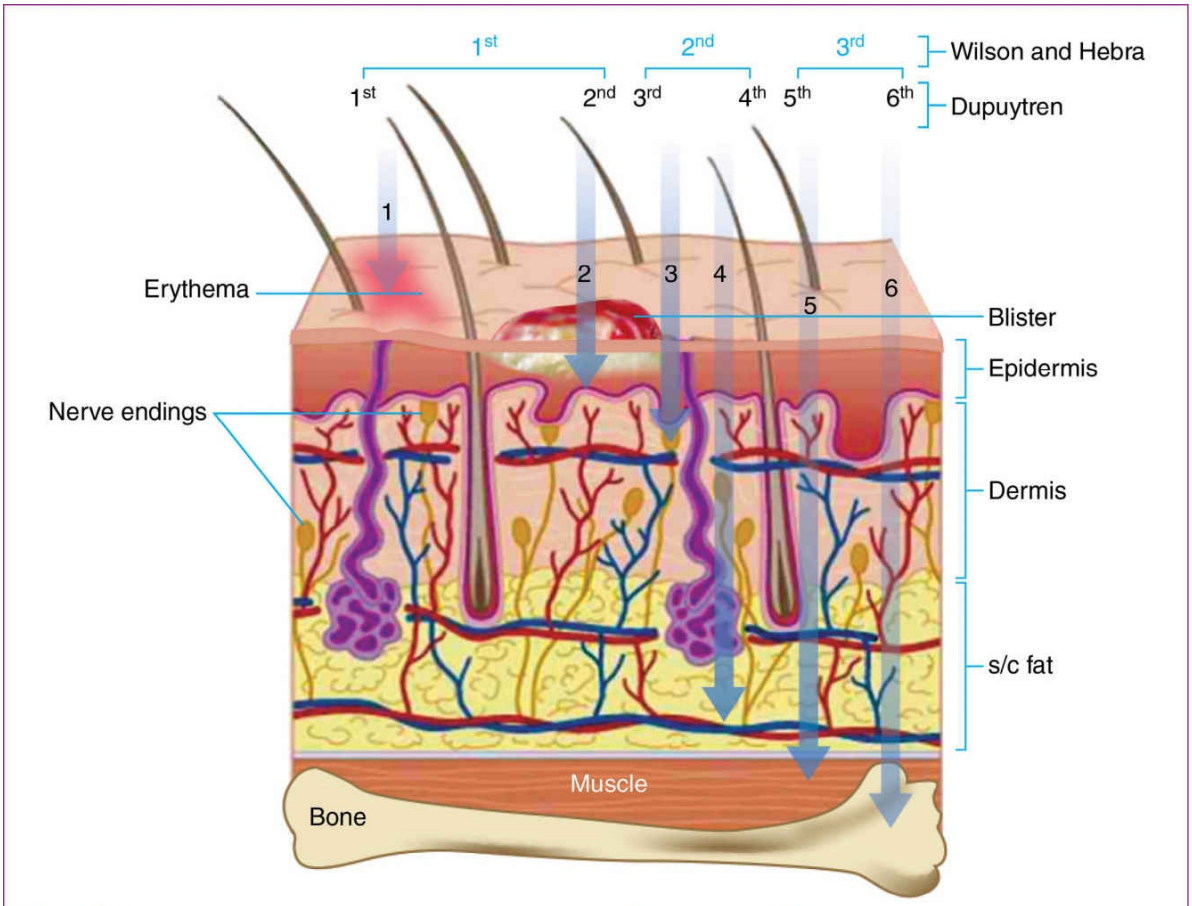


Fig. 14.1: Classification of burns. Correlate with **Fig. 14.2** and **Fig. 14.8**

1. 1st degree dupuytren

Erythema or reddening of skin. **Ex**. One touches a hot object and immediately retreats. His fingers are reddened and inflamed.

2. 2nd degree dupuytren

(1) **Blisters** - A blister (bulla) forms, which is covered by white avascular epidermis, bordered by red hyperemic skin

(2) **Contents of blister** – exudate containing protein and chloride [please see **Table 5** for the contents of pm blister]

(3) **Base of blister** - red

(4) There is capillary dilatation and transudation of fluid into the tissues causing swelling.

(5) **Singeing of hair** - present

(6) Burn is confined to epidermis.

(7) **Repair** – is complete without scar formation. ***Wilson*** and ***Hebra***

Classification combines 1st and 2nd degree to just one - 1st degree burns (***epidermal burns***).

3. 3rd degree dupuytren

(1) Epithelium completely destroyed.
(2) Burn reaches upper layers of dermis.
(3) Destruction of the papillary layer of the dermis
(4) Pain fibers exposed. Most painful burns.
(5) Shock produced is much more than in 1st and 2nd degree burns.

4. 4th degree dupuytren

(1) Whole dermis is destroyed with all dermal appendages and fatty tissue
(2) The burns are painless because pain fibers are destroyed
(3) **Appearance** – (a) Brownish black due to charring and eschar formation (b) Shriveled, depressed areas of coagulated tissue (c) bordered by reddish skin
(4) **Repair** – occurs with scar formation. Contraction of scar tissue may produce disfigurement or impaired function. ***Wilson*** and ***Hebra Classification*** combines 3rd and 4th degree to just one – 2nd degree burns (***dermo-epidermal burns***).

5. 5th degree dupuytren

(1) Muscles are destroyed [**Fig 14.2**]
(2) Burns are painless due to destruction of nerve fibers.

Fig. 14.2: Deep burns; 5th and 6th degree Dupuytren. Please correlate with **Fig 14.1**

6. 6th degree dupuytren

(1) Burns reach up to bone level

(2) Burns painless

(3) There is complete charring ***Wilson*** and ***Hebra Classification*** combines 5th and 6th degree to just one – 3rd degree burns (***deep burns***).

III. EFFECTS OF BURNS

Effects of burns depend on following factors:

A. Surface Area

Total surface area of body in an adult is 1.8 sq m. Surface area of burns is measured by following methods.

1. Rule of nine

According to this rule all areas of the body have areas in multiples of nines as follows (**Fig 14.3**): head and neck 9%, front of chest 9%, back of chest 9%, front of abdomen 9%, back of abdomen 9%, right upper limb 9%, left upper limb 9%, front of right lower limb 9%, back of right lower limb 9%, front of left lower limb 9% and back of left lower limb 9%. This makes 99%. The remaining 1% is

allotted to genitals. The rule was first pronounced in 1951 by **Dr. A.B. Wallace**, a Scottish surgeon who practiced at Bangour General Hospital, near Edinburgh. It is thus sometimes also known as "**Wallace's rule of nine**". Children have larger head and neck compared to adults, and smaller lower limbs. The ratio of their surface area is thus somewhat different (**Table 1**).

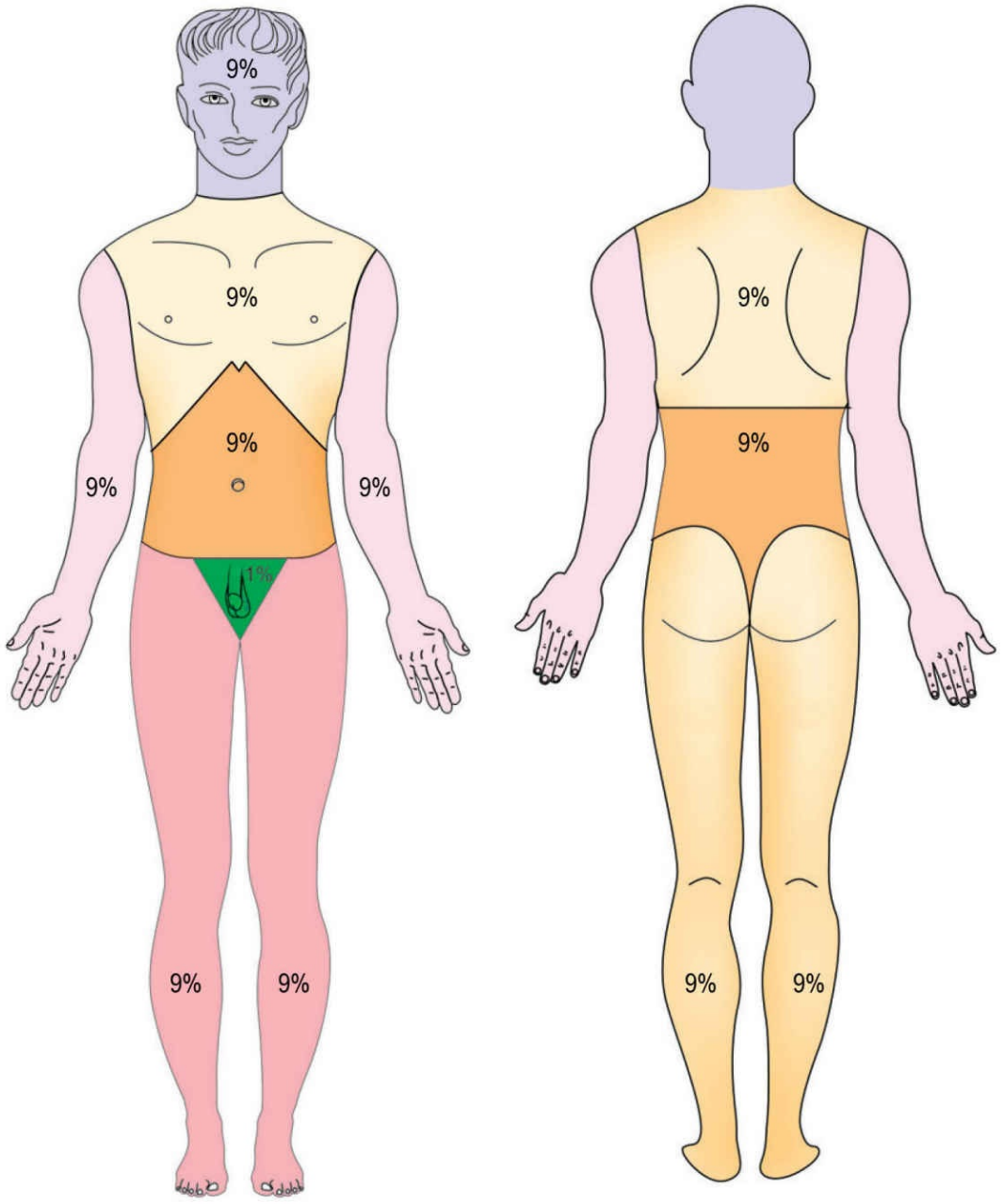


Fig. 14.3: Estimation of the extent of surface of burns in adult by rule of nine. Correlate all captions with **Table 1**

Table 1: Differences in surface areas of adult and children

Area	Adult	Child (1-12 y)	Infant (<1 y)
Head and neck	9%	18%	20%
front of chest	9%	9%	8%
Back of chest	9%	9%	8%
front of abdomen	9%	9%	8%
Back of abdomen	9%	9%	8%
Right upper limb	9%	9%	8%
Left upper limb	9%	9%	8%
front of right lower limb	9%	6.75%	8%
back of right lower limb	9%	6.75%	8%
front of left lower limb	9%	6.75%	8%
Back of left lower limb	9%	6.75%	8%
Genitalia	1%	1%	negligible
Total	100%	100%	100%

2. Rule of palms

Palmar surface of patient [including digital surface area] is roughly equal to 1% of his own body surface area. Useful in case of isolated and scattered burns.

3. Lund and browder chart

The **Lund and Browder chart** [LB chart], first proposed in **1944** by **Charles Lund** and **Newton Browder** is regarded as the most accurate in measuring % of burns and is widely used in everyday clinical practice. It takes into account the fact that 3 areas of body – **head**, **thigh** and **leg** - have **variable % areas** during different ages. These are designated as **A, B** and **C** respectively, and their body percentage is read directly from table. **13 areas have fixed %.** These are - (1)

front of neck [1%] (2) **back of neck** [1%] (3) **Right UL** [9½ %] (4) **Left UL** [9½ %] (5) **Anterior chest and abdomen** [13%] (6) **Posterior chest and abdomen** [13%] (7) **Genital area** [1%] (8) **Right gluteal region** [2½ %] (9) **Left gluteal region** [2½ %] (10) **Dorsum of right foot** [1¾ %] (11) **Sole of right foot** [1¾ %] (12) **Dorsum of left foot** [1¾ %] (13) **Sole of left foot** [1¾ %]. Fixed area thus adds up to **60%**. Rest **40%** is divided among **A** [½ of head], **B** [½ of one thigh] and **C** [½ of one leg], which represent variable areas. The variable areas at **all ages** would **always add up** to **40%.** The formula is: **2xA + 4xB + 4xC = 40%**. Pl note that in this chart, %age of some areas does not coincide with that suggested by **rule of nine**, eg upper limbs have **9.5%** instead of **9%**; anterior chest and abdomen have **13%** instead of **18%** and so on. It is because **rule of nine** is only an approximation.

B. The Degree of Heat

(1) Higher the heat more severe the damage
(2) Temp and time required for cremation – please see **ch 6**"Cremains.

C. The Duration of Exposure

Higher the duration, more severe the damage.

D. Age

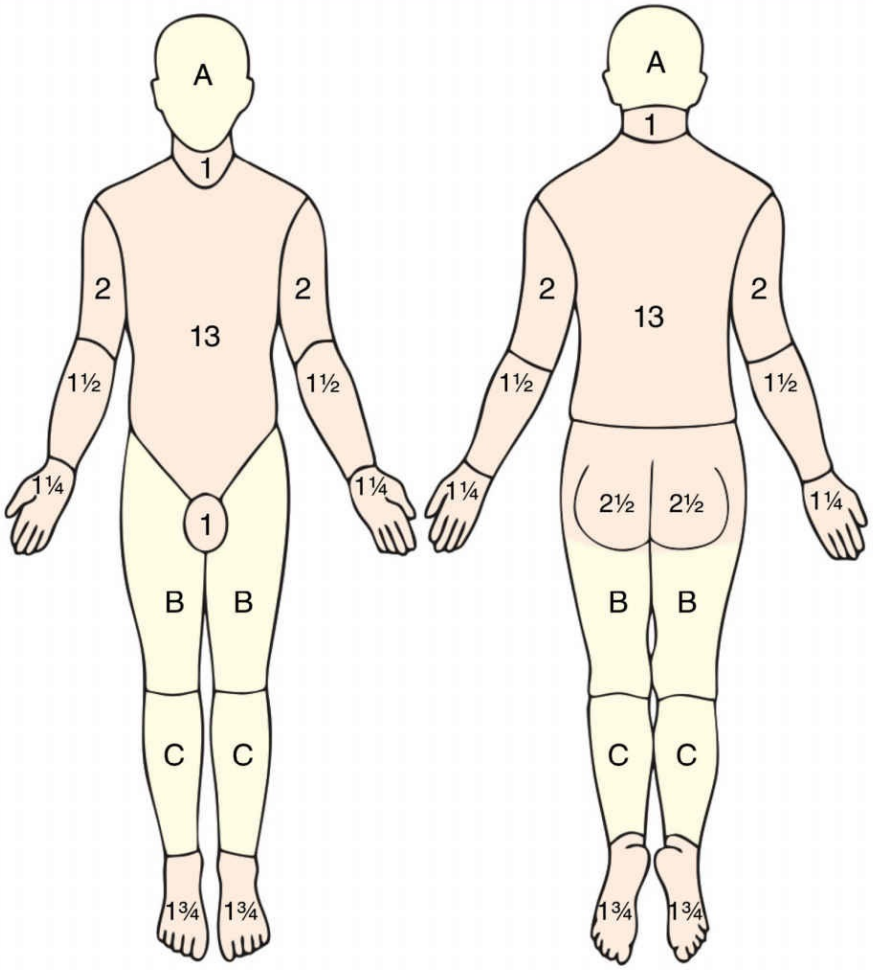
Children are more susceptible.

E. Sex

Women are more susceptible.

F. Site of Burns

Burns on head and neck, face, trunk and anterior abdominal wall are more dangerous.



Age in years	0–1	1–4	5–9	10–14	15–18	(>18)
A (½ of head)	9.50	8.50	6.50	5.50	4.50	3.50
B (½ of one thigh)	2.75	3.25	4.00	4.50	4.50	4.75
C (½ of one leg)	2.50	2.50	2.75	3.00	3.25	3.50

Fig. 14.4: Lund and Browder chart. Pink areas show fixed percentages; yellow areas show variable percentages.

IV. CAUSES OF DEATH IN BURNS

A. Immediate

Immediate causes (death within 24 hours) are

(1) **Accidents** – occurring in an attempt to escape from a burning building or by injuries due to falling masonry, timber or other structures on the body

(2) **Primary (neurogenic) shock** due to pain

(3) **Carbon monoxide intoxication** – If burns occurred in a confined area 4.Acute respiratory insufficiency, lack of oxygen.

B. Late

Late causes [death from 24 h-few months] are

(1) **Biochemical disturbances** – due to fluid loss and destruction of tissue, eg hypokalemia

(2) **GIT disturbances:**

(i) Curling's ulcers [please see below]

(ii) Dilatation of stomach

(iii) hemorrhage into intestines

(3) **Complications of burns:** (i) Gangrene (ii) Pyemia (iii) Tetanus

(4) **Embolism:**

(i) Fat embolism – rare

(ii) Pulmonary embolism – from thrombosis of leg veins due to tissue damage and immobility

(5) **Inhalation of toxic substances** -

(i) CO inhalation

(ii) Cyanide inhalation - cyanides are produced during the combustion of nylon clothes [please also see ch 44"cyanide"building fires]

(iii) Smoke inhalation

(iv) Inhalation of other burn products [Non specific toxic substances]

(6) **Respiratory tract injury:** (i) edema of glottis (ii) Pulmonary edema

(7) **Secondary shock** – due to fluid loss from burnt surface

(8) **Septicemia** – Most important cause in late deaths.

(9) **Toxemia** - due to absorption of various metabolites from the burnt tissue

(10) **Miscellaneous causes:** (i) Free radicals (ii) Oxygen deprivation.

C. Very Late

Very late causes [death after several years] are: **Marjolin's ulcer** - Malignant transformation of a burn scar.

V. PM APPEARANCES

A. External

1. Clothes

(1) **Cotton** fabrics burn faster than **wool**; victim may get more severe burns if wearing cotton fabrics. **Nylon** and **polyester** melt and stick to skin, and may produce far more serious burns.

(2) **Long loose garments** catch fire easily and are thus more dangerous than **close fitting garments**.

(3) **Examination of clothes** - Clothes should be removed, preserved in plastic bags or air tight bottles and sent for examination for the presence of **kerosene**, **petrol** etc. Smell the clothes is indicative.

2. Stature and weight of the body

Markedly reduced due to

(1) organic matter of the body consumed as fuel and

(2) loss of tissue fluid

(3) skeletal fractures 4.pulverization of intervertebral discs. Thus they cannot be used to establish the identity of the body. The stature may be less by several cms and wt loss may be up to 60%.

3. Facial features

(1) **Face:**

(i) Swollen and distorted.

(ii) In severe burns, soft tissues of the face may be completely burnt, exposing the skull.

(iii) Features are changed due to contractions.

(2) **Eyes** - In most cases

(i) the eyes are closed, and the shrunken lids can be opened incompletely and that too with difficulty.

(ii) Cataract – may be seen as a result of coagulation of proteins in the lens of the eye. This may occur at temperatures of 150°C and above.

(iii) Jaundice may be seen [liver failure]

(3) **Nose and mouth** – may show froth due to pulmonary edema [due to heat irritation of air passages and lungs]. Mouth is usually open with shrunken lips

(4) **Tongue** – Burnt. Protruding (due to contraction of the tissues of face and neck).

(5) **Crow's feet** – These are areas without burns or soot deposits on the forehead, in the angles of the eyes, and in naso-oral folds. Mechanism - This is due to

non-penetration of smoke into the base of wrinkles, which may form due to pain. Thus crow's feet may indicate antemortem burns.

(6) **Neck** - Unsooted creases [mechanism similar to that of crow's feet] may be found all over the body, eg in the neck and groins. In the neck such unsooted creases may mimic a ligature mark [**pseudoligature mark**].

Fig. 14.5: Crow's feet.

4. Pugilistic attitude

Pugilistic attitude [L. pugil, boxer][Syn. Boxing attitude (or posture), defense attitude (or posture), fencing attitude (or posture), heat rigor, heat stiffening] is the characteristic attitude adopted by the body after severe burns. It is similar to the one commonly adopted by boxers during fight, and hence the name.

a. Causes

Burns, falling in hot liquids, high voltage electric shocks.

b. Mechanism

(1) Pugilistic attitude is due to

(i) coagulation of muscle proteins

(ii) dehydration and

(iii) contracture of muscles due to heat. Flexors being bulkier than extensors are contract more.

(2) **Temp required** – > 50°C; well-established at 75°C. **Myosin** coagulates (in mammals) at 50°C, and **albumin** coagulates at 73° C. If, therefore the body is subjected to a temperature exceeding 73°C, all these proteins are coagulated. This rigidity is stronger than that produced by rigor mortis.

(3) Normal rigor mortis does not appear in such muscles.

Memory Aid 1: Coagulation temperature of proteins

MAL

Myosin – coagulates first (50°C)

ALbumin - coagulates later (73°C)

Temperatures are rough multiples of 25 (50, 75)

c. Main features [Fig 14.6]

Due to contracture of muscles

(1) **Head** slightly extended

(2) **Arms**

(i) held out in front of the body and

(ii) flexed at elbows and wrists

(3) **Fingers** hooked like claws

(4) **Legs** flexed at hips and knees.

(5) **Body** – showing opisthotonus [arching of body backwards] due to contraction of paraspinal muscles

(6) **Delayed decomposition** – due to intense heat producing “heat fixation” of tissues and inactivating autolytic enzymes.

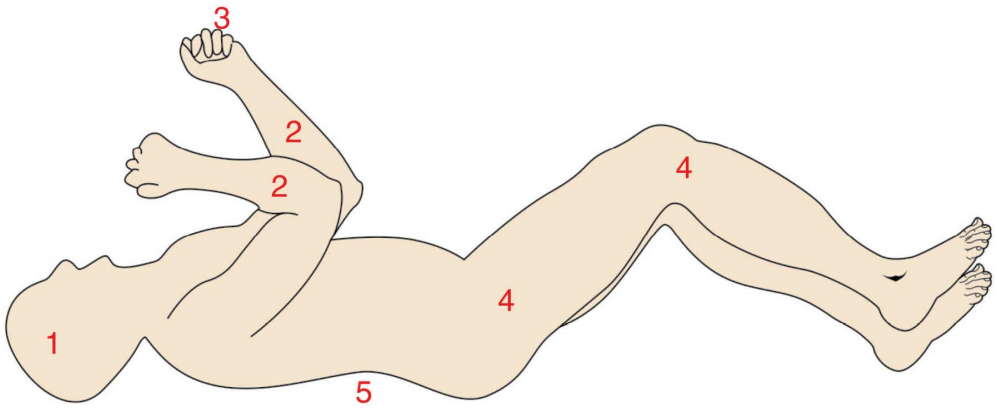


Fig. 14.6: Pugilistic attitude. General features. Please correlate nos (given in red). with text.

d. Appearance of muscles

(1) Contracted, desiccated, even carbonized on the surface. A zone of brownish pink "cooked meat" lies beneath this. Further below, normal red muscle may be seen.

(2) **Degree and severity of contractures** – depend on intensity of heat and duration for which heat was applied.

(3) Stiffening remains until muscles and ligaments soften from decomposition.

e. Interpretation

(1) It occurs irrespective of whether the victim was alive or dead at the time of burning. Thus pugilistic attitude is not a sign of antemortem burns.

(2) Pugilistic attitude is never seen in the living victim. **Case study** – A case of burns was brought to the author for PM. Pugilistic attitude was present. The story was that the victim survived for 4 hours and even gave her dying declaration in which she admitted it was accidental burns. Author opined that this was not possible.

5. Algor mortis, livor mortis, rigor mortis

Difficult to judge in a case of burn. Thus time of death is difficult to determine.

6. Burnt areas

(1) Burnt areas

(i) have a patchy distribution and vary in size and shape. Shape may often take

shape of ornaments worn [**patterned burns**].

(ii) They show **reddening, blistering** or **charring** depending upon the depth of burns.

(iii) Depth of burn in different areas may be different, and must be assessed separately.

(iv) There may be **blackening** over some areas due to deposition of soot.

(v) In severe burns, skin may be leathery, stiffened and yellow-brown. As the skin dries after death, it may leave a stiff, parchment-like surface.

(vi) RBCs may hemolyze in vessels and stain vessels presenting a branching pattern.

(2) **Extent of burns** – should be assessed [i.e. percentage area of body involved]

(3) **Blisters** – may be seen as small islands away from the periphery; or whole burnt area may form one large blister, or there may be a confluence of several blisters. Blisters are also seen in

(i) CO poisoning

(ii) Deep coma

(iii) Gasoline exposure [AM or PM] and

(iv) putrefaction. **Differentiation** – When blisters due to other causes rupture, they leave a moist, pale, raw, yellowish surface, which tans and becomes dark brown and leathery as it dries. Blisters due to burns reveal an angry red base.

(4) **Hands** and **feet** – if burnt, the skin, including fingernails and toenails detaches as a glove [due to appearance of fluid between the layers of the skin; **degloving**].

(5) **Burns absent** – Burns may be absent sometimes

(i) in gums

(ii) in armpits

(iii) Hands and feet – Rarely if burning is sudden and intense, they may drop off and may be preserved with very little damage [because they fall away from the source of fire]

(iv) Unburnt areas on wrists and ankles – may indicate victim was bound before the fire started

(v) On abdomen – if person was sitting and face falls forwards between knees

(vi) on buttocks – if person was in a sitting posture

(vii) in portions of body where clothing is tight eg under the (a) belt (b) brassiere

(c) shoes or (d) tight buttoned collar. The unburnt areas may reveal true color of skin and may establish racial identification in some cases. Unburnt area around tight buttoned collar may look like a strangulation mark [please also see unsooted creases of neck above].

(6) **Flash burns** – Seen in burns due to

(i) Explosions

(ii) Ignition of highly inflammable liquids or gases

(iii) Sudden ignition of fine particulate matter. Their characteristics are (a) seen only on exposed areas of skin (b) Depth is usually 1^{st} or 2^{nd} degree [Dupuytren] (c) ***uniformity*** [in depth]

(7) **Microscopic appearance:**

(i) Cutaneous erythema, characterized by dilated capillaries

(ii) Leukocytic infiltration [within 6 hours]

(iii) epidermal cells show (a) necrosis (b) swelling of nuclei, and (c) condensation of nuclear chromatin

(iv) **edema** of subepidermal connective tissue.

Fig. 14.7: Areas spared by burns may reproduce an "image" of clothing worn. This may reveal the nature of clothes worn at the time of burns, which may prove vital in some investigations This picture shows "image" of a bra on the back. There is also an image of trouser belt and a triangular underwear. This is an important finding because this is not traditionally worn by Indian ladies.

Table 2: Differences between pugilistic attitude and rigor mortis

S.No	Feature	Pugilistic attitude	Rigor mortis
1.	Cause	Intense cooking of muscles. Heat coagulation of muscle protein	Formation of actinomyosin complex
2.	Contracture of muscles	Very pronounced	Minimal, virtually nil
3.	Degree of stiffness	High	Moderate
4.	Time of formation	Antemortem or postmortem	Postmortem only. 3 hours after death.
5.	Role of heat	>50°C; well-established at 75°C.	No role. However -temp may cause acceleration
6.	External appearance	signs of burning eg, blackening, blisters	No specific features

7. Heat ruptures

(1) Heat ruptures [or tears] are produced by splitting of the soft parts. If skin is completely burnt, underlying **muscles** may show rupture. They may be partly cooked and brownish. Sometimes the muscles may be converted into black, brittle masses.

(2) They may be antemortem or postmortem.

(3) **Mechanism**:

(i) Excessive heat – Due to excessive heat, skin contracts [antemortem heat ruptures]

(ii) Disturbance of body - Sometimes external disturbance of the body may produce heat ruptures, eg when a severely burnt body is moved, the charred skin cracks easily producing tears. Such tears are commonly seen around the places where the body was held – shoulders, elbows, knees [postmortem heat ruptures].

(4) **Site** – May be anywhere, but usually over extensor surfaces and joints

(5) **Size** – Several cm in length

(6) **D/d** – May resemble lacerations [rarely even incised wounds] [**Table 3**].

Table 3: Differences between heat rupture and lacerated wound

S.No	Feature	Heat rupture	Lacerated wound
1.	Cause	Exposure to intense heat	Blunt force
2.	Site	Fatty tissue	Anywhere
3.	Bleeding	Absent in wound and surrounding tissues [because heat coagulates blood in the vessels]	Present
4.	Margins	Absence of bruising and vital reaction	Bruising and vital reaction present
5.	Floor	Shows intact nerves and vessels	Shows lacerated nerves and vessels
6.	Associated findings	Of burns	Of trauma

8. Hair

(1) **Singed** and **burnt**, but may be spared in armpits.
(2) In lesser degree of burns, ends may be bulbous or clubbed.
(3) **Color** – Color of hair may sometimes reveal the temp reached.
(i) Gray hair become brassy blonde or even golden at 250°F
(ii) Brown hair become slightly reddish when exposed to 400°F [for 10-15 min]
(iii) Black hair do not change color on exposure to heat.

Fig. 14.8: Brassy blonde hair in burns

9. Miscellaneous findings

(1) **Identification marks** – eg moles, scars and tattoos may be destroyed in 5th and 6th degree burns.

(2) **Abdominal wall** – may show rupture. Caused by

(i) burning of the abdominal wall

(ii) expansion of gases within the intestines exerting pressure on weakened abdominal wall. Intestines may be seen protruding through this gap, which may themselves be ruptured rarely.

(3) **Anus** – Gaping may be seen because of shrinkage of the perianal tissue. This may be misinterpreted for sodomy or homosexuality

(4) **Pre-existing lesions** – may become smaller and change in shape due to shrinkage of the skin. For example, originally slit like stabs may assume a circular shape. Lesions may sometimes migrate toward the center of the thermal damage.

(5) **Complete destruction** - If the flame is unchecked, the body is reduced to a shapeless, coal like mass and finally to a heap of grey and yellow ashes. These are known as **cremains.**

B. Internal

1. Heat hematoma

Heat hematoma is the collection of blood in the extradural space due to excessive heat.

Salient features:

(1) **Degree of heat required** – occurs mostly when the heat is enough to cause charring of the skull.

(2) **Appearance:**

(i) **Gross** - ***Honeycomb appearance*** due to presence of air bubbles inside

(ii) **Color** - light chocolate color, or pink if blood contains CO.

(iii) **Consistency - Soft and friable** [bubbles]

(iv) **Thickness** – 1.5 mm to 1.5 cm. Volume up to 120 ml.

(v) **Differences from antemortem EDH** – please see **Table 4**.

(3) **Adjacent area of brain** – is hardened and discolored (due to heat)

(4) **Distribution** – of heat hematoma follows closely the distribution of the charring of the outer table of skull. Found also along the superior sagittal sinus. Most common site is ***parietotemporal region***.

(5) **Mechanism of development** – Not clear. Could be expansion of blood in the diploe (due to heat) and rupture of dural venous sinuses.

Table 4: Differences between heat hematoma and extradural hematoma [**ch 17**]

S.No	Trait	Heat hematoma (extradural hematoma due to burns)	Traumatic hematoma (extradural hematoma due to antemortem trauma)
1.	Cause	Intense heat	Blunt trauma to head
2.	Location	Parietotemporal. Along superior sagittal sinus	At the site of trauma (coup injury)
3.	Distribution	Diffuse	Localized
4.	Mechanism	Expansion of blood in diploe/rupture of dural venous sinuses	Rupture of middle meningeal artery (most common). Other arteries according to site of trauma.
5.	Appearance	**Honeycomb appearance**. Soft, friable. Light chocolate or pink in color	**No honeycombing is seen**. More rubbery. Reddish purple in color. May be very dark bordering on black
6.	Skull	may show pm fractures (heat fractures)	shows antemortem fractures
7.	CNS	No injury	Injury present
8.	Carboxyhemoglobin (COHb) in clot	Yes. Levels of COHb in heat hematoma same as that in peripheral blood.	No

2. Heat fractures (Thermal fractures)

Heat fractures are postmortem fractures produced by excessive heat.

Salient features:

(1) **Appearance** - Bones are burnt and assume a gray-white color. Usually shows a fine superficial network of heat fractures on its cortical surface, which may crumble on handling

(2) **Types** - Three types; skull fracture, long bone fracture, other fracture.

a. Skull fractures

(1) **Frequency** - Occur most commonly in areas where the skull has been severely burned

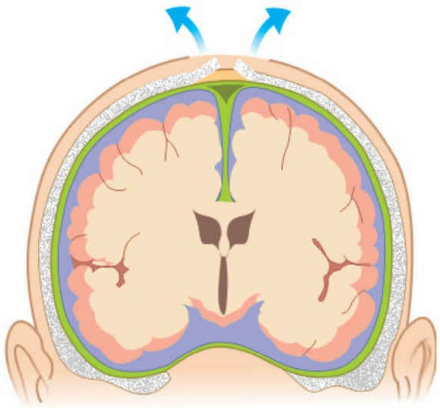
(2) **Site** – either side of skull above temples

Fig. 14.9: Heat fractures of the skull.

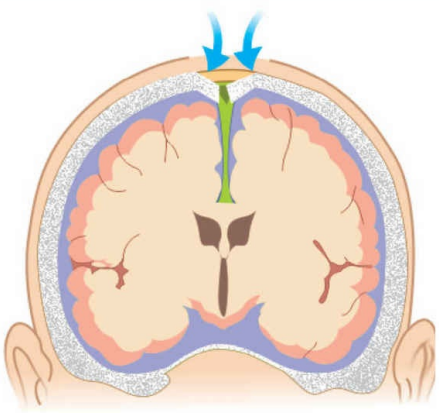
(3) **Types:**

(i) Due to -of intracranial pressure by steam - (-a)Akin to an intracranial explosion (-b)Separation of ununited sutures occurs (-c)displacement - fractures show wide gaping defects and widely separated bony margins. Elevated fracture [**Fig 14.10**] (-d)fractured tables – both

(ii) Due to rapid drying of bone with contraction – (a) fractured tables - Only outer table is fractured (b) Displacement – none (c) Fracture – stellate (d) may cross a suture line.



(1)
Bone fragments bent outwards in heat fractures of skull, due to steam pressure within the skull (Elevated fracture)



(2)
Bone fragments driven inwards in blunt force impact (depressed fracture)

Fig. 14.10: (1) Heat fractures of the skull. They are typically elevated fractures. Compare with (2) depressed fractures in which bones pieces are driven inwards.

b. Long bone fractures

(1) **Cause** – excessive shrinkage of muscles, pulling tendons and bones

(2) **Appearance** – Bones may show curving due to drying out [**curved fracture**].

c. Other fractures

Hyoid – May be so cooked and fragile that it may fracture, or sometimes even if intact, fractures on even most gentle manipulation.

3. Air passages

(1) **Mucosa:**

(i) of entire air passage from nose till terminal bronchi is congested and edematous.

(ii) Laryngeal edema .es rapidly with -ing PM interval and only wrinkling of mucus membrane may be seen.

(iii) Microscopically – eosinophils in walls

(iv) may be thickened

(v) May show frank burns if flame or superheated air is inhaled

(2) **Vocal cords** – destroyed along with overlying epithelium

(3) **Aspirated black carbon particles [soot]**- seen embedded in mucus lying on

the surface in nose, mouth, larynx, trachea and bronchi.

(4) **Presence of carbon particles [soot] in terminal bronchioles** – on histological examination is a very strong proof of antemortem burns.

(5) **Amount of soot depends upon:**

(i) Duration of survival - in smoke contaminated atmosphere

(ii) Type of combustible objects -present on victim's body and around

(iii) Type of fire – eg smoldering fire burning partially wet objects produces more smoke

(6) **Demonstration of soot** – Sometimes soot may not be clearly differentiable because of underlying dark, congested mucosa. In such cases, clean mucus from trachea with a cotton swab and smear it on a piece of white paper. It would blacken due to carbon particles.

(7) **Smaller peripheral air passages:**

(i) may be blocked - by mucus, soot and mucosal cells. The latter are shed from larger air passages higher up due to burn injury.

(ii) may show vomitus – inhalation of smoke" vomiting"inhalation of vomit.

(8) **Ulceration and secondary infection** – in cases of death after a few days of survival.

4. CVS

(1) **Heart** – full of clotted blood. Histology shows interstitial edema and fragmentation of myocardial fibres.

(2) **Pericardium, endocardium**-show petechial hemorrhages.

5. Lungs

(1) **Lungs** – Markedly edematous and congested. Sometimes shrunken. Airless, discolored, hardened, heavy. May present picture of shock lung [**ch 11**]. In late deaths, show bronchopneumonia.

(2) **Pleurae** – inflamed, show petechial hemorrhages

(3) **blood vessels of lungs** – may contain fat droplets due to a physico-chemical alteration of fat already present in blood. Must not be confused with true fat embolism.

6. GIT

(1) **Mucosa:**

(i) of esophagus, stomach may be congested because of entry of hot gases up to this region.

(ii) May show petechial hemorrhages, often with erosions and even acute ulcers

(2) **Aspirated black carbon particles** - seen embedded in mucus lying on the surface in pharynx, esophagus and stomach. Can reach up to stomach due to peristaltic movements and hot gases reaching up to this point. Generally not seen beyond stomach, even in survivors.

(3) **Inflammation and ulceration** – of Peyer's patches and solitary glands in intestines.

(4) **Curling's ulcers:**

(i) Acute peptic ulcer of the duodenum, generally seen after about 7-10 days in extensive burns, but may be seen as early as after 1 day. Appear as sharply punched out mucosal defects. Especially common in children. There may be bacterial or fungal colonies in the floors of ulcers.

(ii) Incidence – 10%

(iii) Site – 1st part of duodenum [**most common**]. May be seen all over duodenum, stomach and even large intestines.

(iv) Mechanism – (a) -ed ACTH and corticosteroids released as result of burn stress. (b) -ed levels of blood histamine (c) gastrointestinal ischemia (d) Hemoconcentration. (e) thrombosis of vessels due to burns

(v) Complications – (a) may erode large vessels "massive hemorrhage"late death (b) may perforate"late death.

7. Viscera

(1) **Well preserved** - Even in badly burnt bodies viscera are usually well preserved because body tissues are poor conductor of heat

(2) Show marked **congestion**. Sometimes may show a "cooked" appearance, i.e. hardened and discolored

(3) **Puppet organs** – All internal organs may shrink to very small size due to loss of water. They are firm, hardened, and cooked by the heat. Systematic observations of cremations show that this change occurs between 30-50 min. If the fire continues, the surface of the organs becomes increasingly **bosselated** [marked or covered with many bosses (protuberances)] and is reduced to a sponge like residual structure in the end. The tissue is completely desiccated and disintegrates into ash at the slightest touch

["Zermürbungspunkt" (crumbling point)].

(4) **Liver:**

(i) shows cloudy swelling

(ii) Fatty changes – due to burns or even due to treatment with tannic acid

(5) **Spleen** – softened. There is edema and necrosis of lymphoid germinal centers.

(6) **Kidneys:**

(i) show cloudy swelling, capillary thrombosis and infarction.

(ii) heme casts in renal tubules

(iii) hemoglobinuria if >30% surface area is burnt

(iv) microcirculatory insufficiency and renal ischemia due to thrombosis

(7) **Adrenals** – enlarged, congested.

8. CNS

(1) **Brain:**

Cooked appearance – (a) Color – yellow to light brown (b) Firm (c) Shrunken

(2) **Dura** – leathery. May be split with brain oozing out forming a mass of frothy paste.

9. Blood

(1) Hemoconcentration

(2) **Color** – cherry red, due to formation of carboxyhemoglobin [COHb]"may turn brownish due to heat

(3) **COHb levels** - usually >10%; in some cases may reach up to 70-80%. Children and old persons may die at much lower levels [30-40%]. Presence of COHb associated with external burns is the surest sign of ***antemortem burns***.

(4) **COHb levels depend upon** –

(i) Victim related factors – (a) Activity of victim (b) Hb content of blood (c) Rate and depth of respiration

(ii) Extrinsic factors - (a) Conc of CO in inhaled air (b) Duration of exposure

(5) **Causes of absent COHb:**

(i) Adequate supply of O_2 - causing .production of CO

(ii) Convection air currents - driving away CO laden air

(iii) Flash fires - eg in conflagrations of chemical plants

(iv) Inhalation of superheated air - constriction of air passages"death occurring

due to suffocation

(v) Instantaneous or near instantaneous [rapid] death - eg in explosions, warfare

(6) **Other gases found in blood:**

(i) Ammonia, H_2S, oxides of sulphur – due to burning of wool or silk

(ii) HCN – due to burning of domestic furnishings made up of polyurethane [**ch 44**]

(iii) Oxides of nitrogen – due to burning of nitrogen containing substances, eg Nitrocellulose films [produce nitrogen oxide, nitrogen tetroxide]

(7) **Histology** – macrophages may show soot particles.

10. Miscellaneous

(1) Tissue edema

(2) Serous cavities – contain excessive fluid

(3) Infection – in delayed deaths.

VI. AGE OF BURNS

A. Macroscopic and Histological

Age of burns is quite inaccurate, as it depends on a number of variables (extent and depth of burns, age and nutrition of victim, causative agent). Broad guidelines are as follows:

(1) **Immediate** – erythema

(2) **1 hour** – (a) Vesication (b) BV in the floor of blister are dilated

(3) **6 hours** – (a) Epidermis – Coagulated, thinned out and deeply staining. Cells in the deeper layers become elongated, with their long axes oriented at right angles to the skin surface (closely resembling streaming in electrical mark) (b) Dermis - Inflammatory reaction starts with infiltration of polymorphonuclear leucocytes

(4) **12-24 h** – Exudate begins to dry

(5) **36 hours** – If burn area is not kept clean, pus forms. After the formation of pus, it becomes very difficult to tell the age of wound. If pus does not form, the healing proceeds as follows

(6) **2-3 days** – Dry brown crust forms

(7) **4-6 days** – Superficial slough falls

(8) **2 weeks** – Deep slough falls
(9) **2-3 weeks** – Granulation tissue covers the surface
(10) **Several weeks to months** – Scar forms.
(11) **10-25 years later** – Marjolin's ulcer may form. It is an ulcerating squamous cell carcinoma in an area which suffered burn injuries (after French surgeon, **Jean Marjolin**, who first described the condition in 1828). It is slow growing, painless carcinoma, which does not spread by lymph (due to local destruction of lymphatic channels). Treatment is usually surgical. May be medicolegally significant if original burn injury was work related, or due to negligence.

VII. ANTEMORTEM AND POSTMORTEM BURNS

Differences between AM and PM burns are given in **Table 5**. If destruction of body is considerable, their differentiation may be difficult or even impossible.

Salient features:

(1) **Line of redness** [**Fig 14.11**]:
(i) **Appearance**
- It is a **zone of hyperemia** between burnt and unburnt area. A reaction to burns.
(ii) **Time of production** – Seen in antemortem burns. But may be seen if heat is applied in the ***early*** **supravital period** (please see chapter on thanatology) too, i.e. up to about **1 hour after death**. Burns produced within this period are difficult to differentiate from true antemortem burns. Presence of COHb is a useful indicator. (iii) **Absent** – (a) when death is immediate (b) when whole body is burnt [No unburnt area; so no zone differentiating burnt and unburnt areas] (iv) **Extent** – involves whole thickness of skin (epidermis and dermis) (v) **Width** – varies, but usually between **5-20 mm** (vi) **Pathogenesis** – due to capillary dilatation (vii) **Permanency** – Persists after death.

(2) **Antemortem blister:** (i) **Appearance** – Appears as a raised dome and contains fluid [exudate]. (ii) **Time of production** – Seen in antemortem burns. Very rarely blisters with a red rim may be seen in PM burns due to **contraction of dermal capillaries**. This forces blood to the periphery of blister, producing a reddish rim.

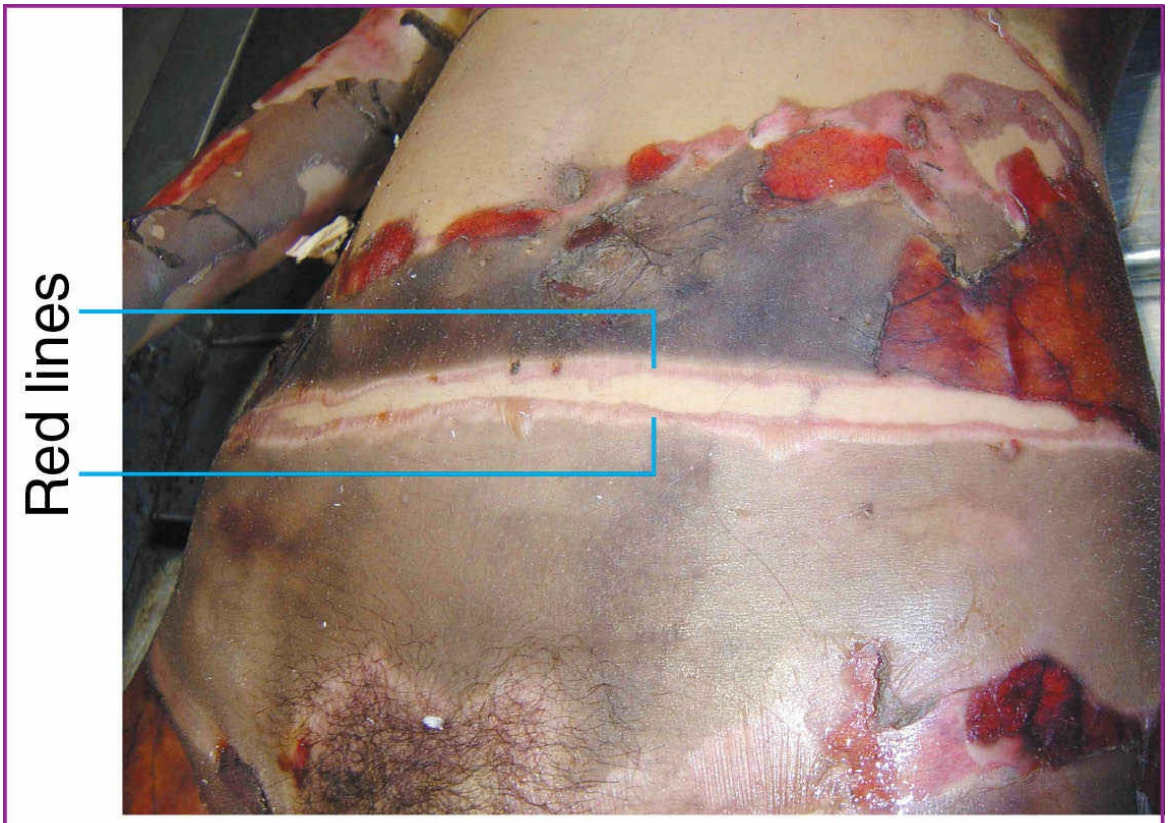


Fig. 14.11: Red lines at the margin of burns

Table 5: Difference between Antemortem and postmortem burns

S.No	Trait	Antemortem burns	Postmortem burns
1.	Line of redness	Present; absent if death is immediate	Absent
2.	Blisters (Vesication)	(a) Base is **red** and **inflamed**. (b) Contain **exudate**, which is rich in proteins and chlorides. Differentiation on the basis of proteins and chlorides is doubtful and has been challenged. (c) Show hyperemia around. (d) Blisters formed by scalds have same characteristics.	(i) **Blister produced by pm burns**"(a) Base is **pale, dry** and **hard**. (b) Contains **air**. (c) No hyperemia around. (ii) **Blister produced by putrefaction**"(a) Base is **pale**. (b) Contents (I) mostly air (II)May contain fluid, but it is **transudate** (very low in proteins and chlorides). (III) Sometimes blisters are in the form of large fragile sacs containing pink-red serous fluid. (c) No hyperemia around.
3.	Vital reaction	Marked inflammatory reaction. Leukocytic infiltration occurs almost immediately.	None
4.	Enzymes	Peripheral zone of burn shows - in reparative enzymes	No such -
5.	COHb	Present	Absent (very useful indicator in cases where burns were produced in early supravital period)

Fig. 14.12: Various depths of burns (1) blisters (2nd degree Dupuytren) (2) Epithelium completely destroyed (3rd degree Dupuytren). Please correlate with **Fig 14.1**

VIII. ML IMPORTANCE

(1) **Branding** – Application of red hot iron rods on skin is very common among the illiterates and tribals of India to treat a vast variety of conditions from jaundice to neurological disorders. Patient may present with multiple burn scars in various stages of healing which may arouse suspicion of physical abuse and torture.

(2) **Manner of infliction of burns:**

(i) Homicide– Very common in India, mostly in the form of dowry deaths

(ii) suicide – very common among females
(iii) accident – bursting of inferior quality stoves, gas cylinders etc
(3) **Necklacing** is the practice of extrajudicial execution carried out by forcing a rubber tire, filled with petrol, around a victim's chest and arms, and setting it on fire. Common in South Africa.
(4) **Negligent conduct with respect to fire or combustible matter** - so as to endanger human life"punishment of 6 months or `1000 fine or both [**S.285, IPC**].

IX. SPONTANEOUS HUMAN COMBUSTION (SHC)

Spontaneous human combustion (or simply ***spontaneous combustion***) is more or less an ***anecdotal*** phenomenon, where a live person is supposed to go up in flames on its own, without any outside source of heat. In the past centuries, the defense sometimes used to raise the plea of SHC, and in some cases it was even accepted by the courts. However it is ***highly doubtful*** if such a phenomenon actually occurs.

X. PRETERNATURAL COMBUSTION

Preternatural combustion is a phenomenon, where a putrefying dead body catches fire spontaneously due to formation of inflammable gases, such as hydrogen, hydrogen sulphide and methane (Ch 9). A flame is necessary for ignition. **Preternatural combustion is different from SHC**; former occurs in the dead and putrefaction is a must. SHC is seen in live people, or immediately after death [please see "wick effect" hypothesis above].

XI. SCALDS

A ***scald*** is an injury which results from the application of liquids or from steam.

Salient features:

(1) **Cause:** (i) Hot non sticky liquids. (a) Water at ***55°C***" produces full thickness scalds in **25s** (b) at ***70°C***"same damage in just **1s**. Can occur through clothing, with no damage to it (ii) Sticky liquids – eg oils, syrup, tar cause more severe

scalds than hot water. (iii) Superheated steam (a) If touches skin"soddens the skin"Becomes dirty white in color (b) If inhaled"scalds on skin are not seen, but death can still occur. Thermal inj to resp tract"Edema of mucus membrane"obstruction"death by asphyxia

(2) **Types:** (i) Immersion burns – caused by immersion in hot liquids. May be accidental or homicidal (ii) Splash or spill burns – by splashing. Mostly accidental (iii) Steam burns

(3) **Destruction** - not as deep as in burns

(4) **Redness** [erythema] - appears at once; due to vasoparalysis; blistering takes place after a few minutes

(5) **Blister Characteristics:** (i) formed due to -ed permeability of capillaries (ii) have a hyperemic zone around them [**Table 5**] (iii) Reddening and swelling of the papillae in the floor (iv) Blister fluid contains WBC and RBC (v) Soddening and bleaching, but no singeing, blackening, charring (vi) Blistered skin removed"raw pink surface"If left exposed"becomes brownish, dry and hard

(6) **Cause of death:** (i) shock (ii) Fluid and electrolyte disturbance (iii) secondary infection

A. Degrees of Scalds

Divided into three (1) Erythema (2) Blister formation (3) Necrosis of dermis.

B. ML Importance

(1) **Accidental** - Scalds are mostly accidental

(i) Bursting of hot water bottles, pressure cookers, boilers etc

(ii) Splashing of fluids from cooking utensils

(iii) Pulling over kettles or saucepans by children. If children suck the spouts of kettles containing hot liquids, can cause severe scalds of mouth and respiratory tract

(2) **Suicide** – by scalding is rare

(3) **Criminal offences by scalding:**

(i) Murder – rare

(ii) Grievous hurt - Boiling water may be thrown on a victim in order to injure. If face is disfigured, it is a grievous hurt [**S.320, IPC**]

(iii) Child abuse by scalding [**ch 27**].

Table 6: Differences between burns due to dry heat, moist heat and chemicals

S.No	Feature	Dry heat (Burns)	Moist heat (scalds)	Chemical burns
1.	Cause	Flame or heated solid body	Liquid above 60^0C or steam	Chemicals, eg. corrosive
2.	Site	At or above the site of contact (as flame tends to rise up)	At or below the site of contact (as liquid tends to flow down). More severe at the point of contact. Areas usually much lesser than burns. 100% scalds may occur if person falls in hot water	At or below the site of contact
3.	Edges	Deep burns may fade into more superficial burns; so sharply defined edges may not be present	Sharply defined edges usually present	Same as in moist heat
4.	Splashing and trickling	Absent	Present	Present
5.	Skin	Dry, wrinkled, charred (if burns are deep)	Sodden and bleached	Destroyed
6.	Vesicles	At the circumference of the burnt area	Over the burnt area. A line of blisters may be visible below the main point of contact, as streaks of hot liquid runs downwards.	Seen with specific chemicals only (eg vesicants), but absent in corrosives
7.	Red line	Present	Present	Absent
8.	Color	Black	Bleached	Distinctive
9.	Charring	Present	Absent	May be present
10.	Singeing	Present	Absent	Absent
11.	Ulceration	Absent	Absent	Present
12.	Healing	Does not form in superficial burns. In deep burns, thick and contracted scar forms	Thin and less contracted scar forms	Thick and contracted scar forms
13.	Clothes	Burnt	Wet, not burnt	Show characteristic stains
14.	Scene of crime	Shows evidence of burning, match box, kerosene bottle, burst stove etc	Shows liquid in a tub, which may be hot if scene examined fairly soon	May show evidence of corrosive burns on floor, walls etc

15. Explosion Injuries

I. INTRODUCTION

[A] **Explosions** can be (1) Natural [volcanic] (2) chemical (bombs) (3) nuclear (4) electrical (produced by a high current electrical fault) (5) magnetic (during

solar flares) and (6) mechanical (steam boiler bursts). Most commonly in medicolegal practice are chemical (bombs) and less commonly mechanical. [B] ***Explosion injuries*** are seen in bombings, the most common method of destruction employed by terrorists today. [C] **Molotov cocktail** (also known as a petrol bomb) has been a favorite of guerillas and terrorists. It consists of a **glass bottle partly filled with flammable liquid**, usually petrol (gasoline) or alcohol (generally methanol or ethanol). The mouth of the bottle is stoppered with a cork, and a cloth rag is fixed securely around the mouth. The rag is soaked in a flammable liquid immediately prior to using it. The rag is lit and the bottle thrown at the target. The bottle shatters on impact, spilling the flammable liquid over the target, which is then ignited by the burning rag. It is a common practice to throw several unlit, full bottles to saturate an area, then finish with a lit one. This avoids the risk of throwing several lit devices. If one uses lit bottles each time, the enemy would see the flaming bottles approaching and know which direction to start shooting. There is also the possibility of missing target or dropping the device after being lit. Many substances such as white phosphorus, tar, palm oil, acids etc may be added to the basic Molotov cocktail to enhance its use as a weapon. The bomb is named after **Vyacheslav Mikhailovich Molotov** (1890–1986) a Soviet politician, who first advocated it. [D] **Letter bombs** are explosive devices sent by mail through parcels. They consist of the detonating fuse, the explosive, the electronics for initiation and the energy source. The cause injuries – sometimes fatal – to the unsuspecting person who opens such parcels. [E] **Booby Traps** - A bomb designed to explode in the presence of a person, when he is engaged in activities like opening a door, picking something up [eg a policeman trying to pick up some incriminating object], switching something on. Often used in guerrilla warfare, and by criminals wanting to protect drugs or other illicit objects and property.

II. CLASSIFICATION OF BLASTS

Three types of blasts are seen (a) air blasts (b) underwater blasts (c) solid blasts

A. Air Blasts

Most frequently seen. Please see “Primary blast injuries” below.

B. Underwater Blasts

The blast in water differs from that in air, physically in that there is no after-wave of negative pressure. The most common organ suffering damage in **underwater blast** is **intestines**. Reasons: (a) Generally people affected are those who are taking bath. Only their abdomen is under water (b) They are wearing life jackets on their chests. (c) **Density of both water and body is same**. So most blast energy is transferred to the body, unlike the air blasts, where most energy is reflected. Ribs provide some kind of shield to this kind of blast energy. **There is no such shield on abdomen**.

C. Solid Blasts

Solid blast is seen when the blast forces a solid material against the body e.g. victim is standing on the relatively rigid deck of a ship when an underwater detonation occurs. In this case, the energy of the shock wave is transferred through the structure of the ship to the **feet of the victim**. The shock wave then travels upward through the inferior extremities resulting in **fractures of the bones of feet, ankles, knees** and **hips**. In addition, intraabdominal blunt force injuries without accompanying surface injuries may occur. The victim may also be propelled a considerable distance and strike intermediatry targets.

III. INJURIES DUE TO BOMBS

Injuries seen in explosions are as follows:

A. Primary Blast Injuries

Primary blast injuries occur due to the direct pressure effects of the blast wave on the victim [Barotrauma]. These are more severe when the blast occurs in a confined space, primarily due to repeated reflection of the blast wave. Organs most likely to suffer damage due to these injuries are those which contain air, e.g. **auditory apparatus, respiratory system, gastrointestinal system**. Blast waves tend to get reflected at air-fluid interphase, and since these organs contain such a boundary, they are more liable to get injured. The organ most sensitive to blast effects is the **ear**. Classic injuries seen in terrorist bombings are **rupture of**

the tympanic membrane, damage to the Eustachian tube, the ossicular chain and to the **organ of Corti** within the cochlea. While ear is the most sensitive organ to blast effects, **injuries to the lungs are the greatest cause of mortality**. Lungs would often reveal the fatal lesion in cases of deaths due to terrorist bombings. **Shock wave** passes to the lungs directly through the chest wall, as well as through the air via oronasal orifices. Main injuries seen are a widespread alveolar damage, tears in the visceral pleura, pulmonary hemorrhage, atelectasis, pneumothorax, hemothorax, pneumomediastinum and traumatic lung cysts. Air emboli are common which can be due to traumatic alveolar-venous fistulae. Subcutaneous emphysema and chest wall damage, including injuries to the ribs are also seen.

B. Secondary Blast Injuries

Secondary blast injuries are produced by flying missiles (nuts, bolts, nails etc) generated by the explosion. These injuries resemble classic ballistic wounds, except that the entrance wound is very irregular. Small missiles striking the body produce the **classic triad of punctate lacerations,**

A
brasions and

B
ruises (Marshall Triad). This triad is very characteristic of bombings. X-rays of victims should be taken to reveal them [detection of small metallic particles].

Memory Aid 1: Marshall triad

LAB

Laceration

Abrasion

Bruise

C. Tertiary Blast Injuries

Tertiary blast injuries are produced either when the victim is actually lifted up and thrown around by the blast wind, or when some ***heavy piece of masonry*** breaks and falls upon the victim. These injuries resemble the classic blunt force injuries.

D. Burns

These are sometimes called **quaternary injuries**. The characteristic burns seen

in explosions are flash burns. They are not due to flame, but due to radiant heat of high intensity. Since the duration of exposure is infinitesimally small, these injuries are **superficial in nature**. Furthermore, since the heat applied is the same, they are of **uniform depth**. If an object happens to be between the seat of explosion and the victim, it would cast its **"shadow"**, just as it would if there were a flash of lightning. Areas protected by clothing (bra, waste bands, belt, shoes) are safe.

E. Fumes

If the bomb explodes in a confined area, CO would be produced, and cause asphyxiation.

F. Explosive Injury

Typical injury seen in this category is the "**dust tattooing**", which occurs due to small particles of dust entering and lodging in the subcutaneous tissues. They cannot be washed off, since they penetrate inside the dermis.

G. Complete Disruption

If the victim happens to be sitting over the explosive device, or if he is in very close proximity, his body would be completely disrupted. The individual body parts are thrown wide apart. These bodies are most difficult to identify.

16. Electrical Injuries, Atmospheric Lightning, Radiation Injury

I. INTRODUCTION

Electrical injuries are produced by electricity. The damage produced by electrical injuries depends on:

A. Nature of Current (AC v DC)

(1) **Alternating current** (AC) is **4-5 times more dangerous** than direct current (DC).

(2) **Frequency – AC between 40 and 150 cycles** per second have the greatest lethality. One reason for the greater lethality of AC is that it induces tetanic contractions in the victim's hands, making it impossible to let go of electric wire. DC throws back the victim. In India, the domestic supply is **220-240 Volts A.C. at 50 Hz**.

B. Amperage v Voltage

Contrary to popular belief, it is the ***amperage***, which is the most important factor in determining the lethality of electric currents, and **not the voltage**.

C. Amount of Current

The amount of current in amperes is given by the formula **A = V/R**, where A is the current in amperes, V the voltage and R the resistance of intervening tissue in ohms. **Table 1** gives the effects of various amounts of currents on human body.

Table 1: Effects of various amounts of AC and DC on human body

mA (AC)	mA (DC)	Effects
1	1	Threshold of sensation
5	5	Tremor of hand and spasm of forearm
10	10	May involuntarily let go of electric line
20	20	Painful muscular contractions (tetany)
25	25-80	No permanent harm
25-80	80-300	Loss of consciousness, arrhythmias, respiratory spasm
80-100	>300	Irreversible ventricular fibrillation, death

D. Path of Current

Electric current runs from the **point of contact** to the **point of grounding**, following the shortest path (**Fig 16.1**). When current runs from arm to arm (**Fig 16.1(A)**) or from arm to leg (**Fig 16.1(B)**), the heart comes in way (may lead to ventricular fibrillation).

When current runs from head to arm or head to leg [**Fig 16.1(C, D)**], respiratory center comes in the way. This may lead to respiratory paralysis.

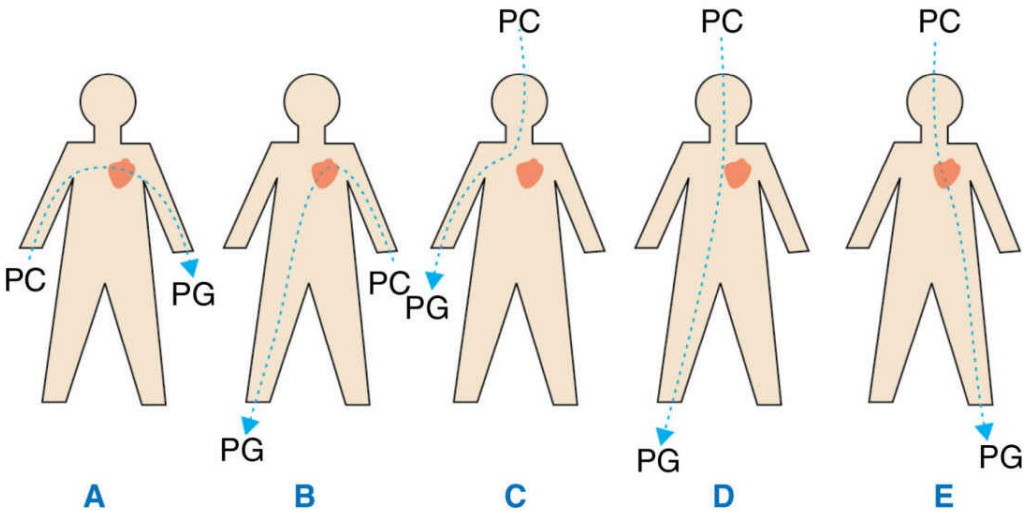


Fig. 16.1: Death may depend on the path taken by the current. This diagram shows the five most dangerous electric current paths. A – Right hand to left hand. B – Left hand to right foot. C-Head to right hand. D-Head to right foot [this path is used by the State of Florida and many other states for judicial electrocution]. E-Head to left foot. In A, B and E above, current passes through heart causing ventricular fibrillation. In C and D, it passes through brain stem, causing respiratory paralysis. PC - Point of contact; PG - Point of grounding.

E. Duration of Current

Joule's first law states (please see below) that heat produced is directly proportional to duration (t) of current.

II. TREATMENT

(1) **Scene safety** – If the victim is in contact with the source of electricity, he should not be pulled off with bare hands. The current should be switched off first. If this is not possible, rescuer must wear rubber shoes and gloves and then attempt to remove him

(2) **Defibrillation** – must be tried in low voltage currents

(3) **Emergency measures** - Give artificial respiration, closed chest cardiac massage.

III. CAUSE OF DEATH

Death due to electrocution depends on

(1) amount of current
(2) area of contact
(3) duration of current
(4) pathway of current [please see above]
(5) resistance of the body
(6) type of current and
(7) voltage. Low voltage currents are **<1000 v**. High voltage are **>1000 v**.

A. Low Voltage (household) Currents

Ventricular fibrillation. Victim may sometimes do some **volitional acts** (walking, talking etc) for **20-30 sec** before dropping dead. Ventricular fibrillation is occasionally self-reversible. The heart reverts to spontaneous rhythm following a short time of fibrillation. Thus in cases of low-voltage electrocution, resuscitation and defibrillation must be tried; this may prevent death.

B. High Voltage (industrial) Currents

Paralysis of the respiratory center (RC). RC is damaged by the hyperthermic effects of the current.

Memory Aid 1: Cause of death in electrocution

In **low voltage currents** organ at **lower level** is involved [heart];

In **high voltage currents** organ at **higher level** is involved [RC]

IV. SCENE OF DEATH

The examination of scene may be much more important than the post mortem

(1) **Photographs** - Photographic documentation of the body, scene and any nearby electrical devices or conductors.
(2) **Examination of any electrical equipment** (eg electric washing machine, cooler, electric iron etc) - found near the body by an electrical engineer or someone fully qualified in electrical assessment.
(3) **Electrical circuits** - Analysis of the electrical circuitry within, and

sometimes connecting to the building where the victim was found is required.

V. PM FINDINGS

If death occurs due to electricity, it is known as **electrocution.** The PM findings are as follows:

A. External

1. Electric entry mark

May or may not be present. If present, it is the most diagnostic PM finding of electrocution. Its absence ***does not rule out*** electrocution.

(1) **If absent** – (a) Prerequisites: (i) Large surface area from where current enters (ii) low skin resistance. Both these conditions are aptly met when electrocution occurs in the bathtub, or when a live electric wire is grasped with a wet hand (iii) Low voltage current also leaves no mark (b) Diagnosis - in such cases is entirely dependent upon the circumstances of death.

(2) **If present** – Three types may be seen as below:

a. Joule burn (endogenous burn)

i. General

Joule burn is typically produced in low voltage (household) currents, where heat is produced by conversion of electrical energy to heat energy within the tissues (hence called ***endogenous burns*** also). The name is after British physicist **James Joule** (1818 – 1889), who first worked out a relationship between current and heat produced, now known as **Joule's first law** or **Joule effect.** It is expressed as: $\mathbf{Q = I^2 \# R \# t}$ where Q is the heat (in Joules) generated by a current I flowing through a conductor of electrical resistance R, for t seconds [I and R are expressed in amperes and ohms respectively]. Most resistant tissue of the body is (i) **skin** (Table 2) followed by (in order of .ing resistance) (ii) bone (iii) fat (iv) nerve (v) muscle (vi) blood and (vii) body fluids. [cf, resistance to firearm ammunition offered by tissues (**ch 13**)].

Table 2: Electrical resistance of skin

Tissue	Resistance
Dry and calloused skin	10^6 ohms(Ω)
Dry uncalloused skin	10^5 ohms(Ω)
Moist skin	10^3 ohms(Ω)
Thin skin	10^2 ohms(Ω)

ii. Characteristics of Joule burn

(1) Appearance -(a) **Crater** [most common] (i) Round or oval shallow crater (ii) 1-3 cm in diameter (iii) has a ridge of skin about 1 mm high around part or whole of circumference (iv) Occasionally the mark may take the shape of the conductor [**Fig 16.2**] (v) Floor is pale (vi) Periphery may be blanched due to arteriolar spasm caused by current. Sometimes there may be a hyperemic border outside the blanched area. (b) **Broken blister** – Sometimes the skin may break at the site of electrical contact. It then resembles a broken blister. (c) **Incised wound** – In some cases, especially when a wire was involved, the skin would be linearly divided, mimicking an incised wound. Also known as **electric splits**. (d) **bullet wound** – Rarely, the mark may penetrate muscle and even bone, simulating a bullet wound. (e) If skin is wet, may not be visible. Becomes more apparent when the skin dries up. In one case, current marks were not discernible in a body lying in the rain, but became visible the next day after the body was moved and the skin dried out. (f) **Charring** – when there is more prolonged contact (g) **Metallization** – Electrical metallization in electrocution was first shown to occur by **Schrader** in **1932**. A number of methods were later devised to detect it (i) Color – of the electric mark depends upon the composition of the electrode (A) Aluminum – silvery (B) Copper – Reddish brown (C) Iron – Brownish black. The color is because of deposition of metal. The metalized skin desquamates within a few weeks. (ii) In rare cases, metallic coating may be seen on **nails** too. (iii) Metallization is a definite evidence that the burn was produced by electricity and not by flame. Even during touching of skin with hot metallic conductors (in the absence of

electricity), metallization does not take place. (iv) **Acroreaction test** - This test demonstrates metallization. (A) It is a much simpler microchemical test for metals (B) applicable for entry marks; not exit marks (C) Procedure - Treat metalized skin with acid (HCl or HNO_3). A portion of the acid is then taken with a tiny wedge of filter paper treated with a suitable reagent. Production of color is indicative of metal [e.g. potassium ferrocyanide detects iron by the production of Prussian blue]. (v) **Other tests** – (A) histochemical (B) microprobe analyzer (C) polarographic analysis (D) scanning electron microscope [SEM] (E) Spectroscopic (F) stereomicroscopic (G) transmission electron microscope and (H) variable-pressure scanning electron microscope (VP-SEM) with energy dispersive X-ray (EDX) microanalysis may determine the nature of the metal, which may be compared with the suspect electrode. These methods may be used even if decomposition has set in. (vi) Depth of penetration – Spectroscopic analysis has shown that while aluminum, magnesium, manganese, molybdenum and zinc penetrate the skin up to 3 mm, chromium, copper and iron penetrate up to 5 mm. No suitable explanation has been found for the difference in penetrating power.

(2) Site – (a) May be on palmar aspects of **fingers and hands** usually, as these areas come in contact with electricity mostly (b) Rarely on **genitals** or **anus** – in sexual perversions (c) May be **under intact clothing** or **under hair** – If under hair, may remain unnoticed. Scalp must be thoroughly palpated, and if any suspect lesion felt, hair carefully shaved from that area. (d) Inside **mouth** – (i) Accidental – when children place a live plug between their lips. Joule burn may be on tongue or buccal mucosa (ii) homicidal - in children. Done intentionally to hide from investigators

(3) Temperature – directly underneath the Joule burn may be as high as 95°C.

(4) Smell – of recent current marks resembles that of **burnt cork**.

(5) Histology – (a) Microblisters – The epidermis is elevated with microblisters developing within the squamous epithelium as well as in the external horny layer. These blisters result from the cooking effect on the tissue. (b) Electric channels – Cells are separated in the form of sharp slits. This pattern is known as electric channels or vacuolation. (c) Palisading and streaming of the nuclei – Nuclei of epidermal cells are stretched, elongated and placed at right angles to the dermis, to produce a palisade type appearance [streaming of the nuclei] (d) Collagen – Stains blue in ordinary H&E stain [due to its thermal

denaturation] (e) Nuclei of vascular media – are twisted so as to resemble spirals. This may occur at points quite distant from the site of contact with the electrode. They are localized in the arterial walls, because of blood being a good conductor of electricity. Indeed most current passes through the body via blood vessels. (f) Vascular intima – tearing of elastic fibres and overlying intima. May cause secondary thrombosis and gangrene.

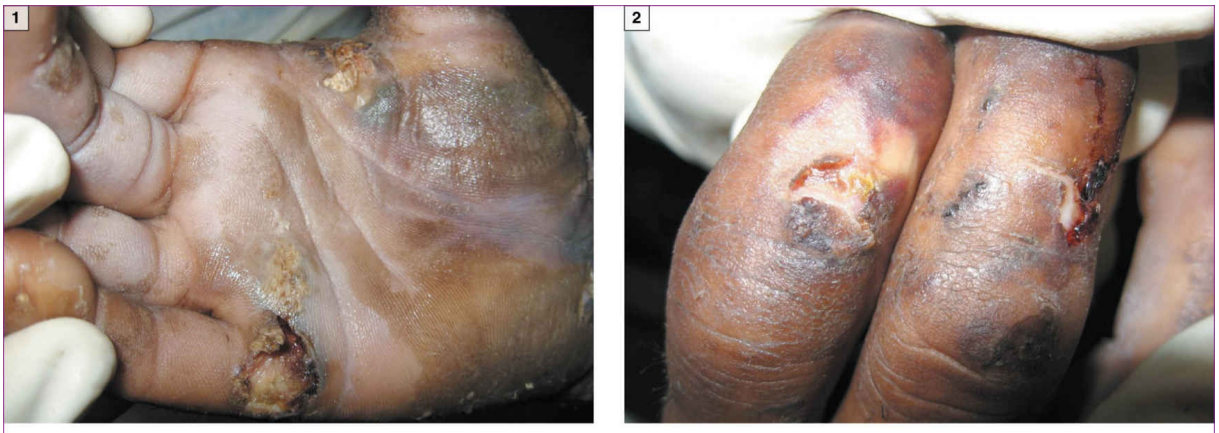


Fig. 16.2: **(1)** Joule burns over palm at the base of little finger and thumb. Between these two large Joule burns, a faint line of induration caused by the current can be seen. Please not blackening at the thenal eminence due to burns **(2)** Joule burns at the back of fingers

iii. Postmortem electrocution

Joule burns are not characteristic of antemortem electrocution. However there is no vital reaction in pm electrocution. Differences are given in **Table 3**.

Table 3: Differences between antemortem and postmortem electrocution

S.No	Criteria	Antemortem electrocution	Postmortem electrocution
1.	Vital reaction	Yes	No
2.	Expression of Heat Shock Protein-70 (HSP-70) mRNA and c-fos mRNA in skeletal and cardiac muscle	Yes	No

Fig. 16.3: Crocodile skin formed over chest, abdomen and left upper arm.

b. Flash or spark burn (exogenous burn)

Flash or ***spark burn*** is typically produced in high voltage (industrial) currents, when sparking occurs between the conductor and the victim. Also known as **exogenous burns**, because the source of heat is outside.

Table 4: The Distance An Electric Arc Can Jump

Voltage (in volts)	Distance Current Can jump
1,000	few mm
5,000	1 cm
20,000	6 cm
40,000	13 cm
100,000	35 cm

Salient features:

(1) **Crocodile skin:**

(i) High voltage currents can jump several millimeters across air and cause lesions [in dry air a current of 100 kV can jump upto 35 mm].

(ii) Such high voltage currents produce extremely high temperatures (upto 4000°C).

(iii) Causes keratin of the skin to melt over multiple small areas.

(iv) On cooling, molten keratin over these areas fuses into multiple hard brownish nodules [as resolidified wax], as in the skin of a crocodile (**Fig 16.3**) [another phenomenon seen due to keratin melting and resolidifying is clubbed appearance of hair tips in firearm wounds – **ch 13**].

(2) **Arc eye** - Electric arc produces heat and UV light. Clinically this produces a superficial and painful keratitis [arc eye]. If the patient survives, the onset of symptoms occurs 6-8 hrs after exposure. The characteristic features are

(i) intense bilateral lacrimation

(ii) blepharospasm

(iii) photophobia. The injury resolves spontaneously within 36 hours. Fluorescein staining of the eye [both in the living and at pm] reveals many punctate erosions of the cornea.

c. Electric splits

(1) In some cases the point of entry shows lacerations in the form of **electric splits**
(2) They may be round, oval, linear or of irregular shape
(3) Skin is shed; some of this is found attached to the conductor
(4) Wrinkling of skin may be present

2. Electric exit mark

(1) **Site** - This mark appears where the body was earthed or 'grounded' [**Fig 16.4**].
(2) **More damage of tissues** than at entry marks
(3) Often seen as splits.

Fig. 16.4: Electric exit marks on foot, where the body was 'grounded'

3. Other external findings

(1) **Face** – pale
(2) **Eyes** – (i) Congested (ii) Pupils – dilated
(3) **Rigor mortis** – appears early
(4) **PM Lividity** – well developed
(5) **Seminal ejaculation** [**Fig. 16.5**] – Due to contraction of seminal vesicles caused by electrical flow through body.
(6) **Secondary burning** – may be present due to ignition of clothing
(7) Additional findings in **high tension electrocutions** –(a) Charring of the body (b) Amputation of extremities.

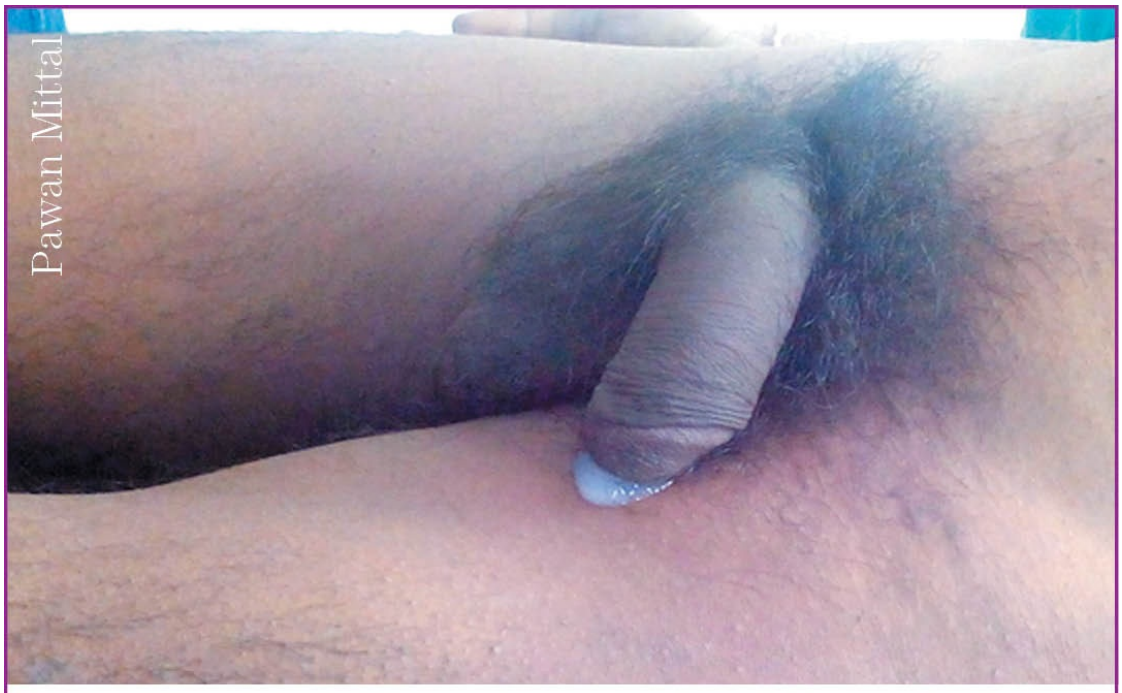


Fig. 16.5: Seminal ejaculation in a case of death due to electrocution.

B. Internal

(1) **Congestion** – of all internal organs.

(2) **Petechial hemorrhages** – along the line of passage of current. Depending on route, may be present in brain, spinal cord, endocardium, pericardium and pleurae.

(3) **Brain** – Irregular tears and fissures

(4) **Muscles and tendons:**

(i) Zenker's degeneration - of skeletal muscles in the path of current [severe glassy or waxy hyaline degeneration of skeletal muscles. Grossly the muscles appear pale and friable; microscopically, the muscle fibres are swollen, have a loss of cross striations, and show a hyaline appearance. Rupture and small hemorrhage are also seen].

(ii) Release of myoglobin – Myoglobin is released into general circulation. This

may cause renal shutdown, as occurs in extensive crush injury [please. see ch 11 on injuries also, under the heading “Crush syndrome”].

(5) **Bones and joints:**

(i) Bone pearls [syn osseous pearls, wax drippings, wax pearls]– (a) Seen in high voltage currents. (b) Mechanism - (I) Heat generated by current melts calcium phosphate. "seen radiologically as typical round density foci (II)Vaporization within bone"seen on the surface of bone with naked eye as grayish white, hollow nodules of the size of a pea

(ii) Necrosis of bone

(iii) Fractures – (a) due to violent muscular contractions or falls. All electrocution deaths should be radiographed for this reason. (b) fractures may be hair thin [microfractures]. May be missed on X-ray. Have an irregular zigzag course [osseus schisis] (c) Bones most commonly fractured in upper limb [in order of frequency]- (I)coracoid process (II)head of humerus (III)neck of humerus (IV)ulna (d) Bone most commonly fractured in lower limb - neck of femur (e) Fracture of spinal processes. It is postulated that these regions are susceptible to fractures due to the large bulk of surrounding muscle [and thus stronger tetanic contractions]. A disproportionately large number of the fractures are bilaterally symmetric, suggesting again susceptibility of certain bones within a relatively extensive path of electrical current. (f) Late bone injuries – are seen due to tissue ischemia from diminished or obstructed blood supply

(iv) Electrical injury of skull – (a) Occurs due to arcing when transformer and switch operators and persons working on utility poles inadvertently touch the cable with vertex of the head. (b) Skull may be split open. (c) Orbital contents extruded eye sockets. (d) Effects attributed to boiling of the brain and formation of steam within skull. (e) Decapitation may also occur.

(v) Subluxation of joints – especially shoulder joint

(6) **Current pearls** - Small balls of molten metal, derived from the metal of the contacting electrode is carried deep into the tissues.

(7) **Heart:**

(i) Auricles - Dilated

(ii) Petechial as well as larger hemorrhages – on endocardium, pericardium and myocardium, particularly at the base of aortic cusps.

(iii) H/P - Fragmentation, rupture and twisting of myofibres, interstitial edema

and foci of colliquative necrosis

(iv) Electron microscopy – detachment of the external myocardial membrane

(8) **Vascular:**

(i) Microscopic lesions in media

(ii) Intima is relatively protected due to cooling effect of circulating blood

(iii) massive coagulation necrosis of the entire vessel wall

(iv) Hemorrhage and thrombosis. May occur immediately or up to 1-6 weeks after the accident. May lead to thromboembolism

(v) Tissue edema [edema electricum] due to immediate and late vascular injury

(9) **Kidney and urine:**

(i) Lower nephron nephrosis [due to myoglobin entering nephrons]

(ii) Urine – shows myoglobin

(10) Fetus – If pregnant female receives electric shock, abortion may occur. Sometimes fetus may survive too.

(11) Additional findings in **high tension electrocutions** – (a) Viscera – may be ruptured (b) Bone - (i) Periosteum elevated (ii) Fractures

(12) **Electroporation** - Injury of tissue through direct cellular Damage.

VI. ML ASPECTS OF ELECTRIC INJURIES

(1) **Electric crematorium - ch 6**.

(2) **Electroconvulsive therapy** [ECT] - fractures may be sustained during it, bringing allegations of negligence against the doctor. Most famous example of this is the **Bolam case** [**ch 2**].

(3) **Manner of death:**

(i) Accidental - Virtually all electrocutions are **accidents**, with suicides rare and homicides even rarer. (a) Accidents occur mostly when live electric wires are touched, especially when the victim is barefooted and the ground is wet. A case is recorded where a 14 y old boy suffered a fracture of radius from his **electric guitar**. He had been playing it when he reached to turn on a floor lamp which was improperly grounded. The boy felt a sudden shock and noted that some of his guitar strings “popped” and burned his arm. Physical exam revealed a small linear burn on his proximal forearm, soft tissue swelling and tenderness of his distal forearm. A plain radiograph of his right forearm revealed a buckle-type fracture of the radius. (b)

Accidental electrocution during autoerotic practices – Can occur. A case of death has been described where one electrode was attached to the anus; an attempt was being made to attach the other to the penis, when accidental electrocution occurred. Death occurred by myocardial fibrillation. Pornographic literature around the scene had been hidden by the parents to avoid embarrassment. It was discovered by the investigators.

(ii) Suicidal - (a) uncommon method of suicide. (b) Used by both males and females, but predominantly by males (c) Suicidal electrocution can be staged to mask a homicide by other means (d) Methods - (I) Climbing a pylon and grasping the wire carrying high voltage current (II) Victim fasten one end of a long wire to one of the limbs (usually the wrist)"throws the other end of the wire (weighted by a heavy object) over the transmission line (III) Victim winds the wires around his waist or limbs, plugs wires in domestic electrical supply and switches on the current. (IV) Miscellaneous – placing the end of an electric line in the mouth; Using an electric saw to simulate an industrial accident.

(iii) Homicidal - (a) Homicide by electrocution is rare (b) Methods -(I) Live wire is placed across a road where the prospective victim is expected to walk or drive (II) Setting up an electrical circuit within an object that victim is expected to touch (III) Putting a live wire, or a defective electrical appliance in the bath tub as the victim is taking a bath. (IV) By simply tricking the victim - A woman and her young son were homicidally electrocuted in **1980** by the ex-husband of the woman. He deceived his ex-wife by telling her that he was playing a harmless game where a bulb could be lit when placed on her bare skin. Thus, he persuaded her to be part of the circuit. He then wound flexible conducting wires around the limbs of the victims and electrocuted them by switching on the current.

(4) **Judicial Electrocution** – is the method of imparting death penalty in some states of the US. For all official methods of judicial execution, please see **ch 40**. (a) **Electric chair** - The condemned criminal is strapped to a wooden chair (sometimes called “the Old Sparky”). (c) **PM findings** - (i) **Temperature** - Beneath the electrodes, the skin and brain temperature may rise as high as 60°C. (ii) **Histologically** rupture of neuroaxons and blood vessels of the brain occurs. (iii) Right leg immediately goes into **cadaveric spasm**. (iv) Often **ejaculation** of semen occurs.

(5) **Pregnancy and electric shock**– If a pregnant mother receives an electric shock, the fetus may be **aborted**. It may lead to claims of **civil compensation**, if the shock was received during work through faulty machine. (a) **Mechanisms** (i) Fetal skin offers 200 times less resistance than does skin of a newborn(ii) Both **hyperemic pregnant uterus** and **amniotic fluid** are excellent good conductors (iii) **Uterine contractions** – are induced by electric current, which lead to premature expulsion of fetus. (b) **How to say positively that fetus died due to electric current** – (i) Pregnancy was **normal** before electric shock (ii) There was **no history of abortion** in earlier pregnancies (iii) Electric shock **proven** (iv) **No other factor** can be explained as being responsible for abortion (v) Stage of development of fetus corresponds to the stage of pregnancy and degree of **maceration** is proportional to the time elapsed between electric shock and death of fetus. (c) **Other significant points** (i) **Current enough to kill fetus** – 25mA for 0.3 secs [AC] (ii) **PM findings on fetus killed by electricity** – [A] Dilatation of the heart chambers [B] hemorrhages in brain, kidneys and liver (iii) **Criminal abortion** – has been tried via electricity. (iv) **Electroshock therapy of pregnant mother** – can cause [A]abortion [B]temporary disturbance of fetal heart rhythm. The doctor may face negligence suit in both. In the latter, it has been alleged that the fetus suffered late ill effects due to disturbance.

(6) **TASER** - A TASER (***T***homas ***A***. ***S***wift's ***E***lectric ***R***ifle) is an electroshock weapon [an electrical gun] that uses electrical current to disrupt voluntary control of muscles.

VII. LIGHTNING

Lightning is an atmospheric discharge of electricity accompanied by thunder, which typically occurs during thunderstorms, and sometimes during volcanic eruptions or dust storms. Lightning is a natural phenomenon which occurs following a massive buildup of electrostatic charge in cloud formations [particularly cumulonimbus clouds]. It is estimated that a lightning flash occurs approx **8 million times/day** throughout the world. As ice particles form within the clouds, +ve and -ve charges build up in the upper and lower levels of cloud respectively. The -ve charge at the base of cloud creates a corresponding +ve charge on the ground underneath. When enough voltage is built up, charge

begins to flow in the air down to earth. This is seen as a visible lightning flash. Discharges occur within the cloud too.

Salient Features:

(1) **Speed** - In the atmospheric electrical discharge, a leader of a bolt of lightning can travel at speeds of 220,000 km/h

(2) **Frequency** - There are some 16 million lightning storms in the world every year.

(3) **Time** - required for passage to earth lies between 0.001 and 0.0001 s [10^{-3}-10^{-4} s]. Flashes lasting as long as 1 second have been reported and are called "hot lightning." These are more dangerous than the short flashes.

(4) **Electrical charge:** (i) The electrical discharge is between a negatively charged cloud and a positively charged object on earth [negative lightning]. 5% of lightning flashes are from positively charged clouds [positive lightning]. Positive lightning tends to occur during winter, during tornadoes, during violent thunderstorms and in mountainous regions. It tends to be more powerful than negative lighting and might have a different injury profile. (ii) Attracted at the highest points [this is the principle of lightning conductors]

(5) **Temperature** – is about 50,000°C

(6) **Amperage** - Lightning produces very high currents [between 12,000 and 200,000 Amperes]

(7) **Voltage** - is equivalent to a 1 million volts DC. However up to 1000 million volts has been described

(8) **Energy dissipated** in a typical lightning flash: (i) 500 million Joules. (ii) Conversion of this energy - occurs into (a) Mechanical energy - leads to massive movements of air ["wind" of lightning; causes mechanical trauma to victims also]. (b) Sound Energy – energy from lightning heats the air up to 18,000°F. This causes a rapid expansion of the air causing **thunder** (c) light – seen as a flash.

A. PM Appearances

1. Clothes

(1) Usually burnt, especially at the point of entrance or exit. Body may or may not show associated burns

(2) Torn [due to "wind" of lightning,]

(3) May be stripped off the body and thrown to some distance

(4) Rarely may remain undamaged
(5) Melting belt buckles and zippers
(6) Shoes may be ripped apart
(7) **Objects in the pockets:**
(i) Metallic objects - [eg coins, medallions, nail cutters, pen-knife etc] may be found molten, fused together or even magnetized [cf H_2S, where they are blackened and serve as a clue to exposure – **ch 44**].
(ii) Plastic objects - eg pens, credit cards, cell phones may be completely molten and distorted.
(iii) USB drives - become completely useless.

2. External

Keraunopathology [Gk keraunos, a thunder bolt] deals with pathological changes seen in lightning injuries.

a. Burns

Except surface burns, all are due to heat generated from within the body [endogenous burns] rather than being applied from the outside. Burns of the body with no accompanying heat damage to overlying clothes is common in lightning and indicates endogenous burns. One of the earliest instances was described in **1680**. After a lightning incident in Westmoreland (present-day Cumbria), one woman suffered burns of the feet at the heel and toe. Her stockings and shoes were completely unharmed.

i. Endogenous burns

(1) **Linear burns:**
(i) Site: moist creases and folds of skin, eg such as beneath the breasts, down the mid-chest, and in the midaxillary line.
(ii) Length: varies from 3-30 cm or more
(iii) Width: 0.3-2.5 cm
(iv) Depth: usually first and second-degree burns
(v) Time of development: may be present initially or develop as late as several hours after the lightning strike. Late burns are most likely steam burns secondary to the vaporization of sweat or rainwater caused by the flashover phenomenon on the victim's skin.
(2) **Punctate burns** – multiple, closely spaced, discrete circular burns
(i) diameter - ranges from a few mm to 1 cm. Usually too small to require grafting in case of survival

(ii) Depth – may be full thickness and resemble cigarette burns.

(iii) Tip-toe sign [**Fig 16.6**]- Small, circular, full-thickness burns involving the sides of the soles of the feet and the tips of the toes. This is the exit mark of lightning.

Fig. 16.6: Tip toe sign in lightning.

(3) **Arborescent burns** [**Fig 16.7**] – [syn, arborization, feathering, ferning, filigree burns, keraunographic markings, keraunographism, Lichtenberg's flowers lightning marks]. These are irregular, superficial, thin, tortuous markings on this skin resembling the general pattern of a fern or tree. These are the most characteristic findings in lightning. There are references to these markings in the **Bible**, in which they were described as representing photographic imprints of the victim's surrounding vegetation.

(i) Site – most common over shoulders, front of chest, flanks

(ii) Usually pink to brownish in color and are sometimes lightly palpable.

(iii) Time of development – Not certain if produced instantaneously or after some time.

(iv) Not associated with burning. There is no associated damage to the skin.

(v) Seen only in 20% - 33% cases.

(vi) Do not lie either along vascular channels or nerve pathways

(vii) Histopathology - shows no pathological changes in epidermis, dermis, nerve, or collagen. The absence of H/P changes indicates that they are purely a physical phenomenon, and not a class of burn.

(viii) disappear in about 1-2 days among survivors. Sometimes after death also.

(ix) Mechanism of formation – There are several theories (a) break down of RBCs due to high current"Hb stains surrounding tissues. The lysis occurs not within vascular channels, but along the path of electric current. This accounts for markings not corresponding to vascular channels. Disappearance is accounted for by pigment dissipating away with the passage of time. Most acceptable theory. A similar branching which truly corresponds vascular channels is seen in burns [**ch 14**]. (b) Minute depositions of copper – in dermis (c) Boiling of intercellular fluid following fascial planes (d) These are representation of static electricity discharges along superficial vasculature and nerves (e) inflammatory response – elicited by electron showers (f) current following the lines of moisture in skin creases. (g) The figures are fractals [a mathematical concept]. Many natural features exhibit fractal like shapes [eg coastlines].

Fig. 16.7: Arborescent burns seen in lightning.

ii. Exogenous burns

Surface burns – These are burns caused by external heat. May occur if the victim's clothing is ignited or if the victim was wearing or carrying metal. Necklaces, belt buckles, rings, pocket change, and other such items in the grasp or in contact with a lightning strike victim can lead to second- or third-degree burns to the adjacent skin as the objects become heated rapidly by the electric energy. Metallic objects may be fused due to excessive heat. Hair may be singed

b. Mechanical lesions

Blast like, disruptive lesions. Due to expanded, displaced air (i) Contusions (ii) Fractures [Skull, facial, limb] (iii) Lacerations [external and internal organs] (iv) Wounds [all kinds] (v) Ears - (a) hemotympanum (b) rupture of tympanic membrane

3. Internal

(1) **Brain:**

(i) Shows congestion, edema

(ii) Hemorrhages - Epidural, SDH, SAH, intracerebral hemorrhage, Peri and intraventricular hemorrhage, Basal ganglia hemorrhage [direct or thermal], brainstem hemorrhages

(iii) Leptomeninges – congested, lacerated

(iv) Coagulation of parenchyma

(v) Postcardiac arrest hypoxic–ischemic encephalopathy and associated cerebral infarction

(2) **Lungs:**

(i) Congested

(ii) Patches of hemorrhages underneath the pleura

(iii) Pulmonary contusion [blast effect]

(3) **Heart** - Myocardial infarct (rare); myocardial contraction bands

(4) **Muscle** - necrosis, myoglobinuria, renal failure (rare compared with high-voltage AC electrocution

(5) **Spinal cord** – damage. Direct or secondary to fracture

(6) **Eyes and ears:** (i) cataracts (ii) corneal edema (iii) secondary otitis media; (iv) Tympanic membrane perforation; (v) uveitis; (vi) vitreous hemorrhages.

B. Cause of Death

(1) Cardiopulmonary arrest- most common cause

(2) Damage to CNS

(3) Electrocution

(4) respiratory center paralysis

(5) thermal injuries

C. ML Importance

(1) Fatality-<50% people struck by lightning are killed.
(2) Manner – always accidental
(3) **Keraunomedicine** deals with medical aspects of lightning injuries.

17. Regional Injuries

I. INTRODUCTION

Epidemiology:

(1) Age - Most injuries are commonly seen between 20–35 y
(2) Sex – More frequent in males.

II. HEAD INJURIES

A ***head injury*** is any trauma that leads to injury of the ***scalp***, ***skull***, or ***brain***. The injuries can range from a minor swelling on the scalp to serious brain injury. No case of head injury should be taken lightly. All cases of head injury should be **hospitalized for at least 24 hours** for observation.

Memory Aid 1: Age determination of antemortem tooth loss

There are total **7 stages** – 2 in days; 3 in weeks; 2 in months. Now write them down as below.

- **1st stage** – only fresh blood;
- **2nd and 3rd stages** – deal with organization;
- **4th stage** – fibrous;
- **5-7th stages** – deal with bone.

ü **7 numbers to remember are – 0, 1, 1, 2, 3, 6, 12**.

ü These are easy to remember because they are in a logical sequence. **Q in superscript denotes a possible viva or MCQ question**.

Only fresh blood

(1) **0 d [Fresh]** – fresh blood

Main focus - organization

(2) **1 d** – Early organization
(3) **1 w** – complete organization^{Q}

Main focus - Fibrous

(4) **2 w** – clot replaced by **fibrous** tissue[Q]

Main focus – bone formation

(5) **3 w** – partial bone [socket partially filled with bone][Q]

(6) **6m** – bone cont'd [filling with bone continues]

(7) **12 m (1 y)** – bone complete

A. Skull

1. Fractures

a. Factors influencing skull fractures

Skull fractures depend on thickness, density and elasticity which varies from person to person.

Thickness

(1) Thickness of skull -

(i) **Average thickness - 6 mm**. Different skull bones are of different thickness though.

(ii) Occipital bone is thickest at 15 mm

(iii) Frontal bone - 8 mm;

(iv) Parietal bone - 7 mm;

(v) Temporal bone - 4 mm.

(vi) Skull is thinner where it is protected by thick muscles.

(vii) Buttresses - Skull is thicker in the midfrontal, midoccipital, parietosphenoid and parietopetrous buttresses.

Memory Aid 2: Thickness of skull bones (from thickest to thinnest)

After **Of**fice to **part**y

Occipital"Frontal"Parietal"Temporal

(2) The thickness of **outer table of skull** is **twice** that of inner table.

(3) The inner table is **thinner** and **more brittle** than the outer table which is more resilient.

(4) The skull is thicker in some races (thicker in Negro than in Caucasians). In all races, bones of the skull are thinner in women and children than in men.

b. Signs of skull fractures

i. Halo sign, ring sign

Clear, pink, watery CSF will leak from the nose [in ACF fracture] or ears [in

PCF fracture]. Drops of CSF on the white sheet wrapped around body form a pink ring around a slightly blood-tinged center, resembling a target [halo sign, ring sign]. Seen in live patients also around pillowcases, handkerchiefs etc.

ii. Battle's sign

(1) After English surgeon **William Battle** (1855 – 1936), who first described this sign.

(2) Ecchymosis over the mastoid indicates **fracture of posterior cranial fossa**).

(3) Results from extravasation of blood along the path of the **posterior auricular artery.**

c. Types of skull fractures

Skull fractures may be classified according whether they communicate with the outside environment or according to their site and appearance.

i. According to communication with outside environment

Fractures of the skull may be (a) simple or (b) compound. In ***compound skull fractures***, the cranial cavity comes in contact with the outside environment increasing the risk of contamination and infection. It happens when there is a laceration over the fracture or when a nasal sinus is fractured along with. In simple fractures there is no such communication.

ii. According to site and appearance

(a) Comminuted fracture

Comminuted fracture (comminuted:"to reduce to powder", "to pulverize"; from L. minuere, "to lessen") is one in which there are two or more intersecting lines of fracture which divide the bone into three or more fragments.

Salient features:

(1) **Caused by** - (i) Fall from height on a hard surface (ii) hoof of an animal [animal kicks] (iii) vehicular accidents (iv) weapons with a **large striking surface** [axe, cricket bat, heavy iron bar, hockey stick, poker]

(2) Often resembles a **spider's web** or **mosaic** [Spider web fracture or mosaic fracture].

(3) If the fragments are pushed inwards, it would be called a **depressed comminuted fracture**. Sometimes small pieces may be lodged in brain

(4) lines of fissured fracture are often seen radiating outwards from comminuted fractures.

(b) Depressed fracture

Depressed fracture is a fracture, in which the fractured bone is driven inwards into the skull cavity.

Salient features:

(1) Produced by **local deformation** of skull

(2) **Causes:**

(i) Weapons – Typically produced by heavy weapons with a **small striking surface** [eg axe, chopper, hammer, spanner, stick, rock, stone]

(ii) Other causes – (a) Fall on a sharp corner of a piece of furniture

(3) Outer table is driven into the diploë and inner table shows comminuted fracture. Rarely only the inner table may be involved, leaving the outer table intact

(4) **Depth** – of the fracture is directly proportional to the force of impact

(5) ***Signature fracture*** [syn, fractures à la signature]- is a special variant of depressed fracture in which the shape of fracture "mirrors" the striking surface of the weapon. Medicolegally they are very important because they can reveal the weapon.

(6) **MLI** -

(i) Identification of weapon from signature fractures

(ii) Compression of brain"severe dysfunction, coma and death.

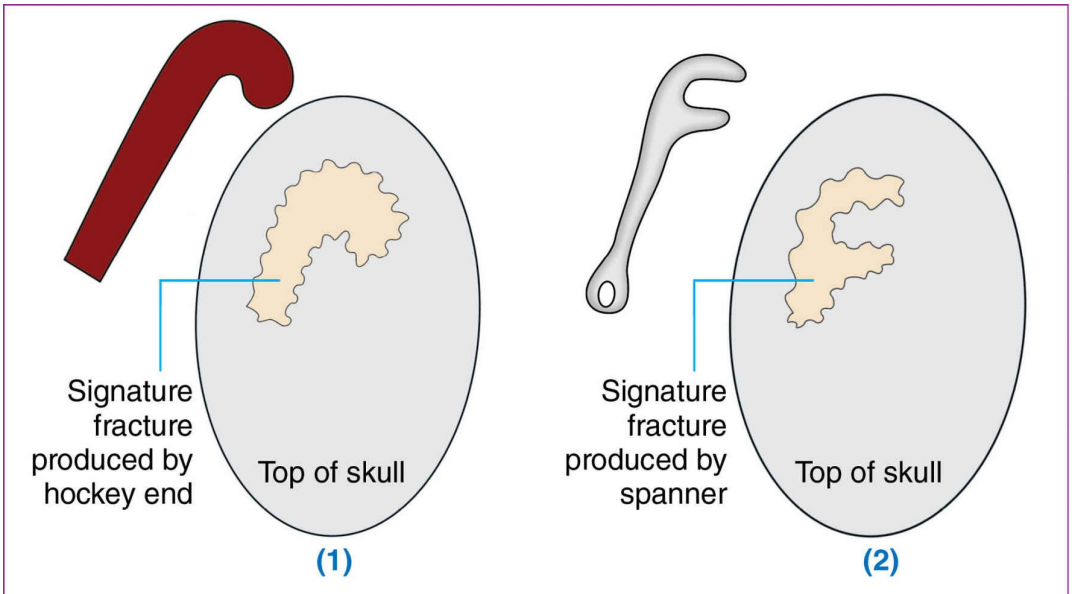


Fig. 17.1: Signature fracture produced by **(1)** hockey **(2)** spanner

(c) Diastatic (sutural) fracture

Diastatic fracture [Gk, dia, apart; histanai, stand] is the traumatic separation of cranial bones at a suture line.

Salient features:

(1) **Age** - Seen only in young persons [because of non union of sutures]

(2) **Suture involved** – Most commonly, sagittal suture [**Fig 17.2**]

(3) **Cause** – blunt trauma

(4) **Association with other fractures** - Generally occurs along with other fractures, but may be seen alone.

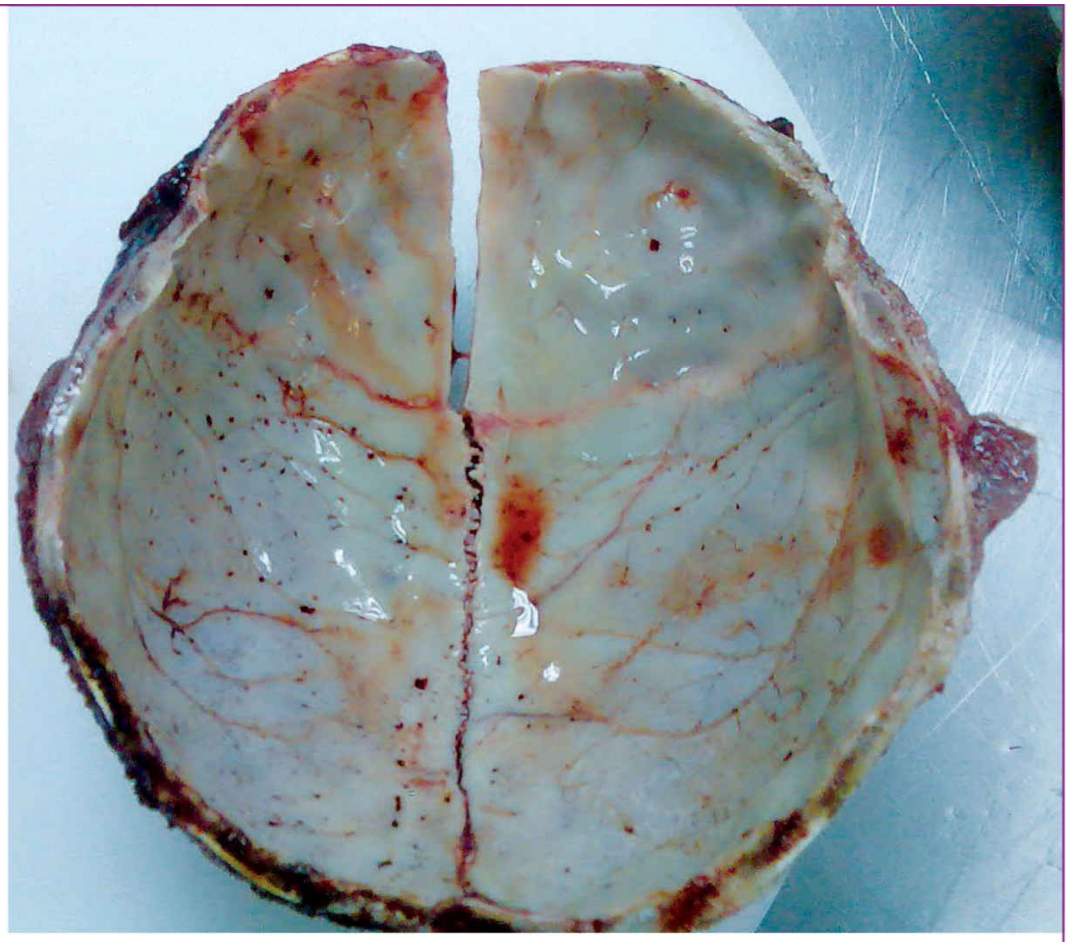


Fig. 17.2: Sutural fractures.

(d) Elevated fracture

(1) Skull pieces are pushed outwards.

(2) Very rare.

(3) **Causes:**

(i) a long, sharp wounding object [eg machete] elevates the fracture fragment by a lateral pull of the object or rotation of the head.

(ii) Through and through penetration of skull. The exit wound causes elevated fracture [**Fig 17.3**]

(iii) Elevation of free fragment while retrieving the weapon

(iv) Tangential force applied to the calvarium associated with rotation of head.

(v) Burns – pressure of gases from inside cause outward displacement of skull fragments [**ch 14**].

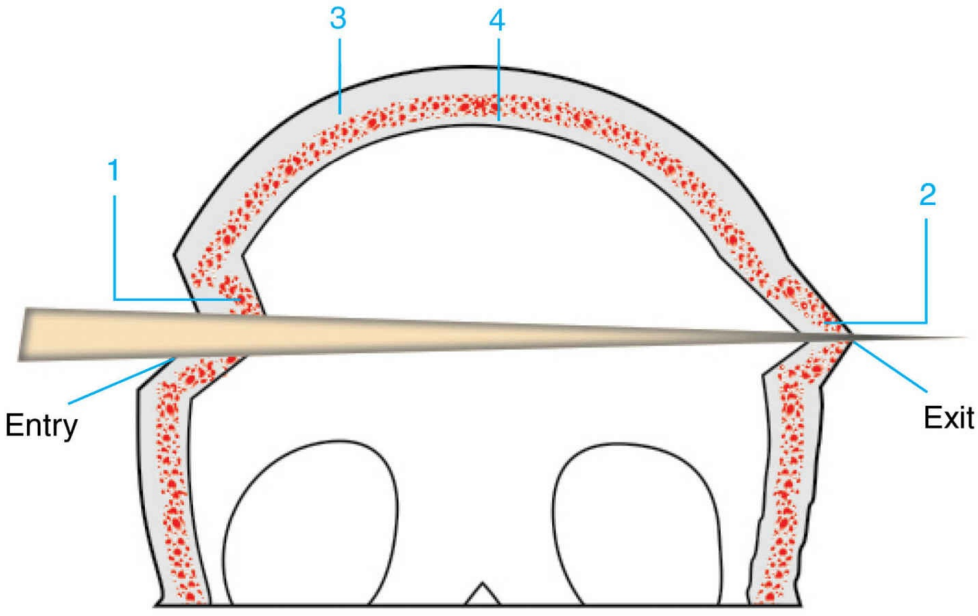


Fig. 17.3: (1) Depressed and **(2)** Elevated fractures of skull **(3)** Outer table **(4)** inner table.

(e) Fissured fracture

Fissured fracture is a linear fracture involving the whole thickness of skull, or only the inner or outer table.

Salient features:

(1) **Frequency** – Commonest type of skull fracture

(2) **Mechanism:**

(i) General deformation of skull - caused by forcible pressure against a broad resisting surface, as in (a) blows with weapons with a **large striking surface** and (b) falls on ground or flat surface – one or several fracture lines are seen radiating from the point of impact; depression of bone fragments

may not be seen

(ii) Movement of head - (a) Head is free to move - fracture starts at the point of impact (b) Head is fixed – If head is fixed [as when the person is sleeping on ground], the head is "sandwiched" between weapon and the hard surface. In such cases, fracture line may start at a point opposite the point of impact. Sometimes the skull may get "ballooned out" between the two pressure points, and fracture line may start at the base of skull [commonly] or at the vertex [less commonly]. The line of fracture runs parallel to the axis of compression

(3) **Association with other fractures** –

(i) May be found alone or associated with other type of fractures

(ii) May be found only on the inner table. This happens because after impact, the more resilient outer table rebounds back to its original shape, while the more brittle inner table fractures.

(4) **Site of fracture** – may be indicated by an extensive hematoma of scalp.

(5) **Characteristics of fracture line:**

(i) Breadth - hair's breadth.

(ii) Course - irregular. Sometimes it reaches a suture"follows its course for a while"then starts again its own way.

(iii) Hemorrhage - almost always present. This makes the fracture line look red. This is also a useful way to differentiate between AM and PM fractures. Red color can also be used to detect fractures at autopsy [**ch 5**].

(6) **Determination of cause** – As a broad rule fracture caused by fall are at the level of pinna, while those due to blows are above this level. Injuries at top of head may indicate direct force over head, or fall.

(f) Growing fracture

Growing skull fracture (***syn***. GSF, craniocerebral erosion), is a rare complication of skull fractures seen mainly during first 3 years of life, and is characterized by progressive enlargement of the fracture line.

Salient features:

(1) **Incidence** – 0.05 to 1.5% of skull fractures in childhood.

(2) **Age** - More than 50% of the GSFs occur under the age of 12 months and 90% under the age of 3 years. Virtually never found after 8 yrs of life.

(3) **Bone involved** - Usually located in the parietal bone.

(4) **Separation** – Usually seen in fractures, where the separation at the time of

injury is > 4 mm.

(5) **Time when enlargement occurs** - 4 to 8 weeks later.

(6) **Why occurs during young age group** – (a) Interposition of dura in fracture - Dura adheres more tightly to the bone in this age group and therefore is more easily torn in skull fractures. Part of it gets interposed in the fracture line preventing osteoblasts from migrating to the fracture site, inhibiting healing [most important cause] (b) Interposition of epidural hematoma in a similar way (c) Lack of nutrition - Dura acts as a periosteum of the skull bones, the latter's blood supply coming mainly from dural vessels. Laceration of dura with or without its interposition between the fractured pieces of skull interrupts its blood supply causing nutritional deficiency of the skull bones. This results in enlargement of the skull defect. The enlargement starts from the inner table (d) Continuous pressure - from tissue herniation through the bone gap. (e) rapid growth of skull - Skull grows very rapidly during this period, preventing stability needed for fracture union (f) Raised intracranial pressure.

(g) Gutter fracture

Gutter fracture is a long, narrow, fracture of the skull, produced by partial removal of the outer table.

Salient features:

(1) **Causes** – most commonly due to a glancing bullet [**Fig 17.4**, **Fig 17.5**].

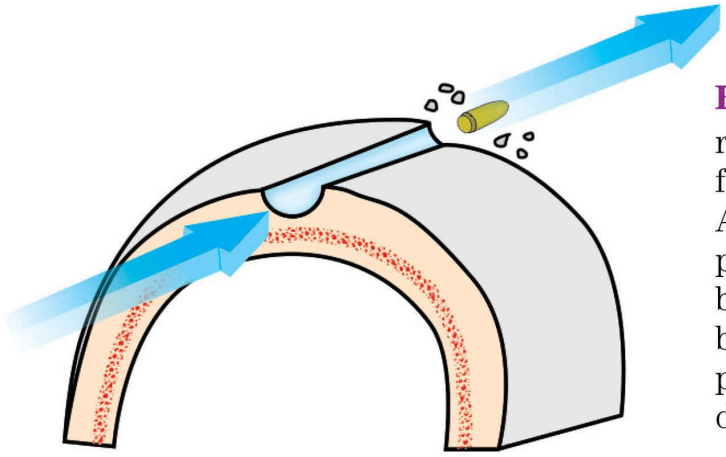


Fig. 17.4: Diagrammatic representation of gutter fracture [1st degree]. Arrows represent the path of the glancing bullet which chips off bone from the outer table producing a gutter like defect in the skull.

Memory Aid 3: Gutter fracture

Gutter # is produced by **G** lancing bullet.

(2) **Types:** (i) First-degree gutter fracture - Only the outer table of the skull is

grooved by the bullet, with resultant carrying away of small bone fragments. (ii) Second-degree gutter fracture - In addition to above, pressure waves generated by the bullet fracture the inner table [Irregular depressed fracture]. Dura mater and brain may be torn (iii) Third-degree gutter fracture - It is synonymous with keyhole fracture [please see below]. (iv) Bullets at a still lower level cause “perforating wounds” where there is production of separate entrance and exit wounds in the bone.

(h) Keyhole fracture

The bullet perforates the skull in the center of the tangential wound [**Fig 17.5**]. The outer table is fragmented, and there are depressed fragments of the internal table if not comminution and pulverization of both tables in the center of the wound track. Fragments of bone can be driven into the brain causing death.

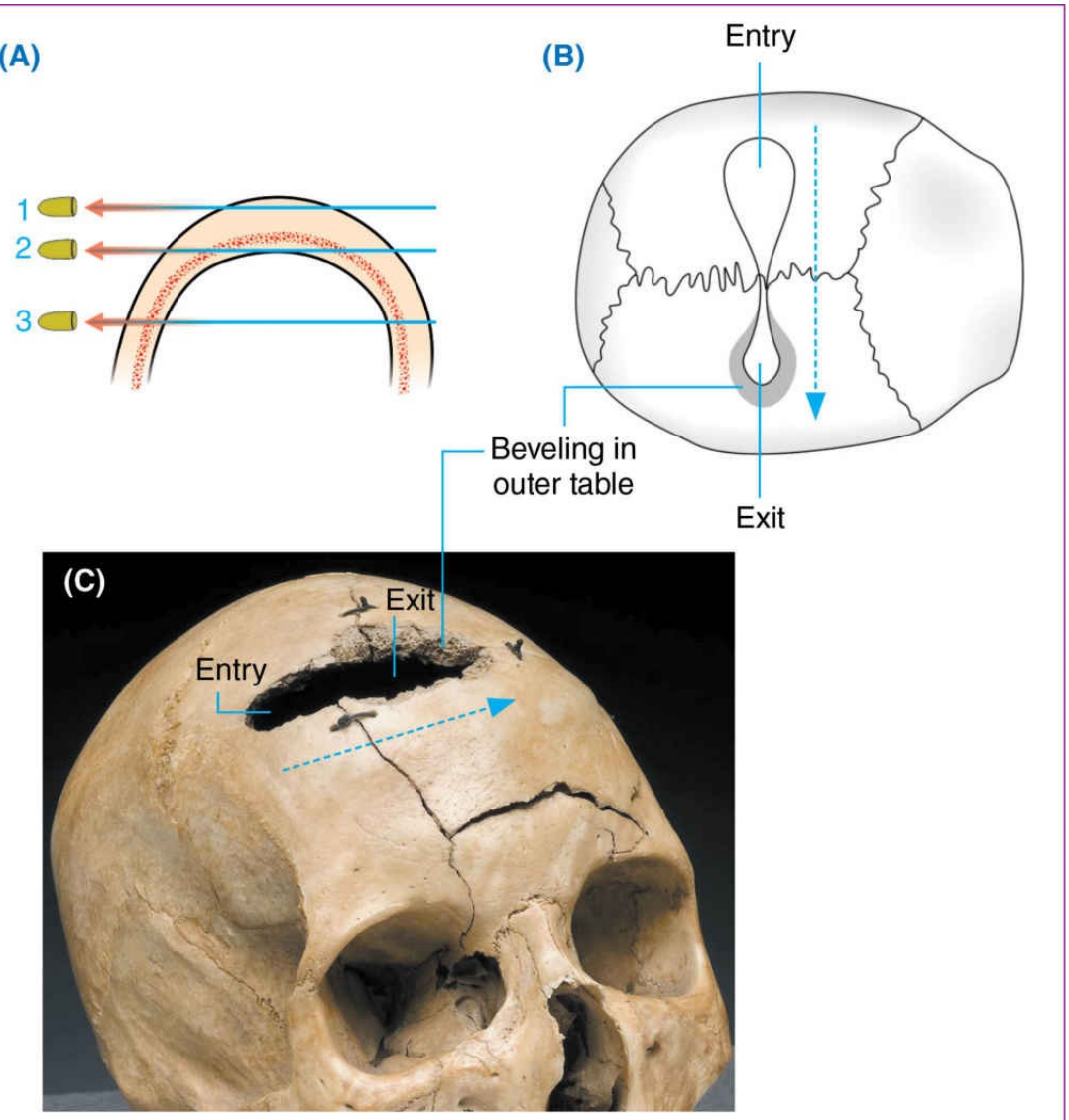


Fig. 17.5: (A) Coronal section of skull depicting hit by a bullet at 3 different levels. Bullet 1 produces gutter fracture of 1st degree [**Fig 17.8**]; bullet 2 produces gutter fracture of 3rd degree [keyhole fracture] and bullet 3 causes perforating fractures **(B and C)** show a typical keyhole fracture. Blue dotted arrow shows the direction of bullet. Pl note that the fracture looks like a keyhole.

(i) Penetrating or perforating fracture

(1) **Cause** – firearms, pointed sharp weapons [axe, dagger, knife, nail]

(2) **Penetrating fracture-**

(i) Characteristics - There is only entry, eg nail driven in the skull.

(ii) Manner - Accidental, suicidal, homicidal or due to supernatural beliefs.

(3) **Perforating fracture** - Characteristics: There is both an entry and exit, eg

firearms.

(4) **Size and shape -**

(i) Almost clean cut

(ii) corresponds with that of the cross section of weapon.

(j) Pond (indented) fracture

A ***pond fracture*** is a circular depressed fracture of the skull.

Salient features:

(1) **Victim** – Occurs only in infants

(2) **Appearance** – Like a simple dent of the skull, much like the indentation of a table-tennis ball produced by finger pressure. Also known as ping-pong fracture.

(3) **Causes -**

(i) Blunt object

(ii) Fall [of an infant from lap of mother]

(iii) Forcible impact against some protruding object

(iv) Obstetric forceps blade [**Fig 17.6**].

(4) **Fracture** – occurs only in the outer table [around the periphery of the dent]. Inner table is not fractured

(5) **Associated injuries** –

(i) Dura mater is not torn

(ii) Usually brain is not damaged.

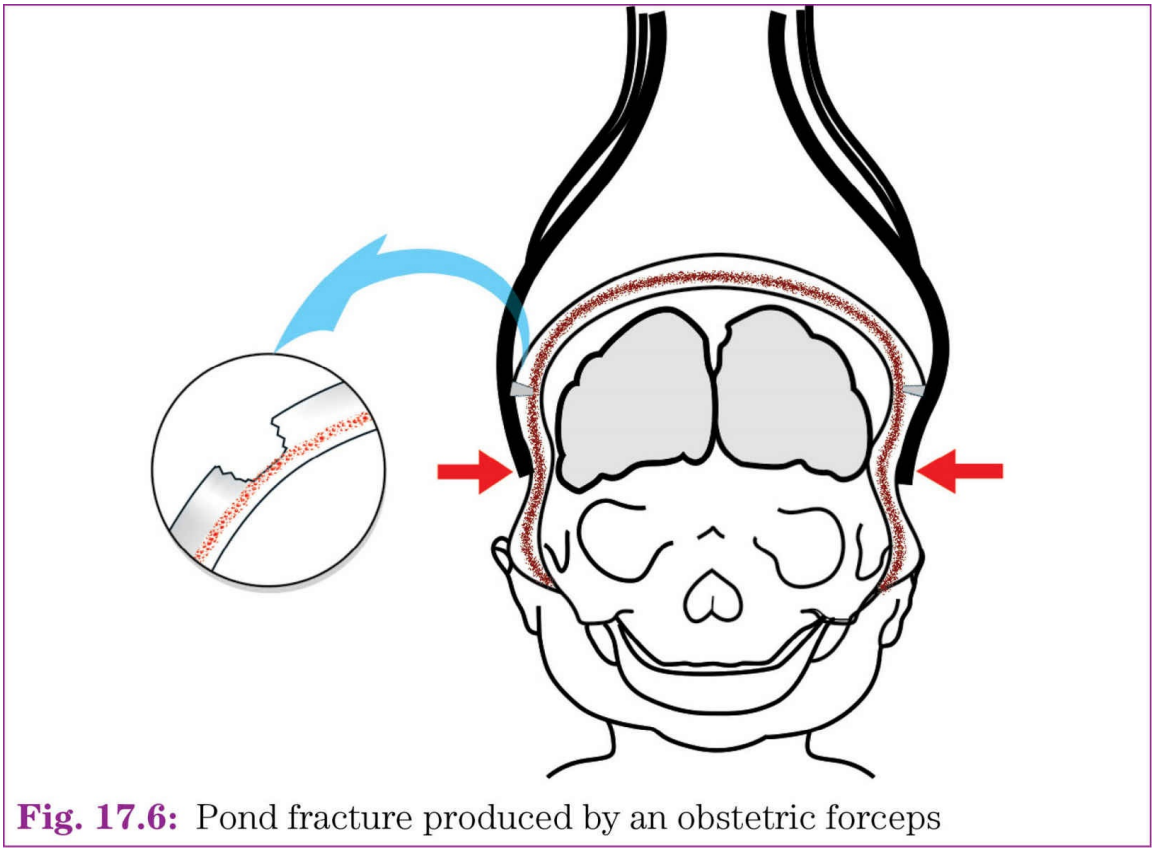


Fig. 17.6: Pond fracture produced by an obstetric forceps

(k) Ring (Foramen) fracture

A ***ring fracture*** (***foramen fracture***) is a circular fracture around the foramen magnum.

Salient features:

(1) Appearance – Fissured fracture running 3-5 cm outside the foramen magnum, all around [**Fig 17.7**]

(2) **Cause:**

(i) fall from height onto feet or buttocks – most common [body weight passing up the vertebral column on to the base of skull]

(ii) heavy weight falls on the vertex.

(3) **Prognosis** - Usually lethal, because of associated brainstem injury.

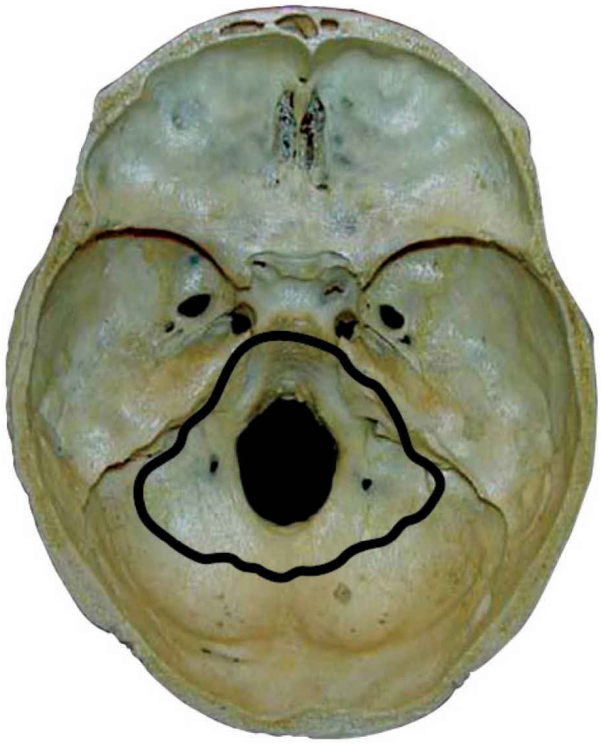


Fig. 17.7: Ring Fracture

(l) Signature fracture

Please see depressed fracture above.

(m) Base of skull fractures

(1) **Causes:**

(i) Application of force to skull specifically to the base (a) directly. **Ex** – ✦ An oblique blow of great force applied to one side of the back of the head"may start a fracture in the underlying posterior fossa"it may cross the middle line to enter the middle fossa of the opposite side"may advance further and may end in the anterior fossa. (b) indirectly through the face [above] or spinal column [below]. When caused by transmitted force (through face or spine), they are usually isolated. **Ex** – ✦ Force of a blow on the mandible [eg an upper cut in boxing]"may be transmitted through the maxilla and its internal angular processes to the base of the skull"may cause an isolated fracture of the cribriform plate of the ethmoid.

(2) Blows on the chin occasionally fracture the mandibular fossa.

(ii) Application of force to skull anywhere – Due to general deformation of skull. **Ex** – ✦ A fall on the back of the head or blow on top of the head usually

produces independent fractures of the orbital roofs, especially in old people.

(iii) Sudden violent - in internal pressure - Produces fractures of the roofs of orbits, especially in suicidal gunshot wounds of the skull. Caused by sudden release of gases in the skull.

(iv) Extension of fracture from vault

(3) **Influencing factors:**

(i) Fracture patterns are strongly influenced by petrous buttresses. Fracture lines which approach these buttresses from middle or posterior cranial fossa are either turned towards its apex or base according to the angle at which they strike them.

(ii) The middle of the body of petrous bone is fractured only when the force is very great.

(4) **Characteristics:**

(i) Most basal fractures tend to meet at and overrun the pituitary fossa.

(ii) Fracture lines usually open into the basal foramina.

(iii) Sphenoid fissure is most commonly involved.

(iv) The foramen magnum is not spared despite its thick margins.

(v) Longitudinally or transversely directed force will generally produce fractures in the corresponding axis.

(5) **Types:**

(i) Incomplete – do not traverse entire base

(ii) Complete or hinge fractures - Traverse entire base creating a “hinge”. Divided in 3 categories [**Fig 17.8**]. (a) Type I -run in the coronal plane, extending from the lateral end of one petrous ridge, through the sella turcica, to the lateral end of the contralateral petrous ridge [**Motorcyclists fracture**; details in **ch 18**]. Most common type of hinge fracture. Due to impacts on the side of the head and, less commonly, to impacts on the tip of the chin [indicated by chin laceration, and less commonly by mandibular fracture] (b) Type II - run from front to the contralateral back, passing through the sella turcica. (c) Type III - run from side to side in the coronal plane but do not pass through the sella turcica.

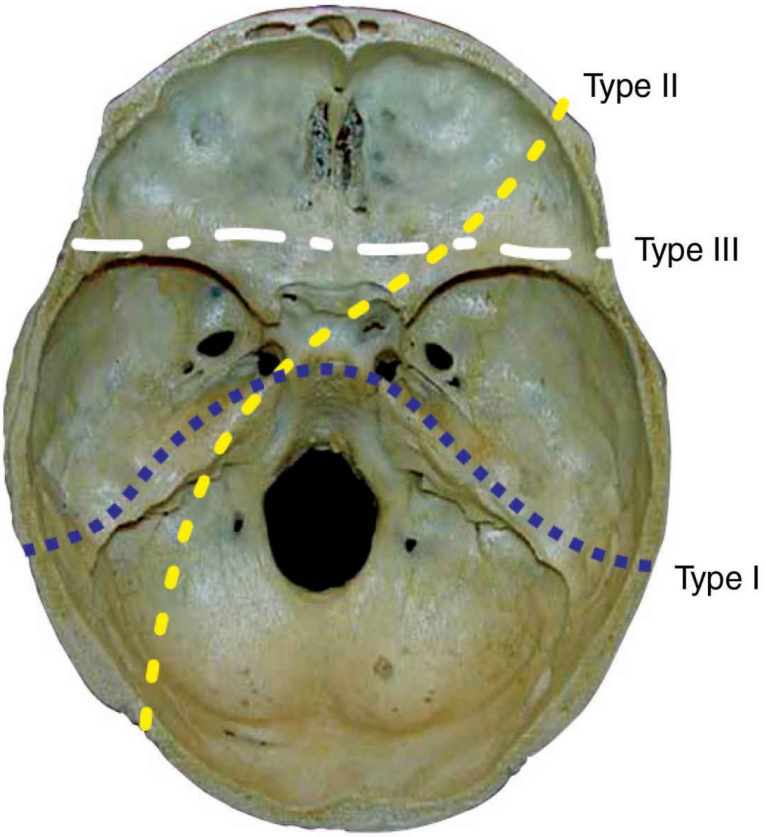


Fig. 17.8: Hinge fractures

iii. According to relationship with impact

(a) Coup fractures

Occur at the site of impact

(b) Contrecoup fractures

(1) **Site** - Occur **opposite** to the site of force.

(2) **Mechanism** - Usually occur when head is not supported. Sudden disturbance of fluid brain content "transmits force received to the opposite side"cranial vault opposite to site of trauma gets fractured.

d. Age of skull fractures

(1) **Healing** - occurs without formation of callus

(2) **Development of external callus** - delayed due to damage to periosteal bv.

(3) **Age:**

(i) 1 wk - Edges of fissured fractures stick together

(ii) 2 wks - (a) edges slightly eroded (b) inner table of skull shows pitting and

deposition of lime salts.

(iii) 3-5 wks - (a) edges become slightly smooth (b) bands of osseous tissue run across the fissure.

(iv) 3 months - Complete healing occurs. If there is much loss of bone, the defect is filled only with fibrous tissue.

e. D/d

Mendosal suture

2. Mandibular fractures

The frequency of fractures of different parts of mandible in .ing order are (1) Condyle [36%] (2) Body [21%] (3) Angle [20%] (4) Parasymphyseal [14%] (5) alveolar [3%] (6) ramus [3%], (7) coronoid [2%] and (8) symphysis [1%].

B. Traumatic Injuries of the Brain

Traumatic brain injury (***TBI***) occurs when an external force traumatically injures the brain.

1. The mechanism of cerebral injury

Brain injury may be caused simply by shaking, without actual blow or fall on the head [ch 27"Shaken baby syndrome].

a. Blunt head trauma

Results in Acceleration/Deceleration injuries.

(1) Linear acceleration in **frontal impact** is **more dangerous** than that in **side impact** (falx absorbs part of the impact)

(2) **Internal structure of the skull:**

(i) determines the most probable location of traumatic injuries of the brain in closed head injury.

(ii) Skull surface of the anterior cranial fossa is quite rough. Furthermore the most anterior frontal vault of the skull is limited in size. As the brain accelerates forward or rotates into the frontal areas of the skull, the infraorbital frontal lobes are impacted by the rough prominences above the orbits"results in contusions or lacerations of bases of frontal lobes"Frontal Lobe Syndromes may occur

(iii) sphenoid ridges of the skull provide a significant structural impediment to the temporal poles"an accordion-like compression of the temporal tips occurs"damage to a number of important structure which temporal lobes

contain [amygdalae, hippocampi, and limbic structures]"disturbances of memory, mood.

b. Penetrating head trauma

(1) **In civilian situations** - Penetrating head trauma by missiles is less frequent than blunt force trauma to the head.

(2) **In military situations** - missile injury to the head is more common than blunt trauma. Missiles are usually bullets, but they also include knives, crossbow bolts, and shrapnel. Even nails, and screwdrivers can become missiles.

2. Contusions of the brain

a. Mechanism of production

Contusions of the brain may be produced in **six** ways (**Fig 17.9**):

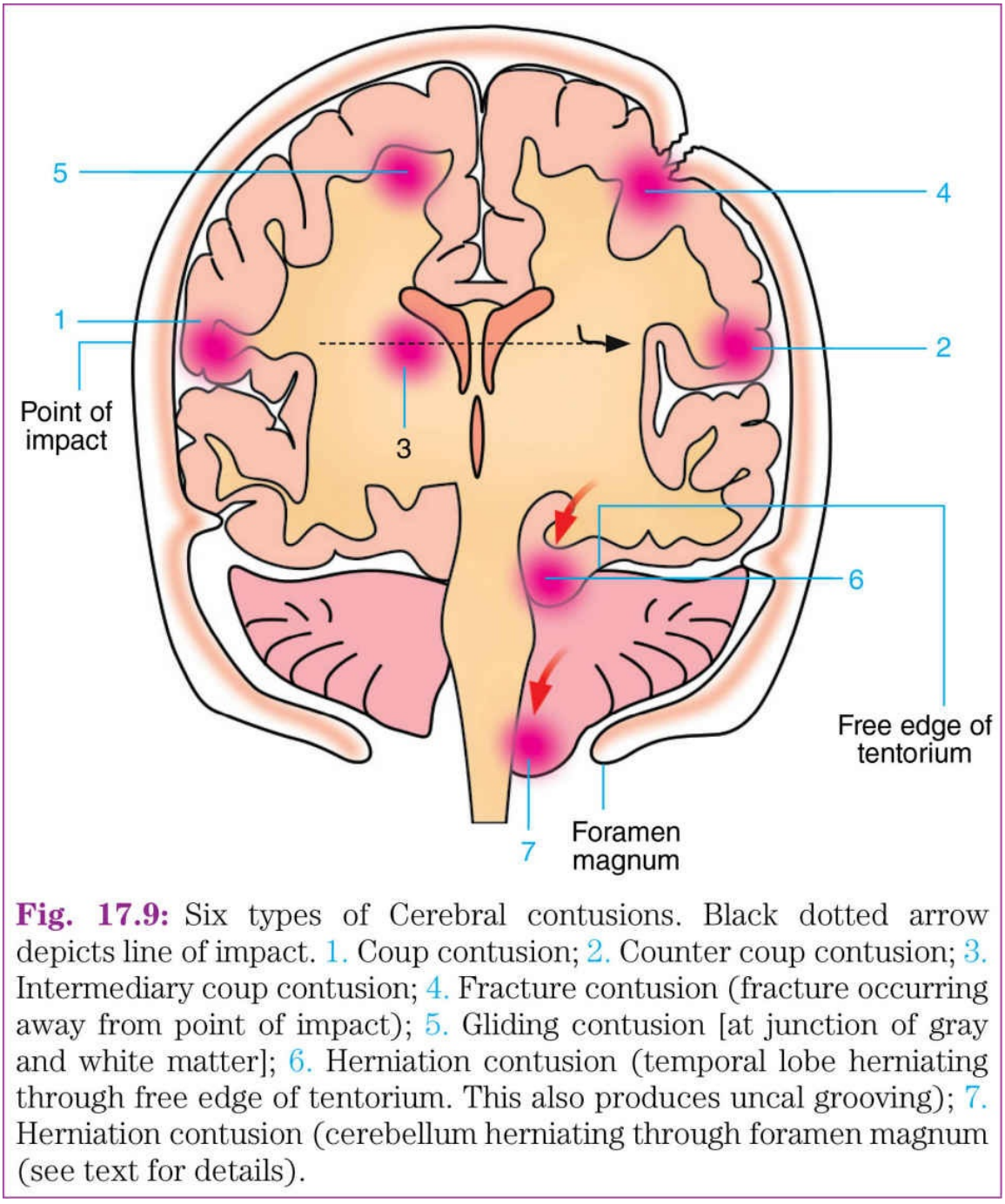


Fig. 17.9: Six types of Cerebral contusions. Black dotted arrow depicts line of impact. 1. Coup contusion; 2. Counter coup contusion; 3. Intermediary coup contusion; 4. Fracture contusion (fracture occurring away from point of impact); 5. Gliding contusion [at junction of gray and white matter]; 6. Herniation contusion (temporal lobe herniating through free edge of tentorium. This also produces uncal grooving); 7. Herniation contusion (cerebellum herniating through foramen magnum (see text for details).

i. Coup contusions

Occur **at the point of impact** due to **in-bending of the bone**, with compression of the brain.

ii. Countercoup contusions

(1) Countercoup [syn, contrecoup] contusions occur in the brain **directly opposite** to the **point of impact**.

(2) Classically associated with **falls**.
(3) Most common in **frontal** and **temporal** lobes. Virtually **never seen in occipital lobes**.
(4) Seen **more frequently** than coup contusions.
(5) Generally **more severe** than coup lesions.
(6) Both coup and countercoup contusions are present over the **crests of gyri**.

iii. Intermediary coup contusions

Occur in the **deep structures** of the brain (white matter, basal ganglia, corpus callosum, brain stem) **between the location of coup and countercoup points**, along the **line of impact**.

iv. Fracture contusions

(1) Associated with **fractures of the skull**.
(2) If fracture occurs at the point of impact, they would be same as coup contusions. They would be called fracture contusions, if **fracture occurs away from point of impact**.

Memory Aid 4: Theories of countercoup injuries

LP done by **R**egistered **N**urse every **H**ou**R**

1. **L**indenberg - **P**ositive pressure
2. **R**ussel - **N**egative pressure
3. **H**olbourn - **R**otational sheer stress theory

v. Gliding contusions

(1) Caused by displacement of the gray matter of the cerebral cortex during **angular acceleration** of the head.
(2) Occur at the **junction** between the **gray matter and the white matter** (cf. coup and countercoup contusions, which occur at gyral crests).
(3) Associated with **diffuse axonal injuries**.

vi. Herniation contusions

(1) Due to **impaction** of medial portion of the **temporal lobes against the edge of the tentorium**, or cerebellar tonsils against the foramen magnum
(2) Independent of site and direction of impact.

Table 1 : Coup and Countercoup Contusions

1. A **blow to the head** produces **coup contusions** (no or minor countercoup contusions) – **head is accelerated**. *Impact occurs first → followed by motion*. In general, **coup lesions** predominate if head is **accelerated** after impact.
2. A **fall on the head** produces **countercoup contusions** (no or minor coup contusions) - **head is decelerated**. *Motion occurs first → followed by impact*. In general, **contrecoup lesions** predominate if head is **decelerated** after impact.

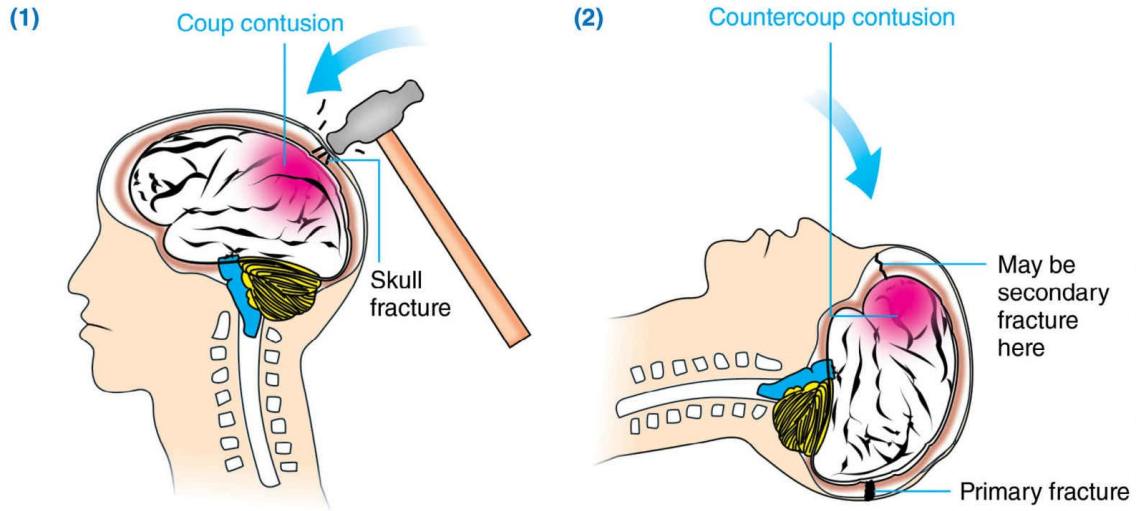


Fig. 17.10: **(1)** Coup contusions are caused when force strikes a fixed head **(2)** Countercoup contusions are caused when a moving head strikes a hard surface (see text for details)

Table 2: Theories of Countercoup Injuries

Following **theories** have been advocated to explain **countercoup injuries**.

1. **Positive pressure theory** - Advocated by **Lindenberg**. Its salient features are as follows (**Fig 17.11**).
   (i) At the beginning of fall, skull is accelerated. **Brain lags behind due to inertia** (stage 1 in **Fig 17.11**).
   (ii) As victim continues to fall, brain lags far and far behind. This creates a **negative pressure** at the ***leading skull surface*** (the surface that is going to hit the surface), and a **positive pressure** at the ***trailing skull surface*** (stage 2 in figure).
   (iii) As CSF continues to move towards the leading skull surface, very little CSF remains at the trailing skull surface, by the time impact occurs. Thus there is **no cushioning effect** of CSF there. Furthermore a **very great +ve pressure** has been built up at the trailing skin surface (stage 3 in **Fig 17.11**). Both factors combine to produce **countercoup lesions**.

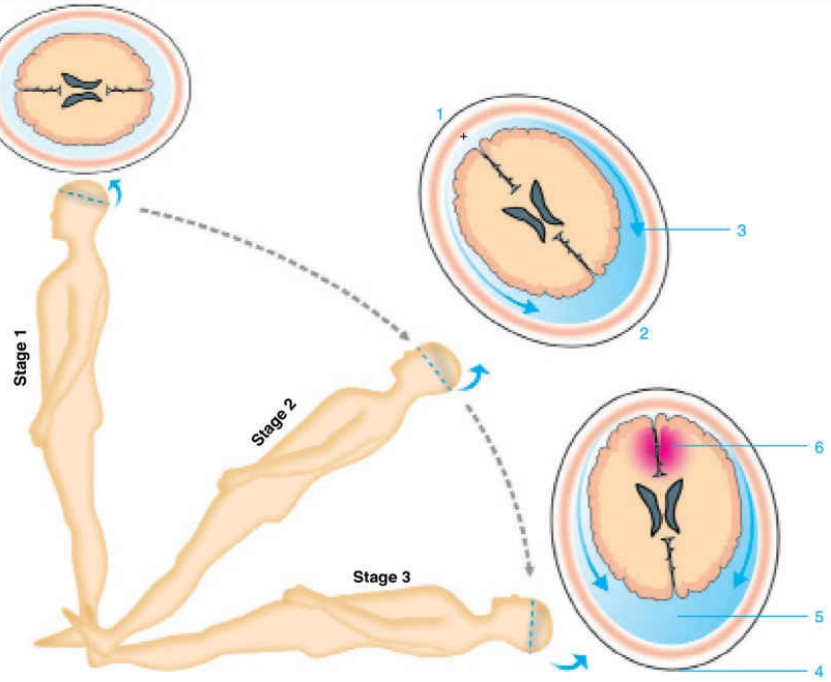


Fig. 17.11: Positive pressure theory of **Lindenberg**. (1) Trailing skull surface; (2) Leading skull surface; (3) CSF shifting towards leading skull surface; (4) Point of impact; (5) More CSF collecting here protects brain; (6) Counter coup contusion. See text for details

2. **Negative pressure theory** - Advocated by **Russel**. Also known as the **cavitation theory**. Russel agrees with the first three stages described by Lindenberg, but asserts that countercoup lesions do not occur at the time of impact, but a fraction of a second after. His salient features are:

(i) Russel goes on to describe stage 4 of the fall (Fig 17.12). The skull has been suddenly brought to rest. But the **brain continues to move**.

(ii) Because of continued movement of brain, **negative pressure** now develops at the trailing skull surface.

(iii) This produces a **tensile stress** on the brain. **Cavitation** occurs within the brain. This causes countercoup lesions.

3. **Rotational shear stress theory** - Advocated by **Holbourn**. Holbourn, a physicist (not a medical doctor) believed that in falls, there is always a **rotational element** involved (Fig 17.13 a). As the skull comes to a stop, the brain **continues to rotate**, and strikes against **fixed bony projections** within the skull (crista galli, sphenoidal wings). This causes countercoup injuries (Fig 17.13 b). Although this theory accounts for injuries frequently observed in inferior portions of temporal and frontal lobes, *it does not adequately account for the contrecoup injury, directly opposite the site of skull impact.*

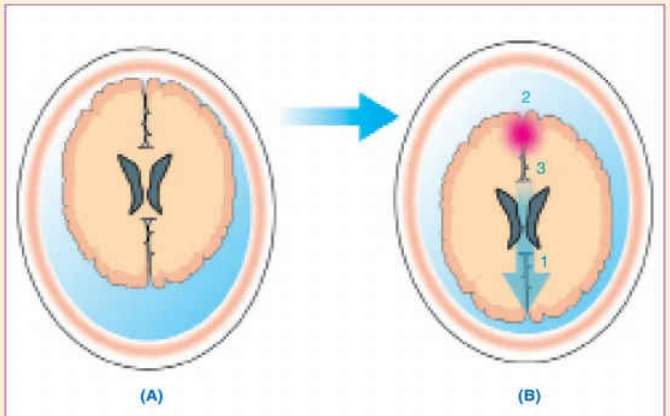


Fig. 17.12: Negative pressure theory of **Russel**. **(A)** Depicts stage 3 of fall as described by Lindenberg. According to Lindenberg, countercoup injury to brain occurs at this stage itself due to +ve pressure [Fig 17.22], but according to Russel, it occurs a fraction of a second later **(B)**. (1) Brain continues to move, while skull stops suddenly, producing -ve pressure at trailing skull surface; (2) Tensile stress produced in brain by -ve pressure; (3) Counter coup contusion produced by this tensile stress. Pl see text for details.

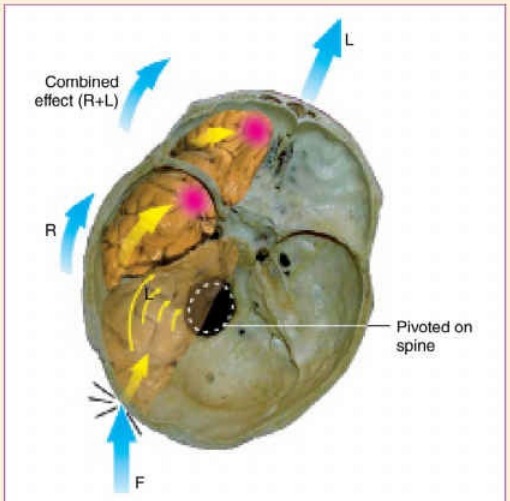


Fig. 17.13: Rotational shear stress theory of **Holbourn**. When a force **'F'** strikes the skull it can be broken in two components–a linear component **'L'** and a rotational component **'R'**. Counter coup contusions are produced by **'R'**. See text for details

b. Morphological appearances

Morphologically, cerebral contusions are of **three** types:

i. Contusion hemorrhages

Contusion hemorrhages are located at the crest of a gyrus. They are multiple in number, streak like and densely arranged (**Fig 17.14**). They point radially towards the white matter.

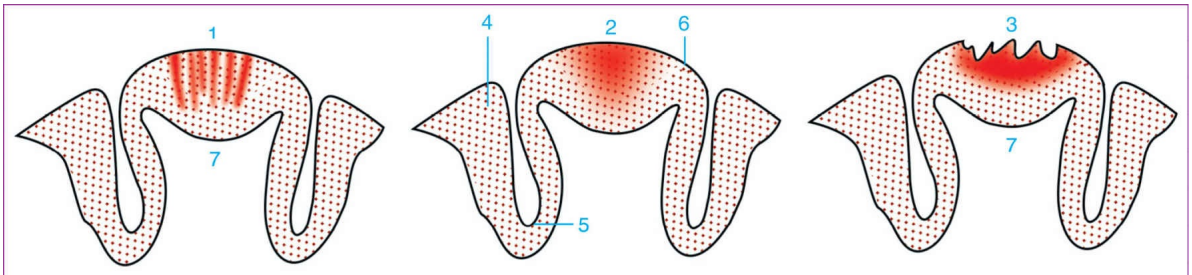


Fig. 17.14: Three main contusion types in the brain. (1) Contusion hemorrhages; (2) Contusion necrosis; (3) Contusion tear; (4) Gray matter; (5) Sulcus; (6) Crest of gyrus; (7) White matter

ii. Contusion necrosis

Contusion necrosis, just as contusion hemorrhage tends to be in the cortex, at the crest of a gyrus, **sparing the sulci**. It is wedge shaped, with the base of the wedge at the cortical crest. The lesion becomes visible **10-12 hours after injury**.

iii. Contusion tears

Contusion tears are caused by stretching and shearing forces within the tissues produced by blunt force. They are the dominant types of contusions in **infants up to the age of five months**. At this age, skull is easily deformed and the brain is quite soft. The tears usually involve the white matter of the **upper frontal convolutions**, **orbital lobes** and **temporal lobes**.

3. Diffuse axonal injury (DAI)

Diffuse axonal injury (DAI) refers to extensive lesions in white matter tracts spread over wide diffuse areas. DAI is generally produced by ***rotational motions*** of the head. Because of this, it is also known as **shearing brain injury** or **axonal shear injury**.

a. Mechanism of DAI

(1) **Earlier mechanical theory** - The hallmark of DAI is **axonal damage**. Earlier it was thought that the main cause of axonal damage was tearing due to **mechanical forces** during the trauma. Because of **different tissue consistencies** within the brain layers and **relative fixation of certain parts of the brain** to the rigid skull, the deep and superficial portions **may not move at the same rate**. This results in **shear strain** that manifests as axonal injury and rupture. Since different tissue consistencies and different degrees of fixation of brain are responsible for DAI, the lesions are predominantly found in some special locations such as the **white-gray matter junction** and **corpus**

callosum (because it is rigidly attached to the falx) [**Fig. 17.15**].

(2) **Modern biochemical theory:** (i) Stretching of axons during injury causes physical disruption of **axolemma** [membrane covering the axon]"(ii) This **opens up Na^+ channels in the axolemma**"(iii) Voltage changes"(iv) **voltage-gated Ca^{2+} channels open**"(v) Ca^{2+} flows into the axon"(vi) intracellular presence of Ca^{2+} **activates phospholipases** and **proteolytic enzymes**"(vii) damages mitochondria and cytoskeleton; separation of axon"(viii) **Axons swell up**; there is impaired transport and **accumulation of axonal transport proteins** [fast transport **β-amyloid precursor protein (β APP)** and the slow transport **neurofilament (NF) protein**] within axonal swellings"(ix) neuronal death.

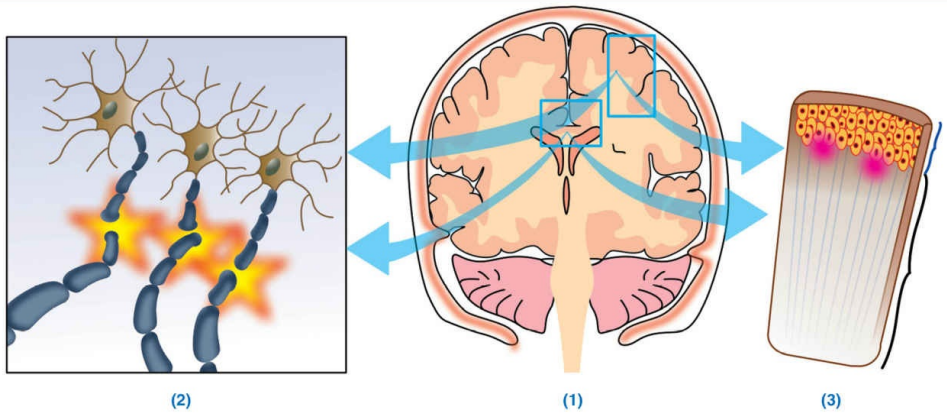


Fig. 17.15: (1) In DAI, the injury is greatest where tissue consistencies differ, eg **white-gray matter junction** or where brain is rigidly attached, eg **corpus callosum (2)** According to the **earlier mechanical theory**, axons are severed. They retract and swollen retractions balls are formed at the severed ends. There is also a "**beads on the string**" appearance along axonal lengths. **(3)** According to the **modern biochemical theory**, only axolemma is disrupted setting in train, a biochemical series of actions, which ultimately results in axonal damage. Pl see text for details.

b. Clinical features

The clinical presentation of DAI depends on the severity of injury. Both severe [Glasgow coma scale (GCS) 3 to 8] and moderate [GCS 9 to 12] head injuries have been associated with DAI.

(1) **Mild DAI - Coma lasts 6-24 hours** [coma lasting <6 hours is termed as severe concussion – please see below]. Patients with mild DAI usually present with a history of **brief loss of consciousness** and **good neurologic status** without subsequent deterioration. One-third to one-half of mild head injury patients develop a **postconcussion syndrome**. They usually have subjective complaints including **difficulty concentrating**, **memory problems**, **headache**, or **disequilibrium**; and **neuropsychologic testing** reveals **deficits**

in information processing.

(2) **Moderate DAI -Coma lasts > 24 h *without*** prominent clinical signs of **brain stem dysfunction**.

(3) **Severe DAI - Coma lasts > 24 h with brain stem dysfunction signs**.

c. PM/Histological appearances

(1) Grossly there are **no pm findings**. In some cases there may be petechial hemorrhages through white matter.

(2) Breakage of axons at the time of brain trauma (**primary axotomy**) rarely occurs. More commonly breakage occurs in the course of hours to days after injury, even several months later (**secondary axotomy**) due to biochemical mechanisms described above.

(3) Most significant finding is bulb formation at the terminal end of the severed axon ("**terminal clubbing**" or "**retraction balls**") [**Fig. 17.15**].

(4) In **severe DAI** axonal pathology is accompanied by **tears in the white matter** and **intraparenchymal hemorrhage**.

4. Cerebral concussion

Concussion (Latin concutere, "to shake violently") is a clinical syndrome characterized by the immediate and transient post-traumatic impairment of neural function such as alteration of consciousness, disturbance of vision or equilibrium due to mechanical forces.

Salient features:

(1) **Common post concussion Signs and Symptoms** [**PCSS**]- anxiety, bell rung [excessive noise sensitivity], confusion, depression, dizziness numbness/tingling, drowsiness, easy distraction, excess sleep, fatigue, feel "in a fog", feel "slowed down", headache, inappropriate emotions, irritability, light intolerance, loss of consciousness, loss of orientation, memory deficits, nausea, nervousness, noise intolerance, personality change, poor balance, poor concentration, poor coordination, ringing in the ear(s) , sadness and stupor. These are used not only for the initial evaluation but for each subsequent follow-up assessment, which is periodically repeated until all PCSS have cleared at rest and exertion.

(2) A second head injury during the recovery phase after a concussion can lead to death or permanent major disability [**second impact syndrome**].

MLI

(1) **Concealed trauma** – causes death without any gross signs [**ch 8**].

(2) **Drunkenness** - Head injury and concussion can often mimic alcoholic intoxication. **Table 3** gives salient differences between drunkenness and concussion.

Table 3: Differences between Drunkenness and concussion

S.No.	Trait	Drunk	Concussion
1.	Face	Warm, flushed, suffused	Pale, clammy
2.	Smell of alcohol	Present	No smell of alcohol
3.	Pulse	Fast, bounding	Slow, feeble
4.	Pupils	McEwen's sign may be elicited. Contracted pupil, dilates on external stimuli and contracts again	Contracted, unequal [anisocoria]
5.	Breathing	Eructations, sighing and puffing	Slow, Shallow, irregular
6.	Memory	No amnesia. Confused	Retrograde amnesia
7.	Behavior	Uncooperative, talkative, abusive	Quiet, co-operative
8.	Blood and urine for alcohol	Positive	Negative

5. Cerebral edema

Cerebral edema is an excess accumulation of water in the intracellular or extracellular spaces of the brain.

a. Classification of Cerebral edema

i. Vasogenic

(1) **Causes** –

(i) cerebral ischemia [late stages]

(ii) focal inflammation

(iii) hypertensive encephalopathy

(iv) trauma [Massive cerebral swelling can occur within 20 min following head trauma]

(v) tumors

(2) **Mechanism** - Due to a breakdown of tight endothelial junctions which make up the BBB.

(i) physical disruption by arterial hypertension or trauma

(ii) tumor-facilitated release of vasoactive and endothelial destructive compounds [arachidonic acid, bradykinin, eicosanoids, excitatory neurotransmitters, free radicals, histamine]

(3) **Subtypes:**

(i) Hydrostatic cerebral edema - seen in acute, malignant hypertension. Results from direct transmission of pressure to cerebral capillary with transudation of fluid into the ECF from the capillaries.

(ii) Cerebral edema from brain cancer - Cancerous glial cells (glioma) of the brain - secretion of vascular endothelial growth factor (VEGF) " weakens junctions of BBB

(iii) High Altitude Cerebral Edema [HACE] - due to the effects of hypoxia on the mitochondria-rich endothelial cells of BBB.

ii. Cytotoxic

(1) **Causes** - (i) cardiac arrest (ii) Encephalopathy (iii) Hypothermia [severe] (iv) intoxications [dinitrophenol, hexachlorophene, isoniazid, triethyltin] (v) Ischemia [early] (vi) pseudotumor cerebri (vii) Reye's syndrome (viii) stroke or hypoxia [early]

(2) **Mechanism** - BBB remains intact. Edema is due to the derangement in cellular metabolism"inadequate functioning of the Na^+ and K^+ pump in the glial cell membrane"cellular retention of Na^+ and H_2O"swollen astrocytes in

gray and white matter.

(3) **PM appearances** – in cytotoxic cerebral edema there is - in intracellular fluid in contrast to vasogenic cerebral edema where there is - in intravascular cerebral blood volume and - in interstitial fluid. In the former [cytotoxic], brain is enlarged, firm and has a relatively dry cut surface; in the latter [vasogenic] the brain is similarly enlarged but is softer and has a very watery cut surface.

iii. Osmotic

(1) **Causes** - (i) excessive water intake (ii) hemodialysis (iii) hyponatremia (iv) rapid reduction of blood glucose in hyperosmolar hyperglycemic state (HHS) [formerly hyperosmolar non-ketotic acidosis (HONK)] (v) Syndrome of inappropriate antidiuretic hormone secretion (SIADH)

(2) **Mechanism** - Normally brain osmolality is < than that of serum osmolality brain osmolality becomes > than that of serum osmolality in above causes"creates a reverse pressure gradient"edema.

iv. Interstitial

(1) **Causes** - obstructive hydrocephalus.

(2) **Mechanism** - rupture of the CSF-brain barrier"trans-ependymal flow of CSF"CSF penetrates the brain and spread to the extracellular spaces and the white matter. Differentiated from vasogenic edema in that interstitial cerebral edema CSF contains almost no protein.

b. Extent

Cerebral edema may be

(1) **Focal** – adjacent to the area of injury

(2) **Diffuse** -

(i) Unilateral [involves one hemisphere only]. Occurs mostly in relation to ipsilateral acute SDH

(ii) Bilateral [involves both hemispheres].

c. PM Appearances

(1) **Dura** - stretched and tense

(2) **Brain** - bulging

(3) Wt -

(4) **Cerebral surface** – smooth (i) Gyri – pale, flattened with thinning of gray matter (ii) Sulci – obliterated

(5) **Cut surface** – pale
(6) Signs of herniations.

6. Cerebral compression

Cerebral compression results from an - in the size of brain.

Salient features:

(1) - **in size of brain** - may occur due to edema [please see above], space occupying lesions or trauma.
(2) **The magnitude of brain swelling** - does not necessarily correspond to the severity of the injury.
(3) Effects of cerebral compression [please see pathological effects of EDH below].

C. Intracranial Hemorrhages

Intracranial hemorrhage [ICH] is any bleeding inside the skull.

Salient features:

(1) **Hemorrhage** – small and thin layered bleeding
(2) **Hematoma** – Large and space occupying bleeding [for definitions of **petechial hgs** and **ecchymosis**, please see ch 19].
(3) **Types** -
(i) Primary – occurs immediately after trauma
(ii) Secondary [syn, Delayed traumatic intracranial hemorrhage (DTICH), Reactionary hemorrhage]– First described in **1891** by **Bollinger** who called it "traumatische Spät-Apoplexie". (a) occurs within first 10 days of injury, particularly in the first 3. (b) Incidence after severe head trauma - 2-10%. (c) Mechanism. (II) Due to weakening of capillary walls and subsequent rupture.

1. Extradural hemorrhage

Extradural hemorrhage [syn ***epidural hemorrhage***, ***EDH***] is bleeding into the extradural space. As the bleeding clots and forms a large firm mass it is often referred to as ***hematoma***.

a. Causes

(1) Traumatic [main]
(2) Spontaneous. Very rare. Seen in (i) coagulation disorders, (ii) dural vascular malformations (iii) frontal sinusitis and (iv) infections.

b. Epidemiology

(1) Incidence – least common type of meningeal bleeding
(i) 1-2% of all treated head injuries
(ii) 5-10% of autopsies in fatal head injuries
(2) Age -
(i) Most common – 10-30 y
(ii) rarest - <2 y and >60 y. At these ages dura is more firmly attached to the inner table.

c. Mechanism

(1) **Fracture of skull** – In 90% cases, the fractures is fissured type. In rest it is depressed fracture"Blood vessel [bv] ruptures"blood starts collecting in space"As more and more blood collects, dura gets stripped further and further"Stripping of dura causes further damage to communicating v between skull and dura"More bleeding"Because stripping of dura is a slow process, typically EDH develops over several hours. Bleeding may continue for several hours or even a day after injury.

(2) **Impact to skull without fracture** - Skull and dura separate momentarily"An empty extradural space is created at the site of trauma"If bv does not rupture, nothing further happens and immediately dura and skull come to adhere once again; if bv ruptures, mechanism is same as above. EDH without fracture of skull is rare.

(3) **EDH in neonates** – is extremely rare because of firm adherence of dura to the skull. If occurs, limited to single bones because of adherence of the dura to the skull along the suture lines.

d. Vessels involved

Depend on site of trauma. EDH is mainly produced from inj to **arteries**. Veins are generally unable to produce EDH, because their bleeding cannot generate sufficient pressure to strip dura from bone. However EDH can be of venous origin and also of mixed origin.

Blow over lateral convexity of head. Can cause 4 **types** of injuries (i) fracture of pterion [weakest part of the skull; overlies anterior branch of middle meningeal artery, **Fig 17.16**]"injury to anterior branch – EDH tends to run into the middle cranial fossa (ii) fracture of squamous temporal bone.

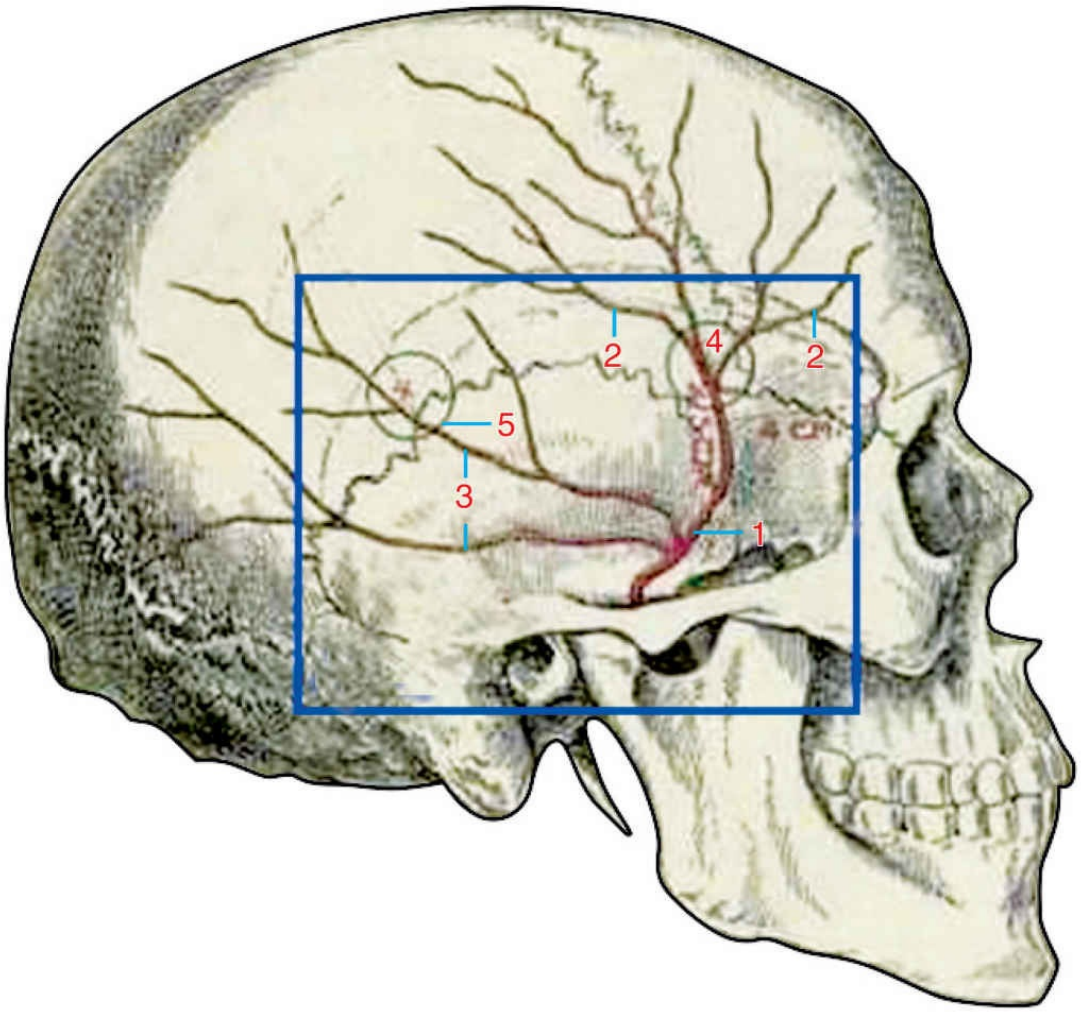


Fig. 17.16: **(1)** Right middle meningeal artery showing areas (**4** and **5**) where it ruptures most commonly; **(2)** Anterior branches; **(3)** Posterior branches; **(4)** Pterion; **(5)** Asterion.

e. Characteristics

(1) **Site:**

(i) Directly underneath the site of impact [in almost all cases]

(ii) Countercoup EDH – very rare. Seen only if skull is grossly deformed

(iii) At base of skull – rare

(iv) Usually unilateral. The sites are (a) Temporoparietal area [most common] (b) Frontotemporal (c) Parieto-occipital (d) Occasionally – frontal region, anterior or posterior fossa

(v) Rarely bilateral – occurs in bilateral trauma or when a median structure eg

sagittal sinus has been injured.

(2) **Size and shape:**

(i) Clot is oval or circular and sharply defined

(ii) Dimensions - (a) Diameter 10-20 cm (b) Thickness 2-6 cm (c) Weight 30-300 g

(iii) Adherent to dura mater

(iv) Presses dura inward

(v) Causes a localized concavity of external surface of brain

(3) **Associated hemorrhages** – about 50% have a concomitant hemorrhage [SDH, SAH or intracerebral]

f. Pathological effects

These pathological effects are not unique to EDH; They would be seen in any space occupying lesion [SOL], or generalized cerebral swelling causing cerebral compression

(1) **Herniations**.

(2) **Classification:**

(i) Supratentorial herniation - (a) subfalcial [Cingulate] (b) Uncal [transtentorial]. (c) downward [central, transtentorial] (d) External [transcalvarial]

(ii) *Infratentorial herniation* - (e) Tonsillar [downward cerebellar] – also known as **coning** [because tonsils herniate like a cone]. (f) Upward [upward cerebellar or upward transtentorial].

(3) **Kernohan's notch** – It is a unilateral grooving of a cerebral peduncle Caused when the midbrain is pushed against the contralateral tentorial edge"compresses the cerebral peduncle. During life, it results in a hemiparesis that is ipsilateral to EDH. While ***Kernohan's notch*** is on cerebral peduncles, ***uncal grooving*** is on uncus.

(4) Cerebral edema

(5) **Duret's hemorrhages** - These are secondary teardrop shape hemorrhages in the brainstem. Range from small streaks to massive confluent hemorrhages.

g. Symptoms and signs

(1) There may be initial unconsciousness due to concussion. In about 25% of cases, initial unconsciousness does not occur

(2) -ing weakness - on the side opposite to that of EDH. Occurs in face and arms first, then spreads to legs. Symptoms may worsen due to cerebral edema or

diffuse axonal injury [DAI]

(3) **Pupils** –

(i) dilated, unresponsive to light, usually on the side of hemorrhage [anisocoria - unequal pupils].

(ii) Later there is bilateral dilatation and fixation of pupils

(4) **Decerebrate rigidity and death** - in about 20-50% cases. Cause of death is respiratory failure due to compression of brain stem.

h. Sequelae

(1) **Small hematomas** – may undergo resorption by phagocytes derived from perivascular dural cells

(2) **Large hematomas occasionally may become smaller** - due to escape of blood through a fracture into s/c tissues. If escapes in scalp"may form a hematoma underneath the scalp.

i. Chronic EDH

EDH is mostly of acute nature; however rarely a patient may report several days after the injury. The term ***chronic EDH*** is typically used, when a patient reports 13 days after injury, because microscopic ossification of an EDH occurs at this period. Ch EDH has however been reported up to 6 y after injury.

(1) **Cause** - Generally due to venous bleeding which is much slower. May originate from sinuses or from the diploic veins.

(2) They represent 7-14% of EDH. In 25% cases the cause is not determined.

(3) Infection of the ch EDH occurs but is uncommon.

j. D/d

Dural metastases – secondary to neoplasia eg bronchogenic carcinoma. Patient may present with hemiparesis and plain CT scan findings highly suggestive of EDH. Such metastasis may also present as ch SDH.

k. Cause of death

Secondary pressure on brain stem"stoppage of respiration.

l. PM examination

Gentle removal of hematoma - may show

(1) a rupture in the vessels [may be visible during operation also] and

(2) a fissured fracture of the skull nearby. It is often confined to the inner table.

m. Dating of EDH

(1) **Recent effusion** – bright red

(2) **4th day** - Bluish-black to brown

(3) **12-25 days** - Pale brownish yellow

(4) **Few months** – coagulum becomes firmer and laminated.

n. MLI

(1) **Minimum amount associated with fatality** – 100 mL.

(2) **Lucid interval** [syn, latent interval]:

(i) Mechanism - Initial trauma [**Fig 17.17**, point A] causes unconsciousness. An artery in extradural space may rupture and bleeding may begin slowly. Initial unconsciousness resolves within a few minutes [B]. The person may remain active for a period varying from few hours to a week [B-C]. This is the lucid interval.

(ii) Incidence – 30-40% of head injuries only

(iii) Not seen in massive injuries to brain – because recovery from initial consciousness is late and EDH develops much before that time; thus the unconsciousness due to two conditions overlaps.

(iv) Also seen in (a) Subdural hematoma [please see below] (b) Fat Embolism Syndrome [FES] (c) mental illness [**ch 28**].

(v) MLI - (a) During this period the person is responsible for all actions. (b) Doctor may be charged with negligence if he discharges the patient prematurely at point A.

(3) **EDH**, **SDH** etc can be preserved for chemical analysis in late deaths, when drug has disappeared from peripheral blood [ch 40"MLI of ethyl alcohol].

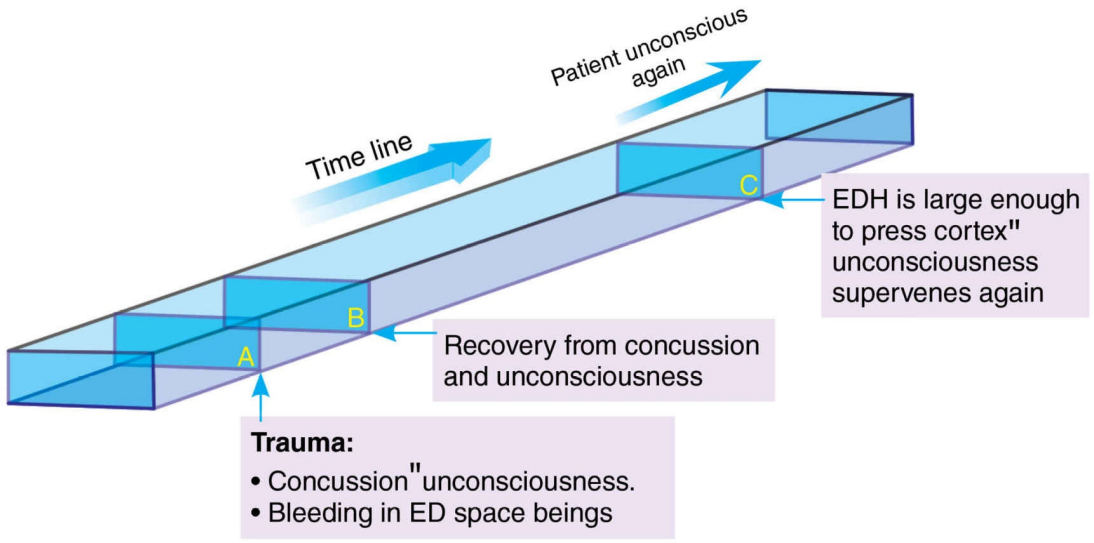


Fig. 17.17: Lucid interval due to EDH. Please see text for details.

2. Subdural hemorrhage

Subdural hemorrhage is bleeding into the subdural space. As the bleeding clots and forms a large firm mass it is often referred to as ***hematoma***.

a. Anatomy

(1) Subdural space is very narrow and contains a small amount of fluid permitting the thin and tough arachnoid to move relative to dura

(2) The cerebral veins [bridging veins] cross this space to reach sinuses

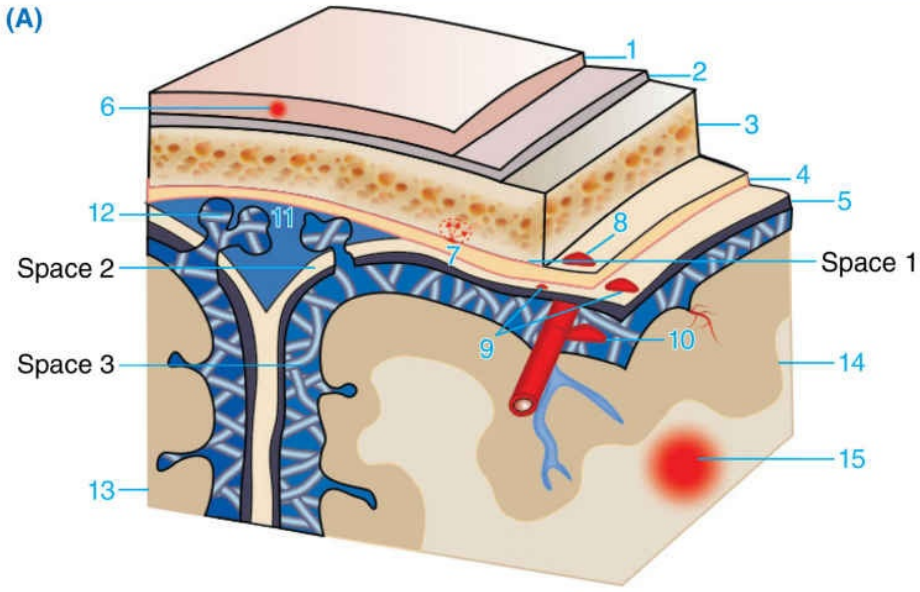
(3) Arachnoid is attached to the dura by arachnoid granulations and venous sinuses

(4) In **Fig 17.18**, **space 1** is the extradural space [as can be seen, there is no actual potential space here]; **space 2** is subdural space, and **space 3** is subarachnoid space. Normally rupture of middle meningeal artery will cause bleeding in extradural space, but if associated dura is torn, bleeding can occur in subdural space. Similarly arteries of the Circle of Willis (where aneurysms are most commonly found) normally cause bleeding in subarachnoid space, but if associated arachnoid is torn, bleeding can occur in subdural space. Since subdural space is the “middle space”, bleedings from adjacent spaces can come in this space through a tear in the corresponding membrane.

(5) **Hemorrhage:**

(i) Origin - mostly venous or capillary. Rarely arterial [please see below].

(ii) Volume – ranges from a few drops to 150 mL [sufficient to cause death], or sometimes even more.



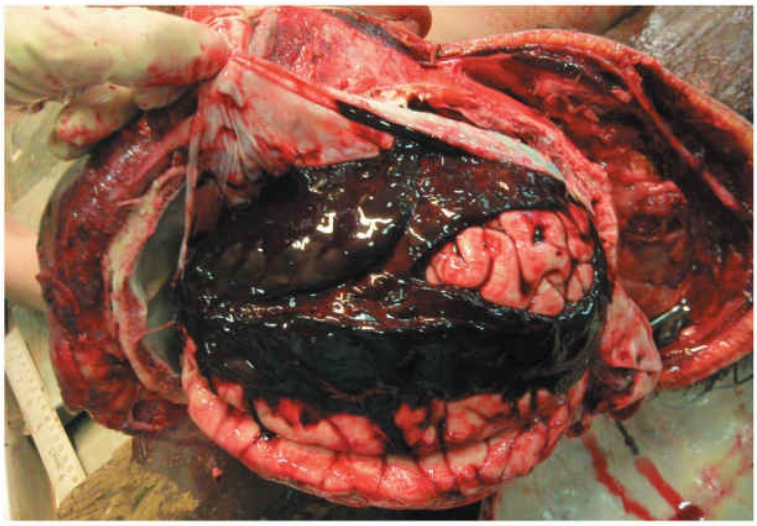


Fig. 17.18: (A) A schematic diagram to understand the location and causation of various cranial and intracranial hemorrhages. 1. Scalp; 2. Periosteum; 3. Skull; 4. Dura; 5. Arachnoid; 6. Scalp hematoma; 7. Middle meningeal artery at the time of rupture. Since there is no potential space here it takes sometime for EDH to develop; 8. EDH; 9. SDH; 10. SAH; 11. Superior sagittal sinus; 12. Arachnoid granulations; 13. Falx; 14. Boundary between gray and white matter; 15. Cerebral hemorrahage. **(B)** Subdural hemorrhage

b. Causes

(1) **Traumatic ruptures** – are the main cause. Rupture of

(i) parasagittal bridging or communicating veins near sagittal sinus [most common]. These veins rupture when brain moves relative to dura. Seen in shaken baby syndrome [**ch 27**]

(ii) aneurysms or a malformed superficial bv through the arachnoid into subdural space [**Fig 17.18**]

(iii) cortical vessels

(iv) dural venous sinuses

(v) inferior cerebral veins entering sinuses at the base of skull

(vi) middle meningeal artery [with associated laceration of dura] so that it bleeds directly into the subdural space, instead of epidural space [**Fig 17.18**].

(2) **Anticoagulants** -

(i) Dicoumarol

(ii) heparin

(iii) Warfarin. SDH may occur even in the absence of history of trauma.

(3) **Contusions and lacerations** - of brain and dura

(4) **Metabolic disorders** –

(i) Galactosemia

(ii) Glutaric aciduria type I.

(iii) Menkes disease. These diseases may indeed be mistaken for shaken baby syndrome [**ch 27**].

(5) **Old adhesions** - between brain and dura breaking up

(6) **Perinatal** -

(i) Following labor – due to head moulding in newborns**.** Usually in the posterior fossa and massive.

(ii) Precipitate labor – [**ch 27**]

(7) **An already formed *SAH*** - may bleed into subdural space, if dura tears due to blunt force

(8) **Secondary to disease** -

(i) Bleeding diathesis

(ii) Cerebral aneurysm

(iii) Cerebral tumor

(9) **Miscellaneous** -

(i) Arachnoid cyst rupture

(ii) -CVP
(iii) Hypernatremia.

c. Epidemiology

(1) **Incidence** – Commoner than EDH
(2) **Age** -
(i) Common in childhood and old age
(3) **Degree and type of trauma** –
(i) May occur from relatively insignificant trauma insufficient to produce any other visible mark. Thus it may be seen in the absence of fractures of skull, cerebral injuries [eg contusions or lacerations] or unconsciousness
(ii) About 70% due to falls; 25% due to vehicular accidents; 5% due to other reasons
(iii) Fatal SDH – usually associated with contusions and lacerations of brain and fractures of skull.
(4) **Site** - Supratentorial – upper lateral surface of cerebral hemispheres [most common]. Gradually the blood drains down under gravity to cover the entire cerebral hemispheres. Large accumulations occur in cranial fossae also [all 3, but most commonly in ACF and MCF]
(5) **Accumulation** – is quicker than EDH, but occasionally may be as slow as EDH. With slower accumulation, considerably larger sizes of SDH may be tolerated.
(6) **May be associated with**
(i) alcoholism [Reasons (a) liver damage" coagulation factor deficiency; (b) more falls; (c) more involvement in fights]
(ii) countercoup injuries.

d. Types

Divided in 3 types according to time interval between trauma and onset of symptoms [apathy, change in behavior, confusion, dizziness, drowsiness, headache, lethargy, nausea and vomiting, seizures, weakness].

(1) **Acute** – immediately to 3 days
(2) **Subacute** – 3 days to 3 weeks
(3) **Chronic** – > 3 weeks.

i. Acute SDH

Cause –

(1) rupture of (a) large bridging veins (b) one of the cortical arteries (c) larger dural sinuses – usually torn by penetrating wounds or depressed fractures. Produce clots in unusual positions, eg posterior fossa underneath the occipital lobes or between the hemispheres over the corpus callosum.

(2) cerebral lacerations.

ii. Subacute SDH

(1) Occurs when bleeding is from smaller bridging veins.

(2) Associated brain damage is less likely

(3) Blood film is thin and watery due either to dilution with CSF or hemolysis.

iii. Chronic SDH

(1) **Location** –

(i) Over frontal and temporal [extending to base],

(ii) near midline [may be bilateral] and

(iii) parietal lobe

(2) **Extent** –

(i) localized and deep, or

(ii) as a widespread film on surface

(3) **Epidemiology** - Seen in old persons. Etiology"brain atrophies in old age"more space in SDH"vessels traversing it have less cushioning"break more easily

(4) **Time course of development** – please see dating of SDH below.

e. Sequelae

(1) **Cerebral shift and herniation** [mostly transtentorial].

(2) **Encapsulation** - SDH may develop its own membrane formed by dural cells. The capsule is firmly attached to the dura. Arachnoid does not take part in this encapsulation.

(3) **Extramedullary hematopoiesis** – may occur rarely in SDH. Evidence is the presence of nucleated RBCs in various stages of maturation in the patient's subdural space.

(4) **Flattening of cerebral gyri:**

(i) Same side – due to direct pressure

(ii) Opposite side – displacement of cerebral hemispheres to the opposite side"gyri of opposite side are pressed against the bone.

(5) **Fresh bleeding** – Causes

(i) SDH may draw in water due to osmosis" expands"compresses brain tissue"causes new bleeds by tearing other blood vessels.

(ii) Original clot gets absorbed"chemical changes occurring in it cause a further hemorrhage to occur in it

(iii) Further trauma.

(6) **Infarction** – must be differentiated from infarction due to stroke [**Table 4**].

(7) **Organization –** Ossification or hyalinization may occur rarely. Symptoms - focal epilepsy, progressive hemiplegia.

(8) **Reabsorption of clot**

(i) Smaller SDH- completely reabsorbed with no trace

(ii) Larger SDH - replaced with granulation tissue.

(9) **Secondary brain hemorrhage** – due to compression of brain stem

(10) **vasoconstrictors** may be released from SDH" ischemia under SDH"ischemic cascade"brain cell death.

Table 4: Differences between infarction due to SDH and stroke

S. No	Feature	Infarction due to SDH	Infarction due to stroke
1.	Location of infarct	Generally directly beneath the SDH, but rarely may be away from it	anywhere
2.	Age of infarct	Same or less than the age of SDH	If older than SDH, it is due to stroke
3.	Evidence of primary disease	Trauma, evidenced by fracture, lacerations, bruises etc	Disease of cerebral arteries [eg atheroma]

f. Dating of SDH

(1) **24 h:**

(i) SDH consists of - partially liquid and partially soft fragments of red-black, gel-like, clotted blood.

(ii) Adherence to inner dural surface - (a) Most clots do not adhere (b) Some clots that do adhere are loose and slide along the surface when pressed.

(iii) Microscopy - (a) No cellular reaction from dura (b) RBC – intact (c) Leucocytes – trapped (d) Fibrin – in discontinuous layers between the dura and the hemorrhage

(2) **24-48 hours:**

(i) Color – Reddish brown with -ed fibrin deposition

(ii) Neutrophils invade hemorrhage

(iii) Proliferation of fibroblasts at the interface of the dura and hemorrhage

(3) **2-5 d:**

(i) -ed presence of the above

(ii) soft clots – stick loosely to dura; resist pressure to move them along surface

(iii) Microscopy - (a) Endothelial proliferation (b) scattered macrophages are present in the clot (c) Occasional spindle cells invade clot from dura (d) RBCs – (I) Early RBC breakdown (II) hemosiderin granules are demonstrable (III) Margins of clotted RBC - become indistinct (e) Neomembrane – Appears on the outer [dural side] (I) 3-4 cells thick closer to day 3 (II) up to 7 cells thick closer to day 5

(4) **1 week:**

(i) Outer neomembrane is yellow; grossly visible around the edges of clot

(ii) Inner membrane [arachnoid side] has not yet appeared

(iii) Coagulated blood is orange brown. Definitely adheres to dura

(iv) Microscopy - (a) Newly formed capillaries enter the hemorrhage along with the more abundant fibroblasts, giving the appearance of a typical granulation tissue (b) More intracellular hemosiderin accumulates (c) Laking [hemolysis] of RBCs

(5) **2-3 weeks:**

(i) Outer [dural] neomembrane thickens and becomes more vascular. Proceeds to partially cover inner surface of clot, which is beginning to liquefy

(ii) Clot – orange to yellow brown

(iii) Foci of red-black blood are common. Reflect the tendency of granulation tissue to bleed

(iv) Microscopy - (a) day 10 - Neomembrane is up to 15 cells thick (b) Up to 14 days - (I) Hemosiderin-laden macrophages (II) Neomembrane – thickness [twice that of the native dura]. Becomes more vascular. Hugely dilated capillaries (c) Up to 21 days - (I) Hemorrhage is absorbed with rare RBCs remaining (II) More obvious vascular proliferation (III) Inner neomembrane – thin and incomplete (IV) Outer neomembrane - mostly loosely arranged fibrovascular tissue

(6) **4 weeks:**

(i) Outer [dural] and inner [arachnoidal] membranes are complete and grossly visible. Outer membrane is approximately the thickness of the dura

(ii) Most blood is now semiliquid and orange brown

(iii) Collagen is deposited

(iv) Formation of arteries

(v) If there was an arachnoidal laceration, SDH adheres to it, otherwise not.

Arachnoid does not contribute to the process of organization.

(7) **1-6 months:**

(i) Rare hemosiderin-laden macrophages

(ii) Fusion of neomembrane and dura

(iii) Blood is mostly resorbed, leaving a thin gold colored membrane adherent to dura

(iv) Rare blood vessels

(8) **6-12 months:**

(i) Thin neomembrane that is difficult to distinguish from native dura

(ii) Hemosiderin-laden macrophages still present.

g. Subdural hygroma

A ***subdural hygroma*** [Gk, hygros, moist; oma, tumor] [syn, hydroma] is a subdural collection of CSF without blood.

Salient features:

(1) **Causes:** (i) Arachnoid cyst rupture (ii) CSF leakage through an arachnoid tear [valve mechanism] (iii) Entry of microorganisms into the subdural space "inflammatory changes with a serous reaction (iv) Post-operative - (a) After ventricular drainage [frequently bilateral] (b) Failure of the brain to re-expand - after surgical resection of a long-standing mass lesion (c) excessive drainage - during the implantation of a CSF shunting device.

(2) **Significance** – Subdural hygromas on CT scans may be misinterpreted as chronic subdural hematomas, and vice versa.

h. Cause of death

Secondary pressure on brain stem"stoppage of respiration.

i. PM examination

Unlike EDH, the bleeding points are so small that they cannot be discovered at operation or PM.

j. MLI

Lucid interval – Seen in subdural hematoma also. If accompanied by cerebral contusion, a lucid interval is relatively less common because the primary disturbance of consciousness persists far longer.

3. Subarachnoid hemorrhage

Subarachnoid hemorrhage is bleeding into the subarachnoid space. As the bleeding clots and forms a large firm mass it is often referred to as ***hematoma***. The latter is more often used when it is present over the spinal cord [spinal subarachnoid hematoma].

a. Anatomy

(1) **Subarachnoid space [SAS]** - Unlike extradural space, subarachnoid space – space between arachnoid and the very thin pia – is a real space.

(2) Pia dips up and down along the surface of gyri and sulci, while arachnoid doesn't.

(3) **Contents of SAS** –

(i) Brain bv

(ii) Connective tissue network

(iii) Cranial nerves

(iv) CSF – produced by choroid plexuses of lateral and 4^{th} ventricles.

b. Arachnoid cysts

Arachnoid cysts are CSF filled collections that occur between two arachnoid layers.

c. Causes

(1) **Trauma** - Most significant cause of SAH. SAH is further classified in 3 subtypes:

(i) brain injury associated [seen in association with contusions and lacerations of brain and pia-arachnoid]

(ii) traumatic rupture of basal cerebral vessels [ruptures normal basal vessels or a preexisting berry aneurysm]

(iii) traumatic focal [bleeding occurs only in focal areas].

(2) **Non-traumatic** (Spontaneous) - please see **ch 8**.

(3) **Hanging** – **ch 19**.

d. Epidemiology

(1) Incidence – Most common ICH. In fact in all cases of significant head injury some degree of SAH is seen.

(2) Associations - May be seen alone or with other craniocerebral injuries, including fractures.

(3) Like all delayed traumatic hemorrhages, it can also be delayed after trauma.

e. Clinical aspects

(1) **Symptoms** -
(i) severe headache with a rapid onset [“thunderclap headache”]
(ii) vomiting,
(iii) confusion or a lowered level of consciousness,
(iv) seizures
(2) **Diagnosis** - confirmed by a CT scan or LP.

f. Microscopic dating of SAH

(1) **<1 hour** - Fresh blood in subarachnoid space
(2) **1-4 h** -
(i) Occasional neutrophils seen
(ii) Some RBCs begin to break down
(iii) RBCs begin to creep down the Virchow–Robin spaces
(3) **4-12 h** -
(i) -ed neutrophils
(ii) Perivascular lymphocytes
(iii) Rare macrophages
(4) **12-24 h** -
(i) Hemosiderin and fibrin
(ii) -ed numbers of lymphocytes and macrophages
(5) **24-48 h** -
(i) -ed neutrophils and macrophages
(ii) Definite hemosiderin deposition
(6) **Up to 3 days** - Peak neutrophilic infiltrate
(7) **Up to 5 d** -
(i) Laking of RBCs
(ii) -ed lymphocytes
(iii) Intense fibrin deposition separating islands of RBCs
(iv) Early collagen formation.

4. Intracerebral hemorrhage

Intracerebral hemorrhage [ICH] is a subtype of intracranial hemorrhage that occurs within the brain tissue.

Salient features:

(1) **Timing in relation to injury** – Most ICH occur at the time of accident, but bleeding is often slow due to oozing from venules and capillaries. As hematoma expands, it injures more vessels and more bleeding occurs. Rarely bleeding may occur several weeks or even months after trauma during which brain softens ultimately rupturing the vessel.

(2) **Location:** (i) Relatively deep seated traumatic ICH - usually accompanied by other types of injuries, eg cortical contusions and lacerations. (ii) Superficial traumatic ICH - generally occur alone.

(3) **Symptoms** – patient becomes deeply unconscious from the moment of injury.

a. Causes

(1) Angioma or malignant tumor of brain

(2) **Capillary hemorrhages** – seen in Asphyxial states, Blood dyscrasias, Fat embolism, Softening due to Anoxia, Arterial thrombosis, Sinus thrombosis

(3) Cerebral trauma

(4) **Hypertensive cerebrovascular disease** – Generally due to rupture of lenticulostriate artery. Causes spontaneous hemorrhages in the region of basal ganglia. Common in middle aged and elderly persons. Other areas of hemorrhage are thalamus, external capsule, pons and cerebellum.

Table 5: Differences between extradural, subdural and subarachnoid hemorrhage

S. No	Feature	EDH	SDH	SAH
1.	Location is between	skull and dura	Dura and arachnoid	Arachnoid and pia
2.	Cause	Mostly traumatic. Rarely spontaneous, eg in coagulation disorders	Mostly traumatic. Could also be due to anticoagulants, iatrogenic, metabolic disorders etc	Both natural and traumatic
3.	Incidence	Least common of all meningeal bleeds. 1-2% of treated head injuries and 5-10% of autopsies	Commoner than EDH. **5%** of all head injuries; **50%** of fatal head injuries.	Commonest of all intracranial bleeds.
4.	Vessels involved	Middle meningeal artery (posterior branches)- most common. Dural venous sinuses	Bridging veins, cortical contusions. Dural venous sinuses	basal cerebral vessels
5.	Extent	Mostly localized on one side, generally beneath pterion or over superior sagittal sinus. Could rarely be bilateral.	Generally diffuse	Much more diffuse than SDH. If due to berry aneurysm, then only on the base.
6.	D/d	Heat hematoma	None	congestion
7.	Effects	Space occupying lesion	May sometimes be space occupying	Rarely space occupying
8.	Distortion of brain surface	Gyri and sulci ironed out	Surface affected less	Not affected
9.	Lucid interval	Seen as a classic case	Seen, but less often	Not seen
10.	Preservation for toxicology	If survival is longer, a very valuable piece for analysis, as toxic material from blood may have been metabolized	May be saved in a similar way as an EDH and under similar circumstances	Generally not useful

5. Intraventricular hemorrhage

An ***intraventricular hemorrhage*** [IVH] is a bleeding into the brain's ventricular system.

Salient features:

(1) **Types**: (i) Primary - (a) bleeding is confined to the ventricular system. (b) Uncommon [3% of all spontaneous intracerebral hemorrhages (ICH)]. (c) median age - 55 y [range 9 to 91 years] (d) Sex - Males and females are equally represented (e) 50% patients have a history of hypertension (f) Causes – Mostly non-traumatic. (I) Coagulopathies, acquired or inherited (II) Fibromuscular dysplasia (III) Intraventricular aneurysms. (IV) Intraventricular tumors (V) Moyamoya disease (VI) Pituitary apoplexy (VII) Puerperal toxemia (VIII) Sympathomimetic abuse [eg methamphetamine] (IX) Vascular malformations [usually AV malformations or fistulae] (X) Vasculitis (ii) Secondary – (a) More common. (b) An intracerebral or subarachnoid hemorrhage occurs first which bleeds into the ventricular system. (c) IVH

complicates 40% of ICH and 10% of SAH cases.

(2) **Source of bleeding** – choroid plexus, vessels of septum pellucidum.

D. Respirator Brain

Respirator brain is a characteristic brain change, which occurs when brain stem death has occurred and cerebral circulation has ceased, but the patient has been kept on a respirator for >12 h.

E. Sequelae of Head Injury

1. Head injury and offending

(1) Head injury is linked to -ed rate of subsequent offending.

(2) **Evidence:**

(i) There is -ed irritability, aggression, impulsivity and lack of foresight, which predispose to offending.

(ii) Follow-up studies of those who have suffered head injury indicate -ed rates of arrest, conviction and incarceration post injury.

(iii) There is an -ed rate of past head injury in samples of people who have committed violent crimes.

2. Post traumatic automatism

Post traumatic automatism refers to a disturbance of consciousness, associated with ***automatic***, ***repetitive***, and ***uncontrollable behavior*** that occurs after head injury.

Salient features:

(1) There is subsequent **amnesia** for the automatic events.

(2) Most typical signs are: (i) Thrashing or flailing limbs in a non-directed manner (ii) Refusing to be directed [eg onto a stretcher or into an ambulance] (iii) Wildly swinging arms if directly confronted or restrained (iv) Aimless wandering in a “robotic” fashion.

(3) Post traumatic automatism may be advanced as a **defense** by the offender, if he has **committed a crime** (say a murder). A successful claim of automatism results in an absolute acquittal. Before certifying, the forensic specialist should note following points. (i) There should be a **documented concussive brain injury**. (ii) The automatism must occur in the stage of **post-traumatic amnesia**. (iii) The automatism behaviour must represent a reactive and

purposeless response to stimulation or confrontation. (iv) **No premeditation**, planning, or concealment should be demonstrable. (v) There should be **absence of goal directed, purposeful, or proactive behaviour**.

3. Post traumatic amnesia

Post-traumatic amnesia (***PTA***) is characterized by a transient state of confusion and disorientation following head injury. The hallmark of this state is **anterograde amnesia** (AA), which is the impaired ability to remember events after the trauma.

Interrogation of persons having sustained head injuries

Because of the possibility of **retrograde amnesia**, it may not always be fruitful for police to interrogate a victim who has sustained head injuries. **Leading questions should not be asked** from him.

4. Increased intracranial pressure

-ed intracranial pressure is seen after

(1) Abscesses and tumors of brain and its coverings
(2) Diffuse cerebral edema
(3) Dural sinus thrombosis [**ch 5**]
(4) Hemorrhages [Cerebral hemorrhage, Extradural and subdural hemorrhage]
(5) Infarction of brain
(6) Leptomeningitis.

5. Miscellaneous

(1) Aneurysms,
(2) Arterio-venous fistula,
(3) Dural sinus thrombosis [Central Venous sinus thrombosis] [DST, CVST **ch 5**].

III. INJURIES OF THE SPINAL CORD

A. Railway Spine

Railway spine (concussion of the spinal cord) is the name given to a constellation of neurological signs and symptoms, appearing after a railway accident.

Salient features:

(1) These were very common in early days of railway travel (19^{th} c), because the

railways were not specially designed for passenger safety.

(2) Since no external injuries were noted on the victims, it was thought that the symptoms were due to concussion of the spinal cord.

1. Cause

Railway accident (generally due to heavy forces involved in railway accidents). May also be seen in

(1) other vehicular accidents

(2) severe blows to the back

(3) compression from dislocation or fracture of vertebrae

(4) fall from height

(5) bullet injury.

2. Mechanism

Concussion of the spinal cord, caused by

(1) momentary collision of the cord against the wall of the canal, or

(2) transient deformity in the profile of the canal due to violent deceleration or

(3) rotational strains.

3. Symptoms

(1) **Time of appearance** - Appear immediately or after a few hours. Sometimes after several days

(2) **Symptoms** - Mainly neurological: (i) headache and giddiness (ii) restlessness (iii) weakness of limbs (iv) Paralysis (v) back pain (vi) urinary and/or fecal incontinence (vii) loss of sexual power (viii) disorders of special senses (ix) insomnia (x) amnesia (xi) failing memory.

(3) **Duration** - Usually transient. Classically they used to disappear after compensation was granted. This gave rise to a widespread suspicion that this condition was merely “invented” by some interested parties in order to get money.

(4) **Current concepts** - It is now known that these symptoms represent Posttraumatic Stress Disorder (PTSD) [**ch 11**"Trauma and neurological disease].

4. Treatment

Conservative.

5. PM Appearances

Nothing specific. There may be petechial hemorrhages in spinal cord. Mostly railway spine results in negative autopsy [**ch 8**].

B. Whiplash Injury

Whiplash injury is an injury to the neck caused by sudden hyperextension of the neck followed by hyperflexion or vice-versa.

(1) **Mechanism** – Caused due to sudden and violent hyperflexion and hyperextension of the spine. Of these two, hyperextension is more dangerous because flexion injuries are protected by the contraction of strong posterior neck muscles. In comparison, the anterior longitudinal ligament which protects from injuries during hyperextension is quite weak.

(2) **Injuries:**

(i) Dislocation of upper cervical spine

(ii) Fatal contusion and laceration of spine without fracture of spine

(iii) Fracture dislocation in lower spine (C5-6)

(iv) Fracture dislocation in the upper dorsal spine (T5-7).

(3) **Causes:**

(i) **Motor vehicle accidents** – Most common [**ch 18**]. (a) Stationary vehicle struck from rear (most common)– Sudden hyperextension of the head followed by sudden flexion [to prevent hyperextension, modern cars now have a head-rest at the top of the seat] (b) Moving vehicle crashing into a stationary object – Hyperflexion of head"strikes windscreen"Rebounds and goes into hyperextension

(ii) **Rabbit punch** – Rabbit punch is a sharp blow to the back of neck [spinous process of an upper cervical vertebra] or to the base of the skull. Because of the fear of whiplash injury, the rabbit punch is illegal in boxing and many other combat sports. The name is derived from the use of the technique by hunters to kill rabbits with a quick, sharp strike to the back of the head.

(4) **Clinical presentation** - (i) cognitive/psychological symptoms (ii) Dizziness (iii) Headaches (iv) Neck, shoulder, or back pain (v) paresthesias (vi) vertigo. If these symptoms persist for some months, it is known as **chronic whiplash injury**.

IV. INJURIES TO BONES

A. Fractures

1. AM and PM fractures

Table 7: Differences between AM and PM fractures

S. No	Feature	AM fracture	PM fracture
1.	Effusion of blood	Present	Absent
2.	Muscles around fracture	Lacerated, edematous, contused. Blood penetrated farther in muscles, as fracture gets older	Not so
3.	Signs of healing	Present	Absent
4.	Histology	Active cellular infiltration	Not so
5.	Sequelae	Fat embolism	Not so

2. Age of fractures

Age of fractures can be estimated by

(1) **Naked eye and histological examination** [in the dead] and by

(2) **Radiological examination** [in the living]. Fractures heal in **4 stages**.

(i) **Stage of inflammation** – Lasts up to 7 days **[total 1 week]**

(ii) Stage of **soft callus formation** (or reparative or proliferative phase) – 1 week to 4 weeks **[total 3 weeks]**

(iii) Stage of **hard callus formation** [or maturing or modeling phase] – 4 to 12 weeks **[total 8 weeks]**

(iv) Stage of **remodeling** (or remodeling phase) – **[up to 2 years]**

(v) Rarely formation of false joints.

V. BOXING INJURIES

1. Punch-drunk syndrome

Punch-drunk syndrome [**syn** - boxer's syndrome, chronic traumatic encephalopathy, Dementia pugilistica, pugilistic Parkinson's syndrome] is a ***neurological disorder*** which affects professional boxers and others who receive multiple serious blows to the head.

Salient features:

(1) Most important chronic boxing injury

(2) **Onset** - The condition develops over a period of years, with the average time of onset being about ***15 years*** after the start of a career in boxing.

(3) **Number of years** the boxer has been in profession is ***more important*** in determining its onset, than the **number of serious blows** the boxer received. It is because even minor, unnoticed traumatic events are important.

(4) **Symptoms** - The major symptoms are (i) ataxia (ii) dementia (iii) memory impairment (iv) slow thought process (v) slurred speech (vi) stiff limbs (vii) tremors [much like those seen in Parkinsonism], and (viii) outbursts of violence. The condition is called "**punch-drunk**" because the symptoms are similar to those shown by a **drunk person**. Famous boxers who have suffered from this syndrome include Jack Dempsey, Beau Jack, Joe Louis and Muhammad Ali.

(5) **utopsy** shows (i) Cortical atrophy (ii) perforation of septum pellucidum (iii) slight hydrocephalus (iv) thinning out of fornices and adjacent corpus callosum (v) presence of scars and patches of gliosis throughout the brain tissue.

2. Boxer's hemorrhages

Pontine hemorrhages.

3. Post-traumatic hypopituitarism

Due to direct injury and vascular problems.

4. Other cerebral injuries

(1) **Cavum septum pellucidum** (CSP) - Septum pellucidum develops a separation between its two leaflets (septal laminae). This cavity contains CSF. It was once called the "fifth ventricle", but the term is not favored now, because CSF in CSP is noncommunicating (2) diffuse axonal injury, (3) cerebral edema (4) ischemia and herniation (5) cerebral atrophy (6) enlargement of the ventricles

(7) cerebellar changes (8) loss of neurons in the substantia nigra (9) chronic encephalopathy (10) dissecting aneurysms.

5. Ocular injuries

(1) choroidal tears (2) Retinal detachment (3) vitreous hemorrhages.

18. Traffic Accidents

AUTOMOBILE ACCIDENTS

A. Injuries to Pedestrians

Three types of injuries are seen:

(1) ***Primary impact injuries*** are caused by the initial strike, i.e, the first part of the vehicle that strikes the victim (usually legs). The victim then revolves round his center of gravity (located at symphysis pubis), and is thrown back on the vehicle, causing further injuries.

(2) These are known as ***secondary impact injuries***.

(3) Then the victim is thrown on the ground. This causes ***secondary injuries*** [**Fig 18.1**].

(4) Finally the victim may be run over by the vehicle, causing crush injuries. A diagram indicating the three types of injuries, their extent and location can help reconstruct the accident.

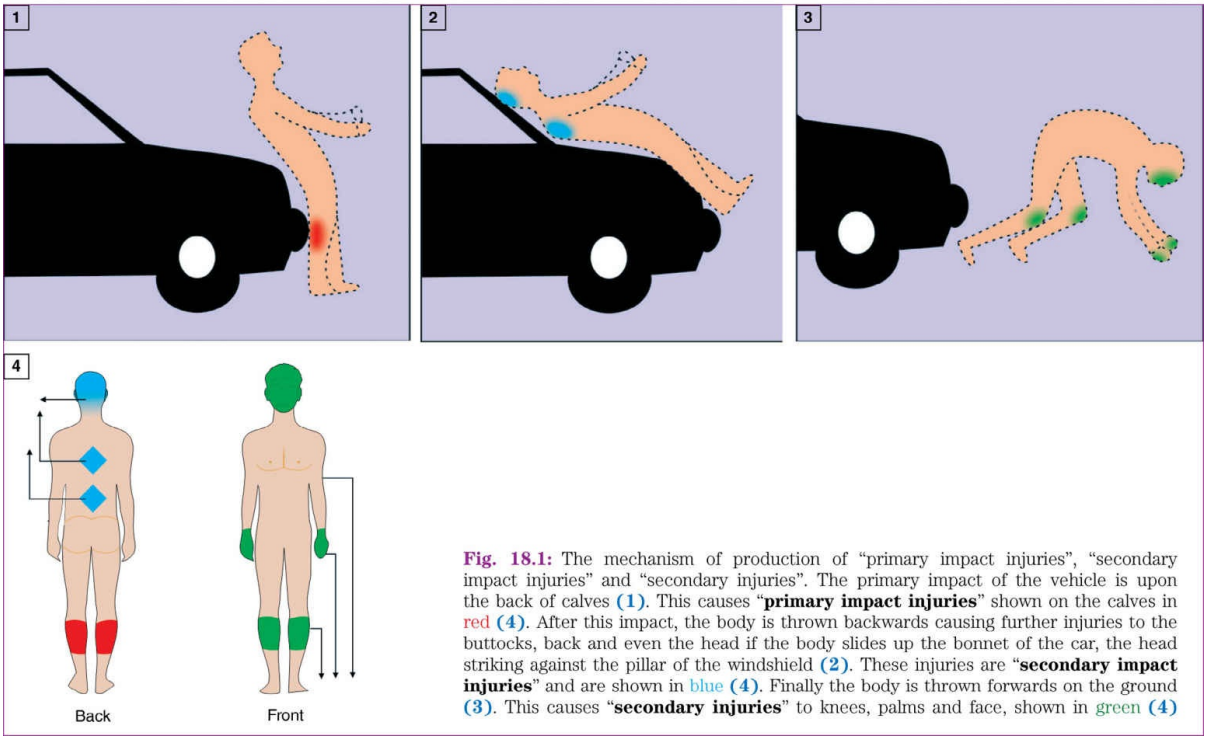


Fig. 18.1: The mechanism of production of "primary impact injuries", "secondary impact injuries" and "secondary injuries". The primary impact of the vehicle is upon the back of calves **(1)**. This causes "**primary impact injuries**" shown on the calves in red **(4)**. After this impact, the body is thrown backwards causing further injuries to the buttocks, back and even the head if the body slides up the bonnet of the car, the head striking against the pillar of the windshield **(2)**. These injuries are "**secondary impact injuries**" and are shown in blue **(4)**. Finally the body is thrown forwards on the ground **(3)**. This causes "**secondary injuries**" to knees, palms and face, shown in green **(4)**

1. Primary impact injuries

Part of body struck – Depends upon

(1) **Position of person** - in relation to the vehicle when struck, i.e. walking with the traffic, against the traffic or crossing the road. If walking with the traffic, he would be struck from behind, and back of legs are struck. Following situations are most important.

a. Pedestrian struck from behind; both feet fixed to the ground

Whether the feet are fixed or not fixed depends upon nature of the road surface; whether it is slippery or not; whether it has rained or not. Injuries produced are

(1) **Bumper injuries** - Injuries at site of bumper impact, in the form of abrasions, contusions, lacerations, internal haemorrhage in the calves etc.

Bumper fractures - part of bumper injuries. Most characteristic fracture in vehicular accidents. Characteristics - (a) Usually tibia is fractured, although very rarely fibula may also fracture. (b) The fractured fragment of tibia is **wedge** shaped, and is displaced forwards. The base of the wedge indicates the site of impact, and the apex points in the direction in which the vehicle was travelling. (c) If bumper fractures are only in one leg or in both legs but at different levels, it suggests victim was walking or running (d) In children – the

bumper usually produces fracture of femur. Femoral head may be driven through acetabulum (e) Identification of vehicle – Height of bumper fracture from the ground = height of bumper of offending vehicle (f) Behavior of the driver of the vehicle – (I) When brakes are applied - height of bumper dips down. Thus height of bumper fracture is less than the height of the bumper of offending vehicle. (II)when accelerator is pressed - height of bumper goes up. Height of bumper fracture is more than the height of the bumper of offending vehicle (g) Sometimes the fracture may be spiral.

(2) **Impact against mudguard or headlamp:**

(i) fracture of pelvis, Pubic ramus fracture

(ii) fracture dislocations of the sacroiliac joints.

(iii) Imprint abrasions - Due to head lamps and radiators

(3) **Vehicle type:**

(i) If vehicle has a **straight and high front end** [eg bus, trailer, truck, van], the entire body may come in contact with front end at once.

(ii) Injuries - would depend on how victim was positioned [including positions mentioned below]. (a) Frontal impacts- Head injuries, chest injuries, fracture of ribs (b) Side impacts – Injuries [lacerations etc] of arms (c) Rear impacts - (I) injuries to buttocks and sacroiliac joints (II) Fracture-dislocation of the lumbar or thoracic spine, sacroiliac joint (III) The fractured portion of the vertebral column may move forward causing **transection of the spinal cord** and **thoracic aorta**. (IV) Associated fracture of adjacent ribs. **Secondary impact injuries** are virtually non-existent in such cases, as there is no way a victim can recoil back on the vehicle. He instead is pushed to the ground and suffers **secondary injuries**.

(4) **Protruding objects** – from vehicles may cause serious, and often characteristic injuries.

2. Secondary impact injuries

(1) If feet slide forward, the whole body falls backwards, with a secondary impact of the head against the windshield [**Fig 18.1**].

(2) **Injuries:** If victim falls on hood – Tangential force directed by hood to the buttock and thigh"Causes separation of the skin and s/c tissues from the muscle"produces a pocket in the upper thigh and buttock"Collection of large amount of blood in this pocket, which is often not visible externally

(3) If feet not firmly fixed on ground and vehicle is at high speed - The victim is "scooped up", i.e. thrown into the air. He may land over the roof of the vehicle, somersaults [head may strike roof] and may even be deposited on the road behind the vehicle, where he may be run over by other vehicles. Atlanto-occipital dislocation and partial disruption of intervertebral discs are quite common in this situation.

3. Secondary injuries

(1) **Due to striking of victim on ground:**

(i) May be caused after secondary impact injuries, or

(ii) immediately after primary impact injuries, when the victim is thrown high up in air and strikes the ground.

(2) **Injuries:**

(i) All kinds – including abrasions, contusions, lacerations, fractures etc

(ii) fracture skull – due to victim falling over road

(3) **Waddell and Drucker's triad** [syn, Waddell's triad]-

(i) Primary impact, secondary impact and secondary injuries may together constitute the Waddell and Drucker's triad.

(ii) The injuries described in the original paper were (a) Injury around the knee [**primary impact by bumper**] (b) Injuries to proximal femur or pelvis [**secondary impact inj**]. (c) Contralateral craniocerebral injury - [**secondary inj**].

4. Run over injuries

(1) **Abrasions:**

(i) Grazes

(ii) Impact or imprint abrasions – in the form of tire marks. They are spread out a little due to yielding and flattening of body from pressure.

(2) **Avulsions** [**ch 12**]:

(i) The rotatory effect against a fixed limb may strip off almost all tissues down to the bone.

(ii) The avulsed wound may be segmental or circumferential [completely encircling arm or leg]

(iii) If head is involved, complete avulsion of an ear may occur

(iv) If intestines or scrotum are involved – extrusion of intestines or testes occurs

(3) **Head injury with egg-shelling of skull** – skull may be crushed from sided to side or forced open with extrusion of brain.
(4) **Multiple fracture of ribs** – if tire passes over chest
(5) **Abdominal rupture** – with extrusion of contents [if tire passes over abdomen]
(6) **Crushing and hemisection of body** - if vehicle is very heavy [eg truck] and tire passes through middle of body, eg abdomen
(7) **Crushing of legs** – if tire passes over legs
(8) **Burns** – due to
(i) heat produced due to friction consequent upon dragging
(ii) Exhaust gases
(9) **Special situations:**
(i) If brakes are applied – wheels will lock"Shearing action of tangential force"Long lacerations, complete amputation of a limb, decapitation
(ii) If victim is a child – fractures may not occur because of elasticity of cartilaginous skeleton.

5. Rolling over injuries

(1) **Mechanism** - Produced when a vehicle with low chassis “rolls” the victim along the roadway, instead of “running him over”.
(2) **Injuries:**
(i) Abrasions – (a) Graze abrasions (b) Patterned abrasions – caused by parts on the undersurface of chassis
(ii) Burns - from exhaust system
(iii) Fractures – of bones all over body
(iv) Grease soiling
(3) **Site** - Present circumferentially all round the body [eg graze abrasions circling around body].

B. Injuries to Vehicle Occupants

Injuries would depend upon type of impact. 2 broad types are recognized – Non ejection and ejection injuries.

1. Non-ejection injuries

a. Frontal impact

(1) **Most common** - About 80% of vehicular accidents are frontal in nature, eg two vehicles colliding head-on or vehicle striking a fixed object, such as a lamp post, tree or a wall, causing violent deceleration.

(2) **Sequence of events upon frontal impact** – Driver and passengers receive some common [deceleration injuries] and some different set of injuries [due to differences in secondary collision].

i. Driver

(1) Secondary accident - The driver (if not wearing seat belt) slides forwards so that his legs strike the fascia/parcel-shelf area [instrument panel and dashboard area], and his chest and lower abdomen strikes the lower edge of the steering wheel. This is known as second collision, second impact or secondary accident.

(2) Facial impact on windscreen – windscreen injuries of face [imprint abrasions, bruises, fracture jaws, facial bones;

(3) Flexion across steering wheel - The body flexes across the steering wheel and begins to rise. Causes steering wheel injuries on the chest and abdomen [chest contusions, bilateral rib fracture, lacerations of liver]. Modern cars are fitted with energy absorbing compressible steering wheel column"results in less serious steering wheel injuries.

(4) Rarely driver’s chin may be suspended against steering wheel causing accidental hanging [**ch 19**]

(5) Flexion of spine - The head [which is heavier] goes forwards causing flexion of the cervical and thoracic spine. If followed by hyperextension, results in **whiplash injuries** [**ch 17**].

(6) Head strikes windscreen - The upward and forward component of the force of impact causes the head to strike the windscreen. Thus windshield is prevented from shattering and drivers [and other occupants] may sustain only relatively minor incised wounds. These are typically vertically oriented and clustered on the forehead.

(7) Windscreen broken and body ejected - The windscreen may be broken due to impact by the head or face, and the whole body may be ejected through the broken glass, to land on the bonnet or even on the roadway ahead.

(a) Air bag injuries

Salient features:

(1) Although airbags . the incidence of fatal injuries, ironically they themselves may be cause of serious injuries.

(2) **Epidemiology:**

(i) Incidence - At least one airbag related injury occurs in 43% of airbag deployments.

(ii) Age - Adults of short stature and children are particularly liable

(iii) Severity - (a) The majority [96%] of injuries are minor, eg lacerations, thermal, mechanical, or corrosive burns of the skin and the eyes. (b) Only 0.2% are considered as serious lesions. The risk of serious injuries or fatalities is particularly high in children sitting in the front passenger seat [chest, abdomen, or cervical spine traumas].

(3) **Fatal lesions** – Caused by impact of chest against air bag. Multiple rib fracture with bilateral hemothorax, SDH, laceration of pericardium and right atrium with hemopericardium, retropharyngeal hematoma with airway obstruction.

(4) **Non fatal lesions:**

(i) Ocular injuries – Most common. Caused by impact of face against air bag, or the projection of an object [eye glasses, tobacco pipe] into the face

(ii) abrasions, contusions, lacerations of face, chest and limbs

(iii) Orthopedic injuries – (a) Mainly occur in upper and lower limbs [for lateral airbags] (b) fracture of ribs and sternum (c) pulmonary contusions (d) cardiac trauma (e) spinal lesions [eg cervical spine fracture] -less common. (f) Forearms are most frequently involved, especially on the distal third. (g) Hand lesions - usually minor and rare.

(b) Deceleration injuries

Deceleration injuries include a variety of thoracic injuries resulting when the moving thorax decelerates rapidly as a result of impact against a stationary or relatively stationary object.

Salient features:

(1) **Injuries:** (i) aortic injuries [laceration, transection]. (ii) sternal fracture (iii) flail chest (iv) myocardial injuries – (a) Contusions and lacerations (b) contusions of pericardium and myocardium may be seen even without fracture of ribs. (c) Posterior surface of heart – shows injuries from impact against the spine (d) Avulsion – May be completely avulsed from its base and may be lying loose in the chest cavity (e) Lacerations – of auricles and ventricles.

Either from fractured ribs and sternum, or through external objects (v) Tracheobronchial disruption (vi) Lungs – (a) Vertical line of bruising on posterior surface – due to blunt impact of lung against vertebral column (b) Air bullae and blood blisters – under the pleura, overlying the bruised areas

(2) **Aortic injury:** (i) "classic" deceleration injury (ii) Location - aortic isthmus, a few centimeters distal of the ostium of the left subclavian artery [Most common] (iii) Associated with large magnitude forces (iv) Most frequently seen in frontal and near-side crashes (v) Appearances – (a) Aortic rupture – circular, clean cut; appears as sharp as if transected with a knife (b) Ladder tears - multiple transverse intimal tears, adjacent to main rupture [**Fig 18.2**]. (vi) Mechanisms - (a) Differential deceleration - of the heart and aortic arch relative to the anchored segments of the thoracic aorta (b) -ed intravascular pressure and hemodynamic forces in vehicular crashes (c) severe whiplash effect on thoracic spine (d) Osseous pinch - Aorta is crushed or "pinched" between the vertebral column and the inner surface of the manubrium, first rib, and clavicle during anteroposterior thoracic compressive deformation. (I) Consistent location of aortic injury - Adequately explained, since the aortic isthmus will be compressed between the bony anterior thoracic structures and the fourth vertebral body. (II) Also provides an explanation for aortic injuries occurring in low-speed crushing injuries.

(3) **Two or more deceleration injuries** – are mostly fatal. Thus while in clinical cases, only one of the above types are seen, in autopsy cases, two or more than two are seen.

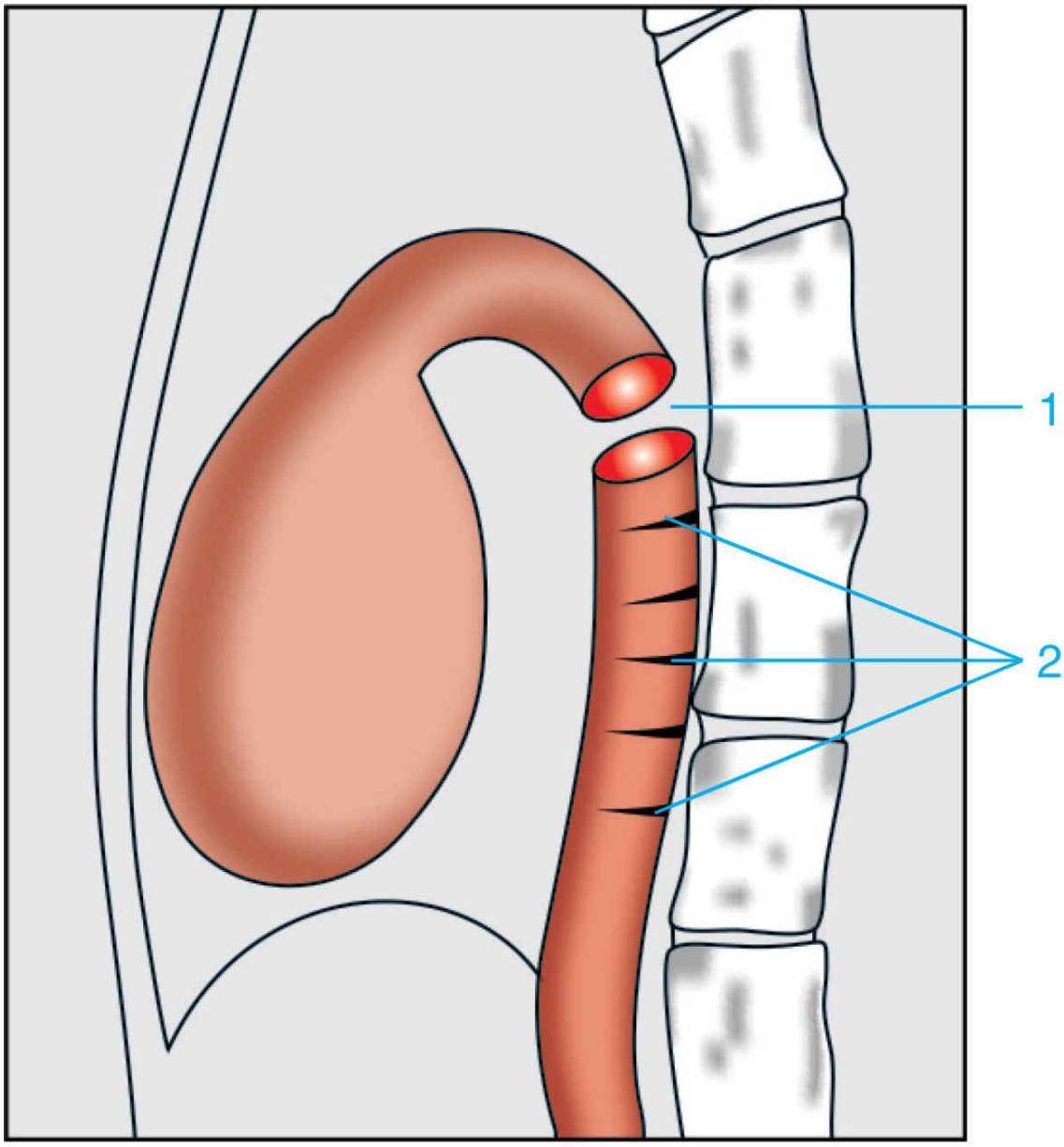


Fig. 18.2: (1) Main rupture site and **(2)** ladder tears in aorta.

(c) Seat belt injuries

Salient features:

(1) **Mechanism of protection:** (i) Restrains body – Keeps it away from potentially injurious objects like steering wheel and windscreen. (ii) Spreads deceleration force – over broad surface area of strap (iii) Reduces G-forces –

strap stretches during deceleration"time of deceleration -es, causing reduction of G-forces (iv) Prevents ejection

(2) **Reduction in risk of death** - 40%. **Nancy Cruzan** died because she was not wearing seat belts [**ch 8**].

(3) Although seat belts .the risk of aortic injuries, the same is not true of side impact crashes.

(4) **Seat Belt Syndrome** [SBS]: (i) Caused by lap-strap seat belts - Frontal collision"driver forced forwards violently"Jack-knifes over the lap belt at the waist"Injuries [**lap belt syndrome**] (a) Surface injuries to lower abdomen [abrasions, contusions, hematomas] (b) Injury to abdominal viscera at the midlumbar level [avulsion, contusion, perforation and tear of intestine; bowel transection, particularly near points of fixation, eg duodenum, suspensory muscle of duodenum [ligament of Treitz], cecum, ileocecal valve and root of mesentery; omental and mesenteric lacerations; lacerations of spleen, liver, pancreas, cecum and bladder; rupture of abdominal aorta, vena cava, common iliac artery, diaphragm, especially left hemidiaphragm] (c) fracture and dislocation of the thoracic and lumbar vertebrae [**Chance fractures**; **ch 17**] and spinal damage causing paralysis. Most characteristic "***triad of injuries***" associated with rapid deceleration against a fixed fulcrum is ***spinal trauma*** [Chance fractures (***ch 17***)], ***seat belt aorta*** (***ch 17)***, and ***bowel injuries***. (ii) Caused by Modern 3-point belts – These were developed to minimize the incidence of SBS. Abdominal injuries were .ed, but the diagonal strap introduced new injuries. It contributes to hyperflexion or hyperextension of the neck"fracture of cervical and upper thoracic vertebrae, Carotid laceration, Tracheal transection, Injuries to brachial plexus (iii) Accidental strangulation - Children may slide under the lap strap, and the diagonal strap passes across throat. May cause accidental strangulation [**ch 19**]. Other types of belts are much less common.

(5) **Strangulation by seat belts** - please see **ch 19**.

(d) Under-running

(1) If a small vehicle eg a car is following a larger vehicle eg a truck at a high speed [tail-gating], and truck suddenly stops, the smaller vehicle may continue to run underneath the larger vehicle, causing severe crushing of the car [under-running].

(2) **Occupants** – may receive severe crushing injuries. Their recovery is very difficult.

(3) **If tailgating vehicle is a motorcycle:**

(i) the motorcyclist's head and shoulders are smashed against the tailboard.

(ii) In extreme cases decapitation may occur.

ii. Front seat passengers

Same sequence as that seen in driver, except that they do not strike the steering wheel. They instead strike the dashboard.

iii. Back seat passengers

(1) **Mechanism:**

(i) violent deceleration

(ii) no seat belts

(2) **Injuries:**

(i) Most common - to head and knees, due to striking of head to front seat including head rests

(ii) Sometimes thrown over the front seat and strike the car's front structures, or even ejected through the windscreen.

b. Side impact

(1) **Frequency** - Second most common – after frontal collisions

(2) **Circumstances:**

(i) An offending vehicle strikes on the side of the victim vehicle

(ii) vehicle skids sideways into a fixed object

(3) **Place** - Commonly occurs at intersections; therefore frequent occurrence in urban areas

(4) **Injuries:**

(i) Severe; because side of car has thin metal walls. No other components to absorb impact

(ii) Occupant sitting on the struck side is injured most

(iii) Injuries caused by shattering of side-windows. (a) When driver [or other vehicle occupant] impacts one of these tempered glass windows during a crash, the glass breaks into fragmented cubes. (b) This may cause **dicing injuries** [caused by "dice" shaped glass fragments] on the face, shoulders, and arms. These are small linear, right angled, V-shaped or sometimes irregular cuts, lacerations or abrasions [**Fig 18.3**]. (c) Location of victims - A driver will have dicing injuries on the right side of the body and a passenger will have them on the left. In left hand driven vehicles, the

pattern would be opposite. May help locate position occupied by the victims in the car, if they were ejected out.

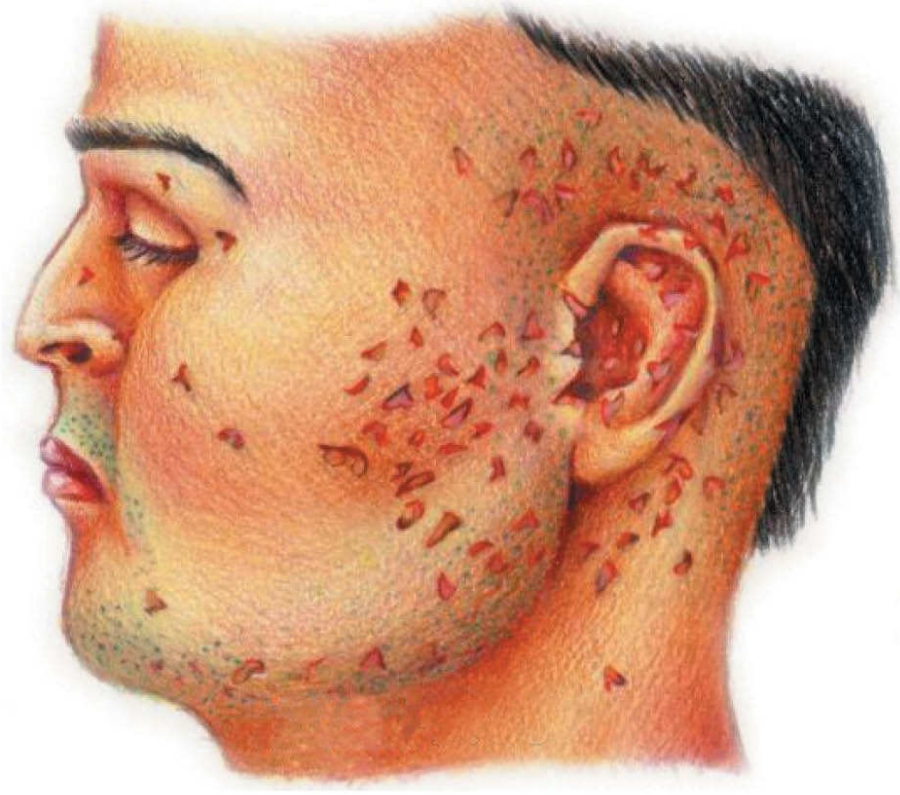


Fig. 18.3: Dicing injuries on the face, caused by striking of the head against windscreen

C. MLI [Automobile Accidents]

1. Manner of death

Most automobile accidents are accidental; few are suicidal and still fewer are homicidal. Pattern of injuries, and circumstances of death often indicate the manner.

a. Accidental

(1) Driver may stop - and may try to assist victim

(2) Hit and run - Driver looses nerve"drives away in order to avoid arrest.

b. Suicidal

(1) Automobile accidents can rarely be suicidal. Sometimes these may be masqueraded as an accident.

(2) Pointers:

(i) The typical scene is when an old depressed individual, suffering from incurable disease and chronic pain is found to have died following an accident.

(ii) He drives the car himself at very high speed, and strikes it against a tree.

(iii) No skid marks [indicating application of brakes] are found.

(iv) Shoe sole shows a deep impression of accelerator pedal, as if shoes were hardly pressed on it.

(v) No alcohol and drugs are found in the body, ruling out drunken driving.

(3) Motive - for masquerading a suicide as an accident is to help relatives claim full insurance amount; for suicide claim is not given.

c. Homicidal

Premeditated murder of a pedestrian with an automobile. Driver hopes to avoid the blame altogether if he is able to successfully drive away, or at least get away with a lesser charge **u/s 304A**.

2. Accident faked to conceal crime

(1) Person killed by other means"body placed in vehicle"vehicle pushed off a cliff, or set on fire to make it look like an accident

(2) Investigation:

(i) Careful evaluation of all injuries

(ii) If vehicle caught fire during fall, burns should be antemortem and there should be soot in fine branches of bronchi

(iii) If body shows signs of putrefaction, the time of which is inconsistent with that of accident, it may indicate an already dead body being kept inside vehicle.

3. Miscellaneous

(1) An already dead body lying on a street may be run over by a vehicle, especially if the street was dark. All run over injuries would be PM in nature. Some other obvious cause of death would be present eg a stab wound etc.

(2) Primary skin tuberculosis has been reported after vehicular accident. Also seen after incised wounds [**ch 12**].

19. Asphyxia

I. INTRODUCTION

A. Definitions

(1) ***Asphyxia*** (Gk a, not; sphyzein, to throb) is exclusion of air from lungs. Literal meaning is without throbbing or pulse, which occurs soon after asphyxia. (For Gordon's classification, please see ch 8).

(2) **Hypoxia** is deprivation of adequate oxygen supply at tissue level that results from asphyxia.

(3) **Anoxia** is complete deprivation of adequate oxygen supply at tissue level. Normal oxygen saturation of arterial blood is around **95 mm Hg** (12.7 kPa). Persons > 60 years of age may have a somewhat lower saturation – around **80 mm Hg**. **Mild hypoxia"60 mm Hg**. **Severe hypoxia"40 mm Hg**. **Fatal hypoxia"20 mm Hg** (1/5 of normal).

B. Classification of Asphyxial Deaths

1. According to mechanism of production

(1) **Mechanical** (a) When exclusion of air from lungs is by ligature around the neck, and: (i) Constriction force of ligature is weight of the body " **Hanging** (ii) Constriction force of ligature is anything other than the weight of the body " **Strangulation** (b) When exclusion of air from lungs is by any means other than ligature around the neck " **Suffocation** (i) External orifices of respiration (mouth, nose) are blocked " **Smothering** (ii) Foreign object is pushed through mouth up to posterior pharyngeal wall " **Gagging** (iii) Foreign object enters trachea " **Choking** (iv) Mechanical obstruction by interference with respiratory movements " Crucifixion, Hog tying, Pickwickian syndrome, **Traumatic asphyxia, Positional** [syn,**postural] asphyxia, wedging** (c) When exclusion of air from lungs is by mechanical entry of liquid into lungs "

Drowning. For sub classification of each entity, pl refer to the appropriate section. Only **mechanical asphyxias** are the subject matter of current chapter.

Other causes of asphyxial deaths are:

(2) **Pathological** -

(i) Acute poliomyelitis [paralysis of respiratory muscles]

(ii) Bronchitis

(iii) Edema of glottis [eg hypersensitivity reaction due to drugs (penicillin)]

(iv) Laryngeal spasm

(v) Tumors and abscess around neck.

(3) **Toxic** -

(i) Hb binding of oxygen lowered [CO]

(ii) Tissues unable to utilize oxygen [CN]

(iii) Respiratory center paralyzed [barbiturates, opium, strychnine]

(iv) Muscles of respiration paralyzed [Gelsemium]

(4) **Environmental** -

(i) High altitude

(ii) insufficient oxygen in environment [eg enclosed places, trapped inside a box, trunk, refrigerator etc]

(iii) irrespirable gases in the environment [CO, CO_2, Sewer gas, Chlorine etc]

(5) **Traumatic** -

(i) Bilateral pneumothorax

(ii) Pulmonary embolism [blood clot, amniotic fluid, fat, air etc]

(6) **Iatrogenic** - Anesthetic deaths

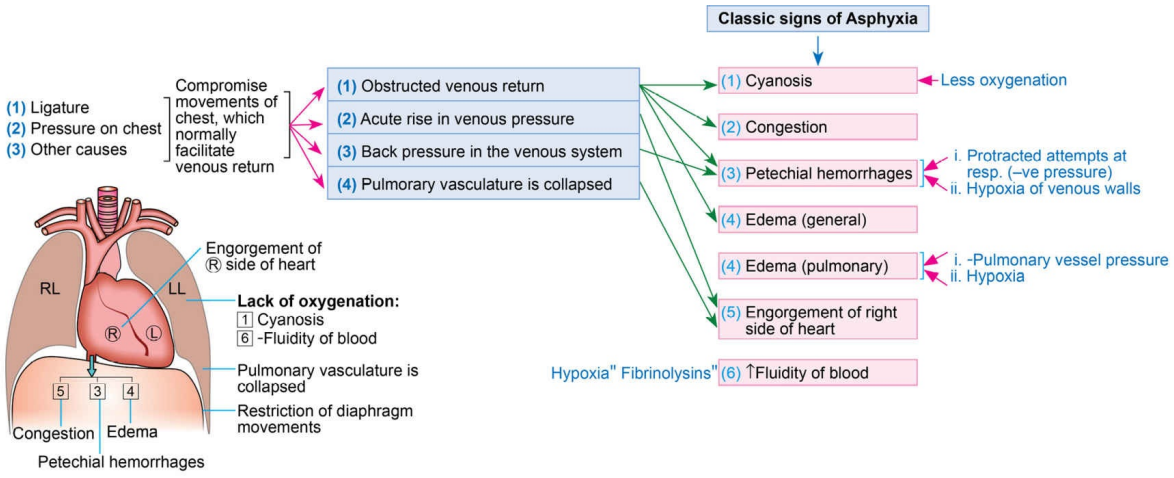


Fig. 19.1: Mechanism of all six "classic" signs of asphyxia

2. According to time of survival

(1) **Rapid asphyxia** [survival <15 min]1 – Hanging, throttling, choking

(2) **Slow or protracted asphyxia** [survival >30 min] – CO intoxication, opiates, initial resuscitations followed by death.

II. CLASSICAL SIGNS OF ASPHYXIA

[A] Classical signs are asphyxia are (1) Cyanosis [**Fig 19.2**] (2) Congestion of internal organs (3) Petechial hemorrhages (4) Edema (5) Engorgement of the right side of the heart (6) -ed fluidity of the blood [see square boxes in **Fig 19.1**].

Memory Aid 1: Classical signs of asphyxia

CCTV FoR Everyone

Congestion

Cyanosis

Tardieu's spots [petechial hgs]

Fluidity of blood

Rt side heart engorged

Edema

Fig. 19.2: Blue nails in a case of asphyxia

[B] **Mechanism** - Application of ligature, pressure of chest or restriction of respiration by other means generally results in compromise of the movements of the chest, which normally facilitates venous return.

This causes (1) obstruction of venous return (2) Acute -in venous pressure and (3) back pressure in the venous system. Since lungs cannot expand fully it also results in (4) collapse of pulmonary vasculature. Normal expansion of lungs is also responsible for “sucking in” of blood into the lungs from the right heart. Since this mechanism also comes to an almost halt, pooling occurs in the right heart. These mechanisms together produce classic signs of asphyxia **(Fig 19.1)**. Hypoxia that results from lack of oxygenation is supposed to release fibrinolysins which are supposed in turn to -the fluidity of blood. These ***“classic signs”* of asphyxia** are no more considered pathognomonic of asphyxia. These are very general signs seen in almost any autopsy. In fact they have been called as the **‘obsolete diagnostic quintet**’ [considering congestion and edema together]. The explanations given above are however generally advanced for these signs as seen in asphyxia.

[C] **Petechial hemorrhages:** (1) Size: (i) Small pin-point collections [***0.1-2.0 mm***] of blood lying in the skin, the sclera, conjunctivae, skin of eyelids, forehead, upper cheeks, lining of the mouth and throat, muscles of the temples, and under thoracic serous membranes such as the pleura or pericardium. (ii) Bleeding spots ***2.0 mm*** are called ***ecchymoses*** [for definitions of **hemorrhage** and **hematoma**, which are still larger bleedings please see **ch 17**, under the heading intracranial hgs]. (2) Petechiae due to asphyxia - known as **Tardieu’s spots** [syn Tardieu’s ecchymoses; Tardieu Flecken]. First described by the French pathologist **Ambroise Tardieu** in **1866**. (3) Mech of asphyxial petechiae - -in venous pressure above the noose"rupture of capillaries and small venules. Typically they are pinpoint to pinhead size. Larger petechiae are infrequent and when they occur, result from confluence. (4) Density of petechiae: (i) Serves as an indicator for the duration of process. -duration "-petechiae (ii) Sometimes they may be so diffuse [and minute] that the head and neck may present a dusky appearance, which may be mistaken for congestion, especially when they are in s/c tissues. (5) Petechiae in other deaths – seen also in (i) plethoric and obese individuals dying of natural deaths (ii) Acute right ht failure with markedly -ed venous pressure. (iii) Cardiopulmonary resuscitation. These petechiae are

usually larger than the asphyxial petechiae. [D] **Engorgement of the right side of the heart** - has been found in many causes other than asphyxia. (1) **Mechanism** - Has been claimed to be due to (i) greater distensibility of the right heart, (ii) the influence of violent respiratory movements and muscular spasms [not convincing because administration of curare still causes fullness of right heart] (iii) an alleged strong contraction of the pulmonary arterioles opposing the discharge of blood from the right ventricle, (iv) diminished suction power of the left ventricle and (v) influence of rigor mortis on the left ventricle after death, though no explanation has been offered as to why the right ventricle should behave differently to the left in this respect.

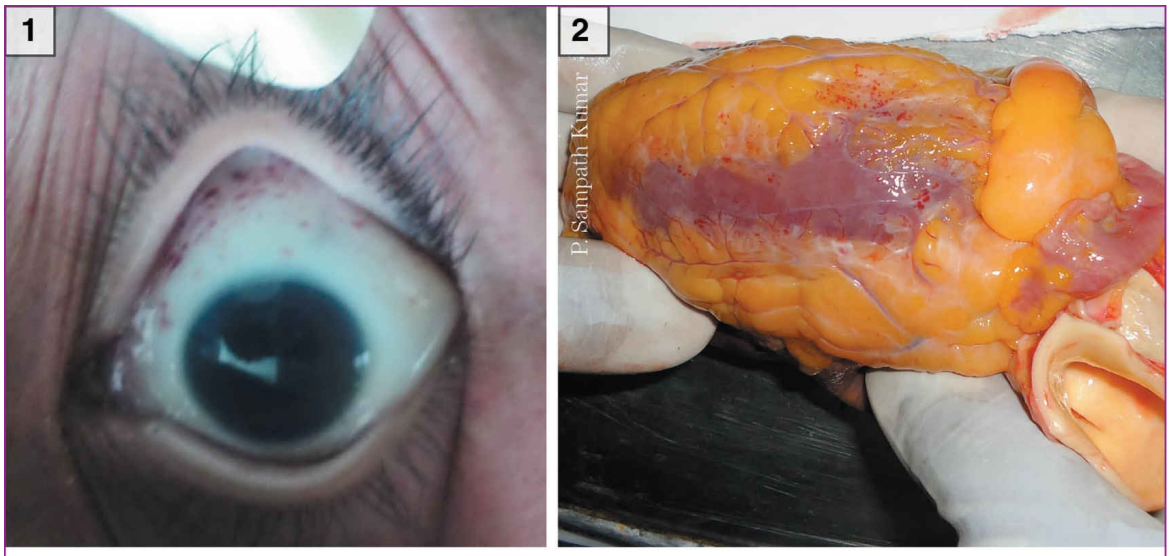


Fig. 19.3: **(1)** Tardieu's spots in the eye and **(2)** over visceral pericardium in cases of asphyxia.

III. HANGING

Hanging is that form of death which is caused (i) either by exclusion of air from lungs or oxygenated blood from the brain (ii) by means of a ligature round the neck, (iii) the constricting force being the weight of the body. When a person survives hanging incident, it is called **near hanging**.

A. Classification

Hanging can be classified according to (1) position of knot (2) position of feet (3) manner of hanging.

1. According to position of knot

(1) **Typical hanging** – if knot is at occiput

(2) **Atypical hanging** – if knot is at any other position.

2. According to position of feet

(1) **Complete hanging** – if feet do not touch ground

(2) **Incomplete** or **partial hanging** – if feet or other parts of the body touch ground (for details please see "ML aspects of hanging").

3. According to manner of hanging

(i) Suicidal (ii) Homicidal [eg lynching] (iii) accidental [eg autoerotic asphyxia] (iv) Judicial.

B. Cause of Death

It is not necessary for ligature to completely encircle the neck in order to cause death. It may be applied only to the anterior part or sides of neck. Happens generally with "stiff" and "rigid" ligatures, eg steering wheels of cars, transverse ladder rungs, during accidental hanging [More examples given below under the heading "accidental hanging"].

1. Asphyxia

(1) **Mechanism** - Trachea is occluded"No air supply to lungs"No oxygenation"Death.

(2) Earliest theory. This mechanism was suggested in **1870** by **Tardieu** [please see above]. He thought that hanging was not associated with cerebral ischemia because intimal tears of the carotid and pale brain were not commonly seen at autopsy.

2. Occlusion of neck blood vessels

(1) **Mechanism** - Neck b.v. are occluded"Blood supply to the brain is stopped"cerebral ischemia [or venous congestion] occurs"Death.

(i) Cerebral ischemia"occurs when both arteries and veins are occluded

(ii) Venous congestion"occurs when only veins are occluded, but arteries are patent. Blood keeps flowing to the brain, but cannot return

(2) **Evidence in favor of occlusion of neck b.v.** – **(a)** In **1880**, **Hoffman** tried to infuse the neck vessels of hanging victims, including those of children [while

they were still hanging], but could not do so. This led him to propose that **occlusion of the neck vessels** occurs during the hanging process [in addition to asphyxia] **(b)** In **1895**, **Reineboth** performed tracheostomy in rabbits and hanged them in two groups. In the first group noose was placed **above the tracheostomy opening** and in the second group, **below**. If asphyxia was the real mechanism of death, rabbits in the first group would not die. But they still died although slower [15-20 minutes vs. 5-6 min]. **(c)** In **1897**, **Brouardel** of France observed the retinas of hanged animals by ophthalmoscopy, and discovered they were devoid of blood. Indicated cerebral ischemia. **(d)** A number of individuals have hanged themselves with the noose above the larynx and a permanent tracheostomy opening below.

3. Reflex vagal inhibition of the heart

Mechanism - Pressure over carotid arteries"Carotid body stimulated"Vagal stimulation"Cardiac inhibition"Death.

4. Fracture dislocation of cervical vertebra

Occurs mainly in hangings associated with a long drop (as in judicial hangings).

5. Current thought

Consensus of opinion is that

(1) ***asphyxia*** is the main cause of death in ***complete hanging***, while

(2) ***occlusion of neck b.v***. is the main cause in ***incomplete hanging***.

(3) Very few authors believe in reflex vagal inhibition theory.

(4) Earlier belief that commonest cause is ***combined asphyxia and venous congestion*** is no more considered correct.

C. Fatal Period

(1) **Suicidal hanging** – If death occurs due to asphyxia, fatal period is 5-10 min; if death is due to occlusion of neck b.v. death takes longer - 12-20 mins.

(2) **Judicial hanging** [i.e. with a long drop]- instantaneous

(3) **Delayed deaths** – Few days. If the victim is saved initially from hanging, he may succumb hours, days or weeks later to (a) aspiration pneumonia (b) edematous or hemorrhagic swelling – of pharyngeal tissues or aryepiglottic folds (c) emphysema – massive s/c and mediastinal emphysema from tracheal and laryngeal lacerations (d) infections (e) edema of larynx and lungs (f) brain pathology [abscess, hypoxic encephalopathy, infarction and softening]. **Mechanism** – In hypoxic injury, higher cortical centers suffer first [being

most sensitive to O_2]"unconsciousness"hypostatic pneumonia"further anoxia"injury to basal ganglia, and finally of respiratory and other vital centers"death.

D. PM Appearances

1. External

a. Description of ligature

Ligature material if present around the neck should always be described. Note especially

(1) **Nature of ligature** – eg bed sheet, belt, dhotie, leather strap, metallic chain, nada, rope, sacred thread, saree, scarf, turban and wires (plain metal wires, electrical wires etc). When a narrow wire is used, it may "eat into" the flesh of the neck ["cheese-cutter" method]. A cheese cutter is a kitchen utensil used to cut a large solid block of cheese into slices, using a thin metal wire. In jails and prisons, convicts typically tear sheets into strips, or they may use whatever is handy eg T-shirts, undershorts, trousers, or even socks.

(2) **Its color and composition** [eg silk, cotton, nylon etc]. May help co-relation if similar cloth is found in possession of someone.

(3) **Pattern and texture of ligature** – Does the ligature show any particular pattern [eg weaving patterns in ropes]? Pattern and texture of ligature is often produced upon the skin in the form of a patterned abrasion.

(4) **Intact** – Whether ligature is intact or has been cut off.

(5) **Cut ends** – if cut, whether appears torn or cut sharply by scissors.

(6) **Extraneous material on ligature** – blood, fragments of epidermis, hair, fibres, saliva, vomit, any other suspicious substance.

(7) **Length** – Is the length of ligature sufficient to hang from the point from where victim's body was found hanging.

(8) **Width** – (a) does it correspond with the ligature mark (b) Narrow ligature tends to produce deeper groove, because wt of body is distributed over narrower area. Broad ligatures produce narrower grooves.

(9) **Multiplicity** – (a) Does the ligature show multiple turns round the neck(b) If there are two turns, it may produce a double mark round the neck – one circular, the other oblique(c) Skin may get caught between multiple turns, and show multiple rings of congestion.

(10) **Strength** – is the ligature strong enough to support the weight and jerk of the body.

(11) **Circumference** – Circumference of the ligature. Compared with the **circumference of the neck**, it can be decided if the neck was horizontal or oblique.

(12) **Tightness** – Note how tight the ligature is wrapped. (a) Tighter ligatures produce deeper grooves all round the neck. Impression from knot may also be found (b) Slip (running) knot does not produce uniform pressure all round; it is maximum just opposite the knot and then tapers off gradually at the sides. (c) If the point of suspension is in the occipital region, front of neck would show the deepest groove (with neck tilted forwards); if below the chin, the deepest groove would be at the back (with neck tilted backwards). (d) Knot and small adjacent lengths of ligature may be stretched away from the body, and may not even be in contact with the skin. Ligature mark will be absent in this area and impression from knot not found.

(13) **Padding** – a piece of towel may sometimes be found between the skin and ligature [placed by victim in order to reduce pain]. Found usually in accidental hanging, especially sexual asphyxias [please see below].

(14) **Spontaneous breakage** - Sometimes the ligature may break spontaneously and body may be found lying on the ground. In such cases, it becomes imperative to determine if the death was due to hanging or strangulation. It can be done by (a) examining the other end of ligature which must be found tied to the suspension point (eg fan, tree etc)(b) Broken ends of ligature must coincide.

(15) **Knot** – (a) Record the location and type of knot. (b) **Location** – whether at occiput, below the chin, below either ear (subaural) or any other specific location. In all cases, it is always above the rest of the ligature. This produces an inverted ‘V’ shaped ligature mark, the apex of ‘V’ corresponding with the site of the knot. (c) **Type** - It may be fixed or running (**Fig 19.4**). **Granny** [syn, **lubber’s**] and **reef** [syn, **f lat**, **square**] knots are rather common. But some other special types of knots are exceedingly rare and may sometimes give away the profession of the person. Even simple knots can reveal predilection or hobby of the person, eg a **sailor** or a **scout** may use a **reef knot** rather than a **granny knot**. (d) **Removal of ligature** – Knot should never be opened. (i) Photography – First step is to take photographs [at close range]

before removal of ligature (ii) The ligature should now be cut opposite the knot, and the two cut ends secured with a string (**Fig 19.5**) [for more on ***Forensic knot analysis***, please see **chapter 30** – Forensic Science Laboratory].

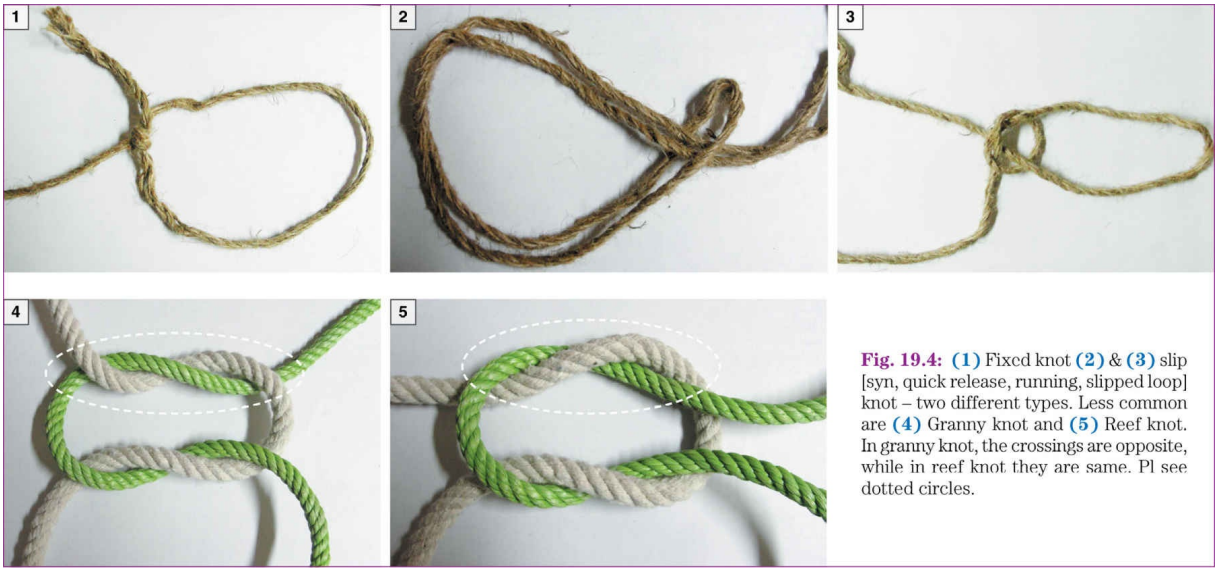


Fig. 19.4: **(1)** Fixed knot **(2)** & **(3)** slip [syn, quick release, running, slipped loop] knot – two different types. Less common are **(4)** Granny knot and **(5)** Reef knot. In granny knot, the crossings are opposite, while in reef knot they are same. Pl see dotted circles.

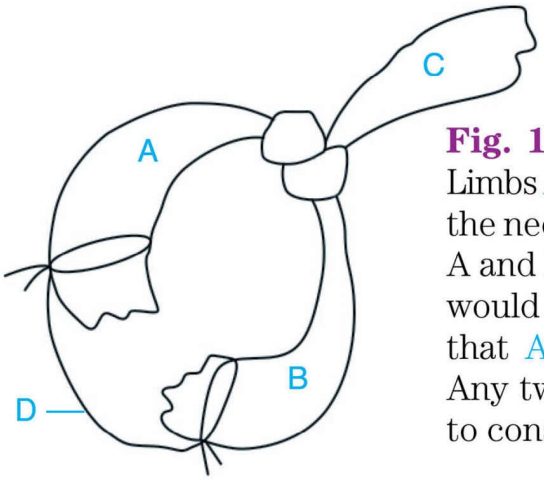


Fig. 19.5: Method of securing the knot. Limbs A and B form the actual noose around the neck. C is the free end of the ligature. If A and B are not secured with a string D, it would be difficult to demonstrate in court that A and B indeed formed the noose. Any two of the three limbs would appear to consist of original noose.

b. Ligature mark

Ligature mark is a type of pressure abrasion due to continued pressure by ligature on the neck.

Salient features:

(1) It is seen both in hanging and strangulation [**Table 3**]. Strangulation mark is described later.

(2) **Appearance** - It is usually seen as a furrow or groove in the tissue which is soft and pale initially, but as the skin dries up, becomes hard (parchment like) and dark brown. It runs from midpoint of the neck upwards, outwards and backwards [from either side of the neck] to reach behind the neck where it is deficient [**Fig 19.6**]

Fig. 19.6: Classical oblique ligature mark in hanging.

(3) It is the ***most important*** and ***specific*** sign of death from hanging.

(4) Following things must be noted (i) **Its position** - Usually it is at the front of neck, but rarely at the back of neck also. If the head has been hanging on one side, it may be one-sided [**Fig 19.7**].

Fig. 19.7: One sided ligature mark. It indicates the head had been hanging on one side.

(ii) **If in front its position with respect to thyroid cartilage** – Ligature mark is situated (a) above the thyroid cartilage in 80% cases (b) at the level of thyroid in 15% cases and (c) below the level of thyroid in only 5% cases. Likelihood of ligature being below the level of thyroid is more in partial suspension. (iii) **its direction**, whether continuous or interrupted (eg if a piece of cloth such as shirt collar is interposed between it and the skin), depth and width, areas of neck involved and its relation to local landmarks (iv) **Compare with** (a) weight of body – Heavier the body, deeper the ligature mark (b) Degree of suspension – Complete suspension produces deeper mark; partial suspension, much shallower (c) Time of suspension – Longer the suspension, deeper the groove. With long suspension times, even lesser weight of body and wider ligatures can produce deeper grooves. Sometimes a thin line of congestion or hemorrhage is seen along the edges of the groove. If present, it is strongly suggestive of antemortem hanging (d) ligature material – if ligature mark is showing a pattern (patterned

abrasion), does it match with that of the ligature? Ligature pattern is better appreciated by using a magnifying lens and examining under oblique lighting. (v) **Effect of slippage of ligature** - (a) As the victim ties ligature round his neck, it may produce a very faint and superficial impression (b) when victim suspends himself, the ligature moves upwards, and produces a second deeper impression above the initial impression. (c) If the ligature material is rough (eg rope), the two ligature marks may be joined by fine abrasions representing the movement of ligature upwards (vi) **Fibres** – Although the ligature mark may look clean, there always are small fibres sticking to the ligature mark. Strip them off with a sticking tape [**Fig 19.6**], secure it on a glass slide and see under microscope. Fibres must match with that of the ligature found at the scene. **Case study** - In one case of homicidal strangulation, with missing ligature, the author could tell the color of the ligature (red) by lifting the fibres from the ligature mark by a cellotape and observing them under microscope. Nothing was visible on the ligature mark with the naked eye, and if cellotape method had not been used, the case may have remained unsolved. (vii) **Reexamination after some hours** – If body is fresh, ligature mark may not be very evident. After drying the mark becomes more prominent (viii) **Pattern of ligature mark** – If the ligature mark is in the form of a patterned abrasion, photograph and describe the pattern. If pattern is not clear in ordinary light, examine in oblique light or under UV light. Compare with the pattern of the ligature recovered, and see if they match. (ix) **Absent ligature mark** – If the ligature mark is absent, it does not exclude hanging. Causes of absent [or indistinct] ligature mark - (a) Soft ligature material eg scarf, towel etc (b) Beard, or a piece of clothing getting caught between ligature and skin (c) Decomposition – may obliterate ligature mark (d) when pressure is applied only on the front of the neck, eg by arm of a chair or rung of a bladder (x) **Ligature mark present in the absence of hanging** – (a) If soon after death, ligature is passed around neck and body is dragged along ground with its help, a ligature mark very similar to that of hanging may be produced (b) in infants and obese persons, skin folds on neck may resemble a ligature mark, especially if s/c fat has been coagulated by refrigeration (c) Jewellery or clothing around neck [eg shirt collar]"Neck tissues swell due to decomposition"a false mark resembling that of hanging [or strangulation] may be produced. Please also see pseudostrangulation below. (xi) **Histological examination** – of the ligature mark is a must. Presence of vital reaction indicates antemortem hanging. (xii) **Suspension peak** – The junction of the noose and the vertical part of the rope

[point **A** in **Fig 19.8**] is pulled upwards and away from the skin due to weight of the body. Thus no ligature mark is left at the back. A triangle ABC is formed the apex of which (A) is called the suspension peak or point. This suspension peak is the distinguishing feature of hanging from strangulation, as weight of the body does not act on the ligature in strangulation. Such a suspension peak is typically absent when the knot is in front because of projecting chin, on which knot often leaves its mark.

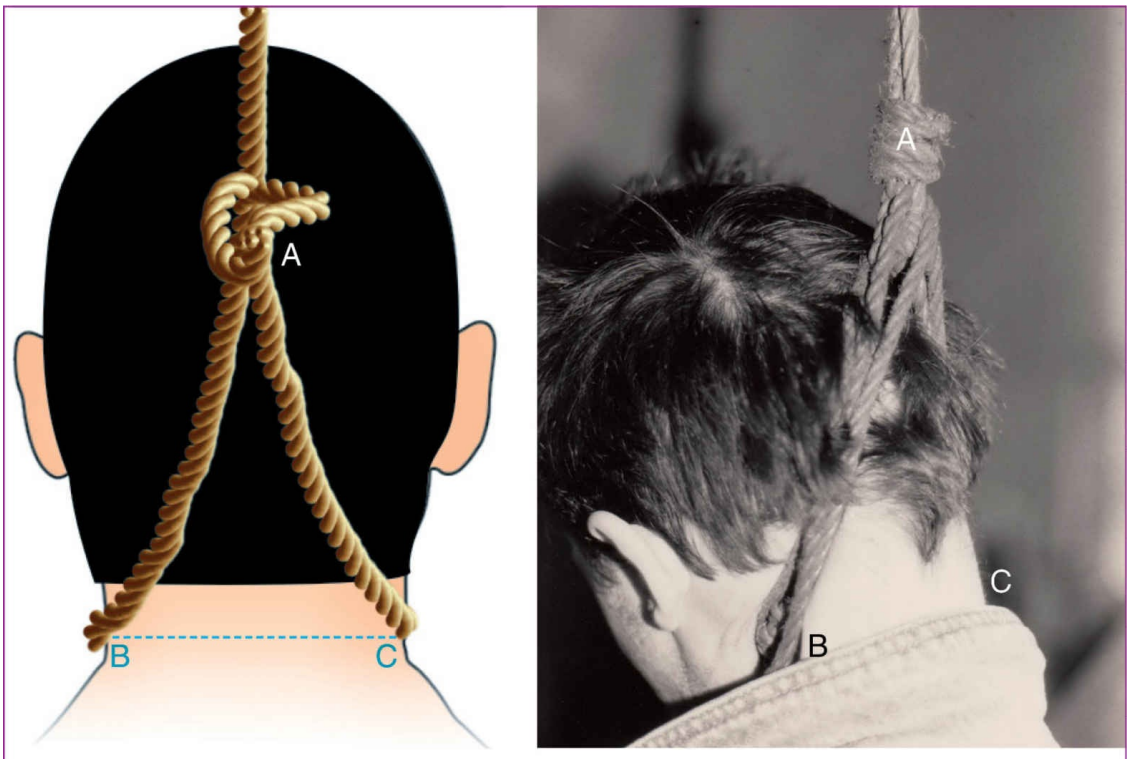


Fig. 19.8: A: Knot; BC: Area where ligature mark in hanging is deficient

c. Other Signs

(1) **PM Staining** – on legs, feet, soles, hands, forearms [**glove-stocking** like distribution], undersurface of breasts, penis [most dependent parts of the body]. Blood may extravasate in these areas in the form of petechial hgs. Upper part pale. If body is unsuspended before fixation, and made to lie down in a supine position, it disappears from these areas and reappears in new dependent parts.

(2) **Head** – inclined to the side opposite to that of knot

(3) **Neck** – elongated and stretched

(4) **Face:**

(i) Usually pale [occlusion of carotids and vertebral arteries]

(ii) rarely congested [occlusion of jugular veins and vertebral venous plexus but not of vertebral arteries]

(iii) may be swollen [same reason as above causes leakage of edema fluid into the tissues]. If body is cut down, swelling may disappear

(iv) Eyes – frequently protruded and firmer than usual due to congestion

(v) Conjunctiva - congested

(vi) ***Le facies sympathiques*** – If ligature knot presses on cervical sympathetic, the eye on the same side is open and its pupil dilated; the other eye is closed and its pupil constricted. It was once believed to be a hallmark of antemortem hanging. Believed to result as a result of unequal tension on the neck structures. No more considered a useful sign.

Fig. 19.9: Le facies sympathiques.

(5) **Petechial hgs:**

(i) In skin and conjunctivae [Overall incidence >50% of cases].

(ii) In typical hanging – No petechiae; face pale [because of complete occlusion of blood to and from the brain]

(iii) When knot is under chin – Lower jaw protects neck from deep pressure by the noose"Arterial flow continues; venous flow stops"Petechiae abundant; congestion prominent

(iv) When knot is at one side, i.e. in front of one ear"subconjunctival hgs are one sided

(6) **Nostrils** – bloody froth [due to rupture of engorged bv]

(7) **Tongue:**

(i) protruding; caught between the teeth. Protruded part is dark brown, even black due to drying

(ii) swollen and blue, especially at the base

(8) **Lips and mucus membranes of the mouth** - blue

(9) **Saliva:**

(i) dribbling from the mouth; may be blood stained [rupture of engorged bv]

(ii) surest sign of antemortem hanging

(iii) Cause - (a) Stimulation of pterygopalatine ganglion (b) direct stimulation of salivary glands by the ligature

(10) **Hands** – Clenched, especially in violent hangings

(11) **Penis** –

(i) engorged with blood [due to hypostasis]

(ii) may be semierect

(iii) Semen may be found at the tip

(12) **Relaxation of sphincters** – causes escape of urine and feces.

Fig. 19.10: Protrusion of tongue and its dark coloration in a case of hanging

2. Internal

(1) **Neck** – Should be examined after removal of brain and viscera from the chest and abdomen. This produces a bloodless field which prevents any artifactual hemorrhages.

(2) **Ligature mark:**

(i) Tissues underneath the mark are dry, white and glistening.

(ii) There may be scattered hemorrhages caused by direct trauma.

(iii) Adjacent muscles may show occasional ecchymoses.

(3) **Strap muscles** – may be torn and may show hemorrhages.

(4) **Carotid arteries:**

(i) intima shows several horizontal splits with extravasation of blood in their wall due to crushing and stretching [**Amussat's sign**], much like ladder tears in aorta seen in vehicular accidents [**ch 18**]. First described in **1843** by **Amussat**.

(ii) More common in long drops as in judicial hanging

(iii) Rarely tears may be longitudinal.

(iv) May be combined with tears of media or adventitia.

(v) Sometimes dissection of the medial layer occurs with subsequent narrowing of its lumen.

(vi) In patients who survive, such tears may lead to formation of thrombi and subsequent cerebral ischemia and infarction.

(vii) Other conditions in which similar tears may be found are (a) blunt neck trauma, (b) extreme overstretching (c) whiplash-injuries.

(5) **Vertebral arteries** – may show similar damage as in carotids with an even higher frequency.

(6) **Epiglottis** and **larynx** – may show petechial hemorrhages

(7) **Trachea** –

(i) Congested

(ii) May be injured rarely

(iii) May show petechial hemorrhages

(8) **Lungs:**

(i) Congested, edematous and exude bloody serum on cut section.

(ii) May be pale sometimes. It has been asserted that lungs are congested if constriction of lungs occurred at end of expiration and pale if it occurred at end of inspiration. However there is no evidence for this assertion. Moreover constriction of lung does not occur in an instant; hanging is a process which continues for several minutes.

(iii) Subpleural ecchymoses

(9) **Abdominal organs** – congested

(10) **Brain:**

(i) may be normal, congested or pale.

(ii) May show subarachnoid hemorrhages

(11) **Middle ears** - hemorrhage

(12) **Hyoid:**

(i) May show fractures. They are anteroposterior compression fractures.

(ii) incidence – varies from 0-60%; average 15-20%.

(iii) Age – rare <30 y, because apices of greater cornu are cartilaginous before 30 y [**ch 3**].

(iv) Site – junction of inner 2/3rd and outer 1/3rd. Fractures of hyoid are also found in ch alcoholics [**battered alcoholic syndrome - ch 40**]. Should be kept in mind when doing an autopsy on an alcoholic.

(v) **Demonstration of fractures** - please see below under the heading "hyoid bone fractures".

(13) **Thyroid:**

(i) Superior horns may show fracture from pressure on thyrohyoid ligament. More common >40 y.

(14) **Vertebral column:**

(i) Hemorrhages - beneath the area of the anterior longitudinal ligament of the intervertebral disks in the lower thoracic and lumbar vertebrae [**Simon sign**, **Simon bleedings**, **Simon hemorrhages**].

(ii) Appearance - look like dark red to violet horizontal lines between vertebral bodies [**Fig 19.11**]; they are limited only to the area of the anterior ligament of intervertebral disks and do not penetrate into the vertebral bodies.

(iii) Incidence - more frequent in (a) younger individuals (b) in complete hanging and (c) in individuals with minimal degenerative changes in the lumbosacral part of the spinal column.

(iv) Cause - overstretching of the spine because of free suspension of the body. Causes stretching and tearing of small vessels within the disks. First described in **1968** by the German forensic pathologist **Axel Simon** [born 1931].

(v) other cases in which similar hemorrhages are seen – Vehicular accidents, falls from heights, drowning, hypothermia. Extremely rarely in natural deaths.

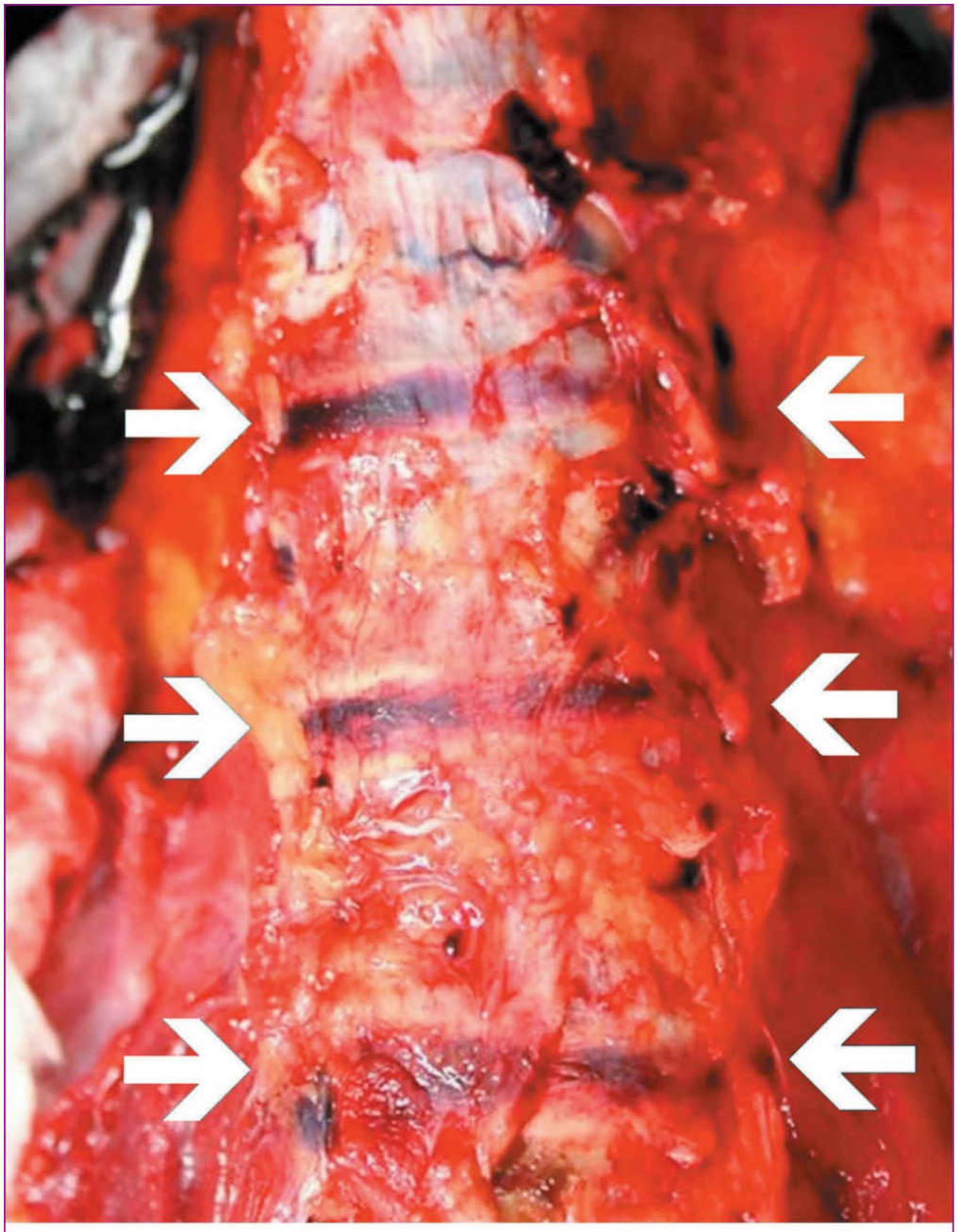


Fig. 19.11: Simon sign seen in hanging. Pl see text for details.

E. Medicolegal Aspects of Hanging

1. Pressures required to obliterate neck structures

These are (i) **Jugular veins**"2 kg (ii) **Carotid arteries**"5 kg (iii) **Trachea**"15 kg

(iv) **Vertebral arteries**"30 kg (v) **Vertebral venous plexus**"undetermined, but between 5 and 30 kg. [A] **Pale face -** If force applied to neck is > 30kg as happens in complete hanging (when entire wt of body acts on neck), blood can neither go towards head nor return from there. The result is a pale face. [B] **Congested face -** If force applied to neck is between 2 kg and 30 kg (as happens in strangulation), blood is able to go to face, but can not return, resulting in intense congestion of face and bleeding from mouth, nostrils and ear (due to rupture of small capillaries). The same is true of partial hanging, when only part of the wt of body acts on the neck.

2. Partial hanging

Partial hanging is that specialized form of hanging in which some part of the body is supported either by ground, or some object other than the neck [**Fig 19.12**].

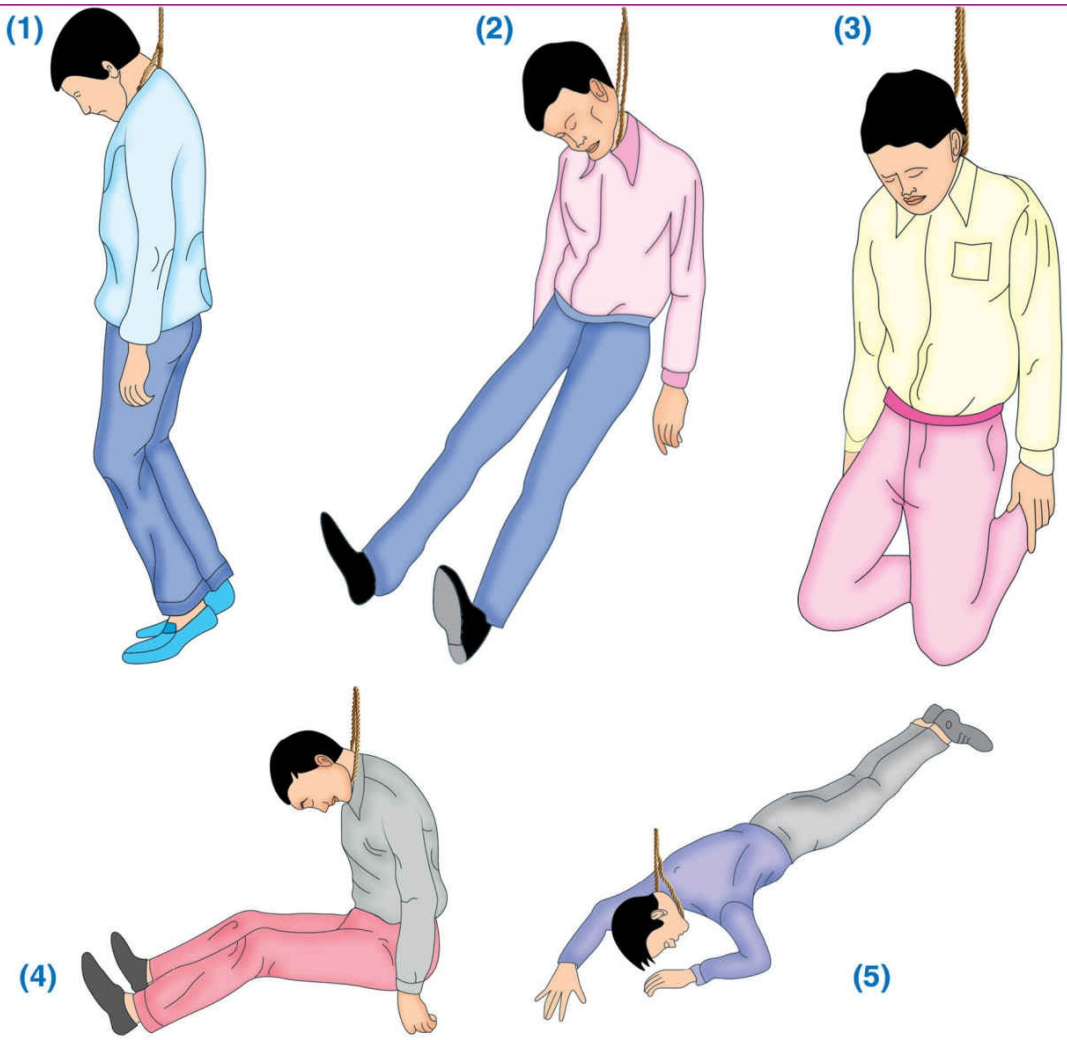


Fig. 19.12: Varieties of partial hanging **(1)** Standing **(2)** Reclining **(3)** Kneeling **(4)** Sitting and **(5)** Lying

Salient features:

(1) **Point of suspension** – in partial hanging is lower down eg (i) bedpost (ii) chair (iii) door knob (iv) leg of table (v) railing of staircase

(2) **Position of deceased** – Deceased may be in (i) sitting (ii) kneeling or even (iii) completely lying down position

(3) **Distribution of the weight of the body** – Weight of the body is distributed on the ligature as well as on all points of body touching the ground. Thus constricting force is less and congestive changes more marked.

Fig. 19.13: Partial hanging in sitting position.

3. Manner of hanging

Suicidal hanging is commonest. Accidental hanging is less common and homicidal hanging still less common.

a. Accidental

(1) **Children:**

(i) climbing railings, trees, walls etc – falling from a high branch etc"clothing or garment [chunni, muffler etc] gets entangled in a branch, nail or other projecting object from wall"drawn tight round neck

(ii) during play - especially when imitating judicial hanging

(2) **Infants** – in cots with restraining vertical bars around it"try to crawl out, move away or wriggle out of cot"fall out between the vertical bars"neck gets caught between two vertical bars

(3) Sexual hanging [please see below]

(4) **Stuntmen** – exhibiting hanging as a stunt

(5) **Suspension of chin against**

(i) edge of a sofa or arm of chair

(ii) Staircase – victim slips"neck gets caught in one of the vertical bars on the sides of staircase

(iii) Steering wheel – May happen during vehicular accidents [**ch 18**]

(iv) Tailboard of a lorry or cart.

(6) **Workmen, masonry men etc** – falling from scaffolding may get entangled in ropes. Rarely while falling from ladder, neck may entangle against one of its transverse rungs.

i. Sexual hanging

Sexual hanging [syn, Asphyxiaphilia, Asphyxiophilia, Autoerotic asphyxia, Hypoxyphilia, Kotzwarrism, Masochistic hanging, Sexual asphyxia] is accidental death caused by a self-induced decrease in oxygenation of blood (hypoxia) produced most commonly by partial hanging.

Salient features:

(1) **Reasons** - Oxygen deprivation of the brain is thought to lead to an enhancement of sexual orgasm for following reasons (i) .ed oxygenation and -ed CO_2 retention produces feelings of giddiness, lightheadedness, and exhilaration, which heighten sexual response. (ii) Induction of erotic hallucinations. (iii) partial asphyxia brings about -ed sympathetic arousal, which in turn heightens orgasmic pleasure.

(2) **Methods:** (i) Partial hanging [a variant of suspension bondage] – Most common method (a) Neck - is protected by a padding [usually a towel] between the neck and ligature (b) Ligature - is passed round the neck in the form of a running noose. Its free end is tied to a limb [wrist or ankles] or to a fixed object. (c) Weight of the body is used to control pressure (d) Associated paraphilias - (I) Masochism - [1] Subject ties up wrists and ankles together with a chain, padlock, rope, string or wire. Sometimes noose is found around waist and genitalia also. [2] May have blindfolded himself [3] Evidence of self-mutilation on previous occasions - burns, cuts, puncture wounds etc on the body [4] Clamps or pincers attached to genitalia or breasts (II) Transvestism - [1] Found dressed up as a female, with bra, panties and sari (**ch 25**)]. [2] Bra may have been padded up to simulate full breasts [3] Sometimes even sanitary pads, wigs and make up may be worn. (e) Sometimes found naked or partially naked, with penis exposed through pants"Indicates penis was being manipulated [**Fig 19.14**] (f) Erotic or pornographic literature, or female nude photographs may be on the floor or elsewhere within view (g) Evidence of recent emission of semen (h) There may be mirror in front to watch the activities (i) A video camera may be there to make a photographic

record (j) Mechanism of accidental death - If the noose touches carotid sinus, consciousness is suddenly lost **(Fig 8.5)** and the victim is incompletely suspended with legs on the ground. He may be found suspended close to an object such as a chair or stool, that would have allowed him to sit upon and release the constriction, after achievement of orgasm. (k) Evidence of previous episodes on the neck - eg old scars (ii) Other methods involving asphyxia - (a) chest compression, (b) inhalation of chemicals such as amyl acetate, carbon tetrachloride, cocaine, chloroform,' ether, ethylene chloride, glue, ketamine, nitrous oxide, paint thinners, propane, trichloroethylene or a volatile nitrite [amyl nitrite]. A pad soaked with a sniffing chemical may be found nearby. (c) plastic bags, masks or impervious bags over the face." These are secured around the neck by an elastic band or a ligature to keep them in place. Interior of bag may be smeared with any of the above chemicals. (d) submerging under water (aqua eroticum),' (e) combination of several of these methods. (iii) Non asphyxial methods - Electrical stimulation. Electrodes are applied to the genitals or on abdominal wall usually with a low voltage supply from a battery or transformer

(3) **Why is it an accidental hanging** - Death is not intended by the victim, but it usually results accidentally when the "safety" mechanisms employed by the deceased fail or when there is an overdose of chemical inhalation.

(4) **Incidence** – Western countries: 1-2/million population/yr. Much less in India.

(5) **Sex** - male: female ratio > 50:1.

(6) **Scene of death:** (i) Follow all protocols of a scene of death investigation [**ch 1**]. Following specific points are important (ii) Evidence of abnormal sexual behavior, eg masochism, transvestism (iii) Evidence of previous occurrences of similar events - (a) grooves in the rafter or door from ropes (b) verbal communications with friends regarding nature of activities (c) evidence of surfing net with sites related to sexual hanging (d) diaries (iv) Evidence to protect neck - padding underneath ligature (v) No motive or other evidence of suicide.

(7) **Similar activities** - Choking game [Please see below under accidental strangulation].

(8) **MLI:** Differentiation from suicide – In insurance policies there is a clause stating that no money would be paid in cases of suicide. Differentiation is not always easy, especially when a strong reason for suicide appears to co-exist. In

some cases, family members are so embarrassed that they are reluctant or unwilling to provide enough data surrounding the circumstances in which the patient was found, and the cause of death may be mislabeled as suicide.

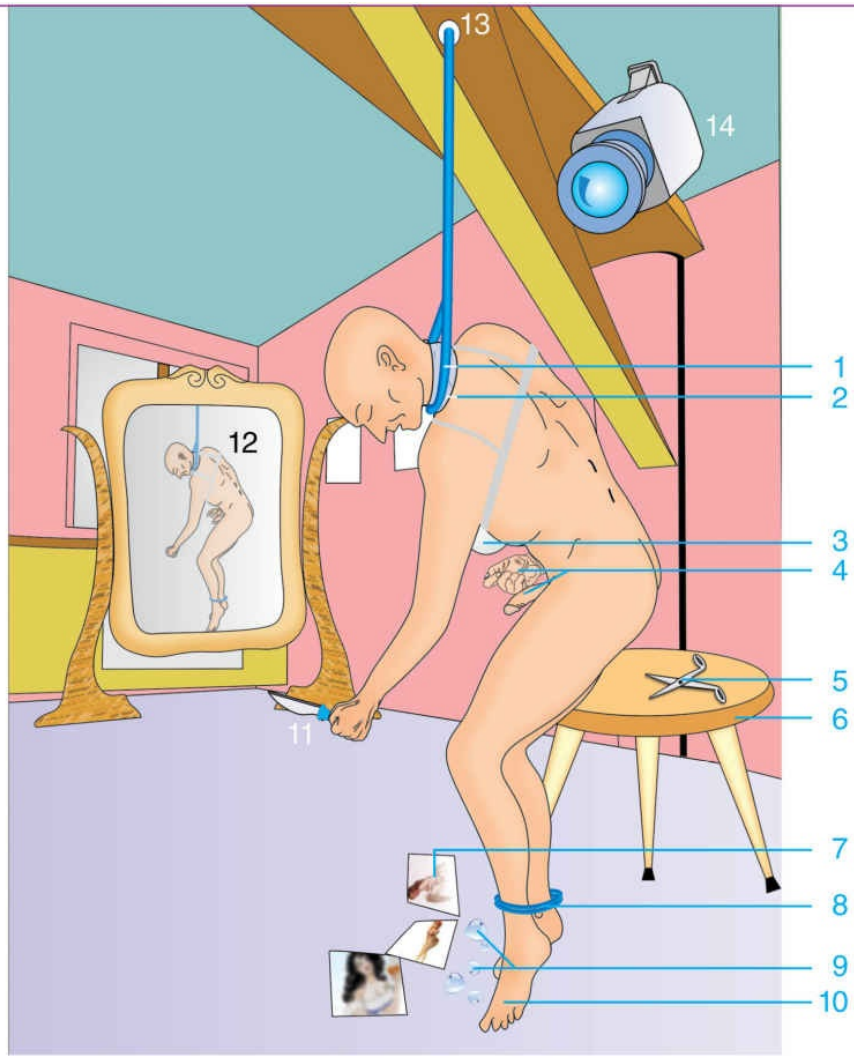


Fig. 19.14: Scene of a typical sexual hanging. (1) Ligature (2) Padding between neck and ligature (3) Bra (evidence of transvestism) (4) Evidence of self stimulation (5) Scissors to cut noose (6) Stool (7) Erotic literature (8) Tying up of legs (evidence of masochism) (9) Seminal stains (evidence of recent orgasm) (10) Weight on the legs used to control pressure on ligature (11) Knife for cutting self (evidence of masochism) (12) Mirror (13) Grooves in the rafter indicating similar previous episodes (14) Video camera for recording.

b. Homicidal

Incidence - Extremely rare, because it is difficult for a single person to overpower a man.

Lynching

Lynching is an extrajudicial execution carried out by a mob by hanging without a fair trial.

Salient features:

(1) It is a form of **homicidal hanging**, since hanging was done without a fair trial. A popular belief is that it was done when a black man raped a white woman. However only about 1/4th of lynch victims were accused of rape or attempted rape. Most blacks were lynched for outspokenness or other presumed offenses against whites, or in the aftermath of race riots. Lynchings took place most frequently in the Southern US from 1890 to the 1920s, with a peak in 1892.

(2) One of the common methods was to hang the suspect from a tree by a rope

(3) PM findings would be same as those in homicidal hanging; signs of struggle may be found.

c. Suicidal

Hanging is a common method of committing suicide.

4. Postmortem suspension

Postmortem suspension is when a person is first killed and then suspended to simulate suicide [simulated hanging]. Some books use the word "**postmortem hanging**" for this phenomenon. However since hanging signifies "death by asphyxia", this term should best be avoided.

Salient features:

(1) Ligature applied to the neck during **supravital period** will produce a ligature mark

(2) **Differentiating features of postmortem suspension from true suicidal hanging** [in contrast **Table 1** gives differences w.r.t. antemortem hanging which can be both suicidal and homicidal]. (i) Signs of dragging - to the place of suspension (ii) When a dead body is suspended, the rope is usually tied first around the neck and then around a beam, rafter or top of tree etc. then the rope is pulled up. Both the support [beam etc] as well as rope would show evidence of rope having dragged from below upwards. Fibres of rope would show abrasions consistent with rope having pulled up. In true suicidal hanging, such

abrasions would not be there. (iii) Beam paint - on rope (iv) Beam pain - rubbed off the beam where rope was pulled up (v) Hands of victim - show rope fibres in suicidal hanging, but not in pm suspension. May not be visible with naked eye, but can be seen under the microscope after tape lifting [apply adhesive tape to palm"lift it off"stick to a microscopic slide"observe under microscope] (vi) Internal signs - not consistent with those of hanging, but with the true cause of death [poisoning etc].

Table 1: Differences between antemortem hanging and postmortem suspension [sometimes called "postmortem hanging"]

S.No	Feature	Antemortem hanging	Postmortem suspension [simulated hanging]
1.	Ligature mark	Shows vital reaction. Tissues under ligature mark are dry, hard, condensed and parchment like	No such reaction. Tissues underneath show no appreciable change
2.	Salivary dribbling mark	Present	Absent
3.	Cyanosis	Seen, particularly in face, ears, lips etc	Not seen
4.	Tongue	Protruded	Not protruded
5.	Fecal and urinary expulsion	Yes	No
6.	PM Staining	On legs, soles, hands [glove-stocking like distribution]	If dead body hanged soon after death, distribution would be same as that in AM hanging. However if body was kept in a supine or prone position for >8 h and then hanged, PM staining would get fixed in respective positions, and would not show typical glove-stocking like distribution
7.	Struggle marks	No	Yes
8.	Evidence of injuries, defense wounds	No	Yes
9.	Cause of death	Hanging	Other than hanging [eg throttling, poisoning etc]
10.	Suicide note	Present	Absent
11.	Door closed from inside	Generally yes	Never
12.	Point of suspension	At such a height that victim could have reached it	May or may not be so.
13.	Overturned stool, chair etc below feet	Generally present	Generally absent
14.	Psychological autopsy	Points to suicidal tendency	Not so

5. Judicial hanging

Judicial hanging is a method of execution in which a noose is tied round the neck of condemned criminal, and he is made to drop between 1-5 m depending on his weight.

Salient features:

(1) It is an official method of execution in India and several other countries

(2) **Procedure** – (a) The face of the victim is covered with a black mask (b) hands tied behind back(c) Made to stand on a platform above trapdoors when opens downwards when a lever is pushed. (d) On pushing the lever, the victim falls equal to the length of the rope which lies loose (e) As the victim comes to a sudden stop, his head is thrown in a direction opposite to where the knot was placed. If it is placed submentally (the ideal position), the head would be hyperextended, causing traumatic spondylolisthesis of axis [the so-called "**hangman's fracture**"; bilateral fracture of pars interarticularis of C2]. Depending on its severity, 3 types have been described (**Fig 19.15**). In subaural position of the knot, typical hangman's fractures are rare; fractures of the base of skull are more common. Other fractures that have been described in both placements of knots are bilateral fractures of pedicles or laminae of the C1, C3 and C4, with associated dislocations, and fracture of odontoid process of C2. Their frequency is lesser.

(3) **Factors essential to cause quick death** – In order to reduce the suffering of the victim, it is desired that there be a quick death, or at least a rapid onset of loss of consciousness. To achieve this, two most important factors are knot positioning and the length of drop. (a) **Position of knot** – Submental is most effective, but **left subaural** was used officially because it does not slip. (b) **Length of drop** - Ideally it is between **1-5 m** depending upon the weight of the victim. If drop is too short (<1 m), asphyxiation would result; if it is too long (>5 m), decapitation. Hence, in 1875, tables were proposed that regulated the drop length in proportion to the individual's weight. This list of drops was then modified in 1886 by the Capital Sentences Committee concluding that for an 'efficient' hanging the victim's neck should be subjected to a force of **126 pounds**.

(4) **Cause of death** - (a) **Bilateral fracture of C2** with anterior subluxation (C2-3 dislocation), resulting in fatal injury to the spinal cord [most commonly with submental knot]. (b) **Dislocation of atlanto-occipital joint** with injury to spinal cord [most commonly with left subaural knot] (c) **Odontoid process of C2 fracturing** and causing pulping of spinal cord (d) **Complete transection of the cervical spine** causing the neck to be lengthened considerably. In proper judicial hanging there should be instantaneous and irreversible loss of consciousness and irreversible apnea. Heart keeps beating for 15 minutes due to autorhythmicity. Spasmodic involuntary muscular jerking is seen for the

same period.

(5) **PM findings** - (a) Usual findings seen in hanging. In addition (b) Fractures of base of skull and CV with trauma to spinal cord as mentioned earlier (c) **Intima of carotid** shows transverse lacerations. Sometimes the arteries may show complete tear (d) Injury to **pharynx**.

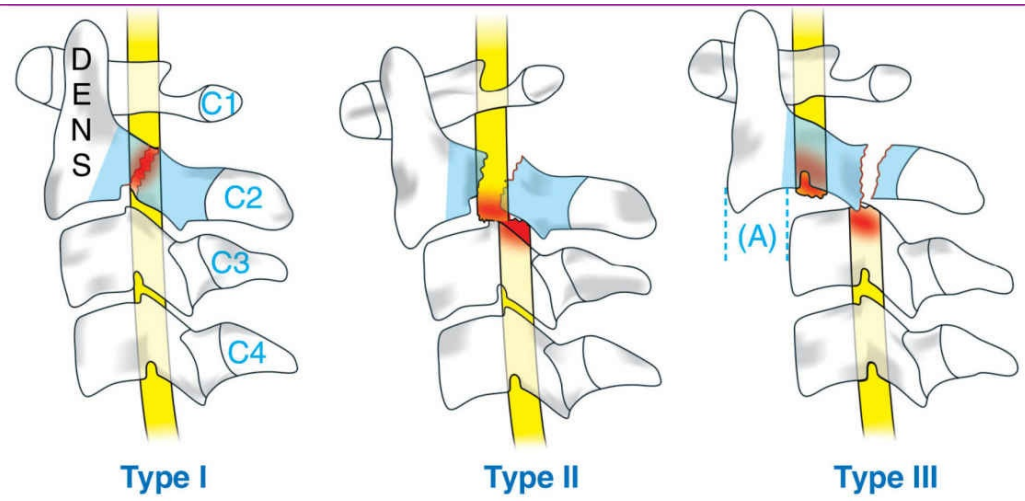


Fig. 19.15: Various types of **hangman's fractures** (*traumatic spondylolisthesis of axis*) classified according to severity. **Type I** hangman fracture - Nondisplaced fracture of the pars interarticularis (in blue); **Type II** hangman fracture - displaced fracture of the pars interarticularis; **Type III** hangman's fracture – Fracture of the pars interarticularis with dislocation of C2–C3 facet joints. Note the anterior subluxation (A) of 2nd cervical vertebrae. Yellow-spinal cord; Red-injury to spinal cord.

IV. STRANGULATION

Strangulation is that form of death which is caused (i) either by exclusion of air from lungs, or oxygenated blood from the brain (ii) by means of a ligature round the neck, (iii) the constricting force being ***anything other than*** the weight of the body.

A. Classification

Strangulation is classified according to the type of ligature used (**Table 2**).

Table 2: Types of strangulation

S.No	Ligature used	Type of strangulation
1.	Traditional ligature (rope, cloth, wire etc)	Ligature strangulation [most common; classical strangulation]
2.	Hands	Throttling (Manual strangulation)
3.	Palm	Palmar strangulation
4.	Bend of elbow	Mugging
5.	Executioner's cord	Garroting
6.	Two pieces of bamboo	**Bansdola**

B. Cause of Death

(1) Same as that in hanging.

(2) However **occlusion of carotid arteries** is more common.

(3) In **throttling**, groping fingers may suddenly impinge upon carotid sinus causing sudden death by vasovagal inhibition. Thus there may be a period of partial asphyxia terminated suddenly by the onset of cardiac arrest.

C. PM Appearances [in classical ligature strangulation]

1. External

a. Ligature Mark

i. Examination

Must be done in (1) regular light (2) oblique light (3) UV light [to visualize faint bruises made by soft materials.

ii. Features

(1) **Regular features** - please see **Table 3**

(2) **Peculiar [additional] features** –

(i) Effects of lateral displacement of blood from the squeezed area - (a)

Reddening and congestion – especially above and below the ligature mark (b) Preservation of mark during putrefaction

(ii) Ligature mark as a patterned abrasion – If a ligature with a pattern [eg chain with links, plaited electric wire, spiral rope or woven belt] is used, a patterned abrasion may form. It details should be noted and photographed, as it is a vital clue in tracing the ligature

(iii) Abrasions around ligature mark – (a) may be vertical, linear, irregular or crescentic. (b) May not be readily visible if the area is wet [due to perspiration, application of wet cloth as ligature]. Body should be allowed to dry and then examined again (c) Could be due to assailant's fingernails [more common in throttling] or victim's own fingernails trying to remove ligature. (d) Victims' fingernails must be examined. May be fractured, broken or may contain blood or tissue tags underneath. (e) Must be clipped and sent for analysis in paper bags. DNA from blood or tissue tags may reveal own DNA, or sometimes DNA of assailant, if he tugged at his skin.

(iv) Bruising of neck muscles – more common if (a) rough ligature is used (b) there was movement of ligature over the skin, as in struggle

(v) Oblique ligature mark – Traditionally it is horizontal [**Table 3**], but may be oblique if the victim was dragged by ligature, after he was strangled in a recumbent position

(vi) Ligature mark interrupted at front – due to (a) victim's fingers attempting to pull the ligature away (b) presence of clothing

(vii) Ligature mark interrupted at back – due to (a) presence of clothing or long hair [esp in females] between the ligature and skin (b) If assailant presses from the front, or pulls from the back, using the ligature stretched between two hands

(viii) If knot has been applied – Wider area of bruising at the site of knot

(ix) If hard ligature [eg cord, rope, wire] is used – Abrasions may be produced, which become hard, yellow-brown and parchment like on drying. There may be exudation of tissue fluid "forms a stiff film on drying

(x) If soft and yielding ligature [eg scarf or towel] is used – (a) only flushing of skin or at most a slight depression may be seen (b) The marks may fade soon after death. Thus in these cases, body should be examined soon after death [importance of examination of body at the scene of crime itself] (c) If mark is completely obscure – (1) Examine under UV light (2) dissect skin

and muscles"presence of hemorrhages would reveal the position of ligature mark

(xi) If a narrow ligature [eg cord, thin rope or wire] is used – The ligature is forced deeply into the skin. After the ligature is removed"skin recoils due to elasticity"Ligature mark may become narrower than the width of ligature [**cf** stretchable ligature]

(xii) If a stretchable ligature [eg nylon, silk, terylene] is used – Ligature may be broad, but by stretching becomes longer and thinner"ligature mark much narrower than the width of ligature [**cf** narrow ligature]

(xiii) If a folded cloth [eg a rolled sari or dupatta] is used – There may be a great difference between the appearance of ligature mark and the ligature [because it is examined unfolded]

(xiv) If a complex ligature composed of several pieces knotted together is used – Marks of knots may be produced at several points, which may be confusing to an untrained mind

(xv) If two sticks are used [bansdola] – only those areas of neck will show marks, where the sticks came into contact with skin

(xvi) If foot is used – (a) wide mark corresponding to width of foot (b) wide area of bruising (c) fracture of laryngeal cartilages more common

(xvii) If ligature has been tightened pulling on the cross ends – (a) Mark more prominent at site of cross-over (b) marks produced by two ends will be at different levels (c) Cross-over mark may be at front, back or side depending on relative positions of victim and assailant

(xviii) Multiple turns – (a) produce a complex mark, in which sometimes it may be possible to count the number of turns (b) if folds of skin are caught between multiple turns – pressure on capillaries may rupture them leading to petechial hemorrhages. These confirm the antemortem nature of strangulation. Vesicles containing fluid may also form

(xix) Effect of strong pressure – (a) Laceration of skin (b) fracture of cartilages

(xx) Effect of tissue edema – Ligature pressing on neck tissues"edema develops around ligature, especially above"Ligature gets tightened further"more edema"vicious cycle may continue even after death due to passive transudation of tissue fluid"Ligature mark appears much deeper"Impression to the untrained eye is that the ligature was applied very tightly [possibly reflecting anger and rage of assailant], while in fact the ligature may not

have been applied so tightly.

Table 3: Differences between Hanging and strangulation

S.No	Feature	subfeature	Hanging	Strangulation
1. 2.	Ligature mark	1. Appearance and position	(i) pale (ii) hard parchment like (iii) more depressed in skin [due to more wt] (iv) Oblique (v) does not completely encircle the neck (vi) more prominent at front and sides than at the back [in typical hanging] (vii) above thyroid cartilage(viii) damage to skin less likely – if present, damage is more in the front of neck than at the thicker, tougher skin at the back of neck (ix) petechial hemorrhages around ligature less common	(i) reddish (ii) soft (iii) less depressed in skin (iv) transverse [rarely oblique – please see text for details] (v) completely encircles the neck [may be deficient – please see text for details] (vi) equally prominent all round the neck(vii) below thyroid cartilage (viii) damage to skin more likely. Distribution of area of damage same as in hanging (ix) petechial hemorrhages around ligature more common
		(2) Abrasions and ecchymoses at the edges of ligature mark	Not common	Common
3.	Face	(1) Appearance	Usually pale. Petechiae rare	Usually congested. Petechiae common
		(2) External signs of asphyxia	less marked	More marked
		(3) Bleeding from nose, mouth and ear	Not common	Very common
		(4) Swelling and protrusion of tongue	less marked	More marked
		(5) Saliva	Often runs out of mouth [stimulation of parotid gland]	Absent
4.	Neck	(1) Length	May be stretched or elongated	Not so
		(2) Bruising of neck muscles	Less common	Common
		(3) S/c tissues under the mark	White, hard, glistening	Ecchymosed
		(4) Carotid arteries	Damage may be seen [more force (body wt) acting]	Damage rare [less force acting]
		(5) Hyoid bone fracture	Usually absent	Usually present, especially in manual strangulation
		(6) Thyroid cartilage	Usually absent	Usually present, especially in manual strangulation
		(7) Laryngeal and tracheal cartilages	Usually absent	May be present in old people, when the cartilages have ossified
5.	External features	Involuntary discharge of urine and feces	Less common	Very common [Parasympathetic stimulation]
		Seminal fluid at glans	More common	Less common
6.	Internal organs	Emphysematous bullae on surface of lungs	Not present	Very common
7.	Circumstantial evidence	Suicide note	May be present	Absent
		Place of occurrence	Usually own bedroom. Doors and windows bolted from inside	Outside in open spaces. If inside room, it is not bolted from inside
		Signs of struggle, eg disturbed furniture, rumpled bed sheets etc	Absent	Present
		Psychological autopsy **[ch 5]**	Previous h/o unsuccessful suicide attempts may be there. Victim often depressed or under stress	No such history. Known enmity with other people

b. Pseudostrangulation

Pseudostrangulation refers to a situation, where a ligature mark resembling a strangulation mark [pseudoligature mark] is seen on the neck, but it is produced by mechanisms other than antemortem pressure by a ligature.

Salient features:

(1) **Causes:** (i) Blanching of pm staining - by a tight collar or necktie etc [**ch 9**] (ii) Decomposing bodies – with buttoned shirt at the neck, tight collars, or other clothing round the neck. As body decomposes, tissue swells around the tight fitting garment, producing a mark simulating strangulation mark. (iii) Mummified bodies – May show similar marks (iv) Infants and children (a) if they have short neck – Produced from skin folds due to bending of head (b) hypothermia - [**ch 22**] (v) Short necked adult individuals – Same mechanism. Much less commonly seen than in children.

c. Signs of Asphyxia

(1) Classical signs of asphyxia are seen [please see at the beginning of chapter]. The signs are marked if pressure applied >30 kg [please see below]. **Additional features**.

(2) Head, face and neck – congestion and petechial hemorrhages.

d. Other signs

(1) **Abrasions** and **contusions** on **face** – if there was struggle

(2) **Effects of Pressure** – [For principles, please see above under “Medicolegal aspects of hanging"Pressures required to obliterate neck structures”]

(i) If pressure applied is <2 kg - no major structure is blocked. Death is due to vasovagal shock. Most external and internal signs will be absent

(ii) If pressure applied is between 2-30 kg – Most common situation (a) Marked congestion of face. (b) Bleeding from nose [epistaxis], mouth [stomatorrhagia] and ears [otorrhagia] due to engorged capillaries [**Fig 19.16**]. (c) Examination of tympanic membrane [TM] with an otoscope – If there is frank bleeding from ears, TM will be found ruptured. Source of bleeding is middle ear. If no bleeding"TM would still be found congested.

(iii) If pressure applied >30 kg – Asphyxial signs are marked.

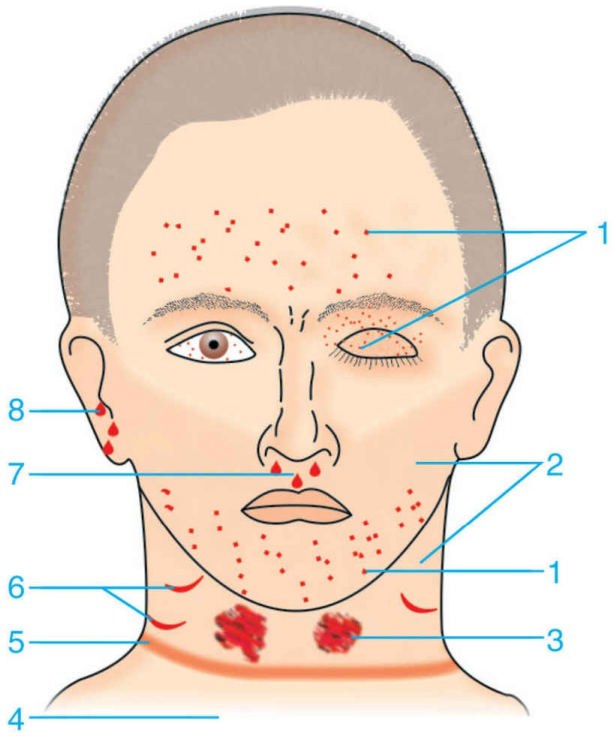


Fig. 19.16: Classical features of strangulation. **(1)** Petechiae **(2)** Congestion **(3)** Abrasions and bruises **(4)** Paler skin below neck **(5)** Ligature mark **(6)** Fingernail marks **(7)** Nose bleed **(8)** Ear bleed

2. Internal

(1) **Neck** - Hemorrhages
(i) beneath the ligature mark under the skin
(ii) in the strap muscles
(iii) sides of tissues around larynx and trachea
(iv) in laryngeal structures
(2) **If assailant kneeled on the chest** – fracture of ribs
(3) **If assailant kneeled on the abdomen** – laceration of abdominal organs, eg liver, spleen, rupture of intestines, gall bladder
(4) **Lungs** - show emphysematous bullae.

D. Medicolegal Questions [in case of ligature strangulation]

1. Whether death was caused by ligature strangulation?

(1) Characteristic PM signs of ligature strangulation would be present

(2) If ligature mark is invisible"use UV light

(3) In presence of putrefaction - Most signs of strangulation cannot be relied upon. fractures of larynx and hyoid may be the only reliable characteristic signs

(4) Mere presence of a ligature around the neck does not indicate strangulation, as it may have been placed there after death to mislead the police.

2. If an infant was found strangulated due to umbilical cord, was it homicidal or accidental?

Please see **Table 4**.

Table 4: Differences between homicidal strangulation and accidental strangulation by umbilical cord in infants

S.No	Feature	Homicidal strangulation	Accidental strangulation by umbilical cord in infants
1.	Cervical tissue injury	More	Less
2.	Knot	More complicated [eg reef knot]	Less complicated
3.	Lungs	Completely expanded	Incompletely expanded

3. Whether the death was suicidal, homicidal or accidental?

a. Homicidal strangulation

(1) Strangulation is mostly homicidal, followed by suicidal and accidental in that order.

Memory Aid 2: Manner of death: hanging and strangulation

Hanging is mostly **s**uicidal; **s**trangulation is mostly **h**omicidal

(2) Differences from suicidal strangulation [**Table 5**].

(3) **Incidence** – much commoner than suicidal strangulation. One of the common methods of homicide.

(4) **Victims** – Usually women. Motive is sexual assault [please see case studies – Boston Strangler].

(5) Homicidal strangulation is sometimes associated with erotic feelings [lust murder, **ch 25**].

b. Suicidal strangulation

(1) **Incidence** – rare

(2) **Methods:**

(i) Like a tourniquet using knots – Simplest and commonest. Ligature may be tied simply like a tourniquet using conventional knots

(ii) Spanish Windlass technique - (a) Technique - A stick or a rod is introduced underneath the ligature and the rod twisted to tighten noose.

Table 5: Differences between suicidal and homicidal strangulation

S.No	Criteria	Suicidal strangulation	Homicidal strangulation
1.	Incidence	Uncommon	Common
2.	Ligature	Always present around neck [in situ]	May or may not be present around neck. If absent, indicates, sure homicide
3.	Number of ligature materials	Usually single. Would lose consciousness after tying the first ligature	Usually single, but if multiple ligatures are present, each firmly tied around the neck, it confirms homicide
4.	Knots	Usually single	Usually multiple. Two or more firm knots each on separate turns of ligature, confirms homicide
5.	Position of knot	Usually in front	Usually at the back.
6.	Venous congestion	Much more developed above the ligature site [because of slow tightening of ligature]. Especially prominent at the root of the tongue	Less developed than that in suicidal
7.	Injuries around neck	Less marked	More marked
8.	Associated injuries	Usually none	Associated stab, firearm or head injury strongly indicates homicide
9.	Signs of violence and marks of struggle	Absent	Present
10.	Scene	Not disturbed	Disturbed
11.	Door	Usually closed from inside	Always open
12.	Suicide note	May be present	Absent

c. Accidental strangulation

(1) **Isadora Duncan syndrome** - Long scarf, end of a sari or a dupatta traditionally worn by women around their necks can get entangled within the spokes of a moving cycle rickshaw and can cause quick death by accidental strangulation." To prevent such deaths, dupattas etc must not be worn while traveling in a cycle rickshaw. Improved designs of cycle rickshaw wheels with protective covers have also been proposed. The syndrome is named after the world famous American dancer **Isadora Duncan** [1877 – 1927], who died on 14 Sep 1929, when the long scarf, she was wearing, got caught in the wire wheels of her Buggati car. She died on the spot. At autopsy she was found to have sustained a fractured larynx and carotid artery injury.

(2) **Seat belts** - In severe accidents, especially involving roll-over of vehicles, there may be "submarining" with sliding of the body under the lap belt if it is not properly adjusted. Much more common in short individuals and those who are incapacitated in some way.

E. Types of Strangulation

Classical strangulation is the **ligature strangulation** (described above). Other types are as below:

1. Throttling (Manual strangulation)

Throttling is compression of neck by human hands. It is a form of strangulation where human hands are used in place of ligature [Manual strangulation].

Salient features:

Cause of death – Occlusion of b.v. of the neck.

a. PM appearances

i. External

(1) **Asphyxial signs** – eg congestion etc on face. Develop within 15-30 sec of pressure on neck, and would continue to -in intensity if pressure is maintained. However fingers may slip and press carotid structures in which case reflex cardiac arrest occurs, causing immediate death. Thus intensity of asphyxial signs would vary in all cases.

(2) **Tongue** – Usually protruded. May nor may not be bitten.

(3) **Neck** – Shows bruises and abrasions (i) Bruises - (a) Mechanism - produced by tips or the pads of fingers. Usually more force is used than is necessary to

kill the victim. (b) Shape – (I) Initially - (1) If fingers are static - Oval, Round and of the size of digits. Size "1-2 cm **[Six penny bruises - ch 12**] (2) If fingers skid across skin surface [as during struggle] – longer irregular marks, especially along the jaw margins (II) Later – in both cases size -es due to continued bleeding (c) Situation and extent – depends upon (I) degree of pressure exerted over throat (II) manner of grasping neck (III) relative size of victim's neck in comparison with the assailant's hands (IV) relative positions of the assailant and victim - (1) Grip from right hand from front [**Fig 19.17**] (i) Thumb impression on right side of victim's neck. (a) Situation - under the lower jaw over the cornu of thyroid (ii) Several finger marks on the left side of neck. (a) Situation – left side of neck, obliquely downwards and outwards and one below the other. Sometimes grouped together; may not be distinguished separately (2) Grip from left hand from front [**Fig 19.17**] - (i) Rare. Seen if the assailant was left handed. Findings are mirror image of the above, i.e. thumb impression on left side of victim's neck etc.

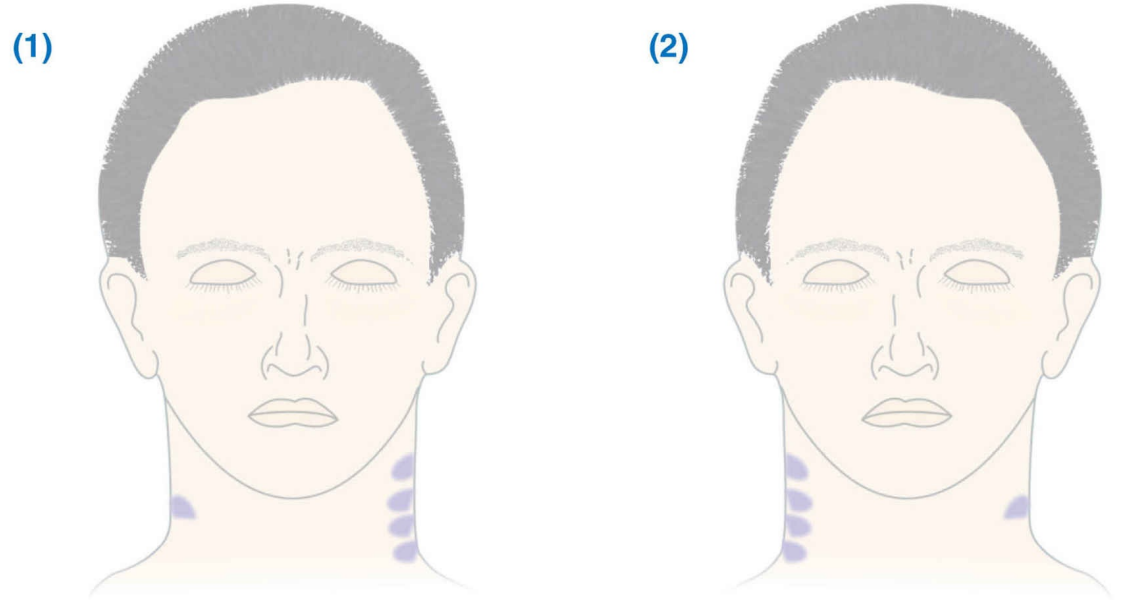


Fig. 19.17: Different types of bruises found in a typical case of throttling. Only 2 representative types are shown here. **(1)** Grip from right hand from front **(2)** Grip from left hand from front

ii. Internal

(1) **S/c tissues and muscles of neck** –

(i) compressed backwards against cervical vertebrae

(ii) engorgement at and above the level of compression

(2) **Bruises and frank hemorrhages** -

(i) Present in (a) dermis (b) superficial fascia (c) deep fascia (d) muscles, especially those surrounding the larynx both anteriorly and posteriorly. (e) neck tissues, especially those surrounding or adjacent to the bifurcation of the common carotid artery (f) base of tongue (g) pharynx (h) tonsils (i) Larynx - especially immediately beneath the vocal cords. Sometimes accompanied by severe laryngeal swelling. May or may not be accompanied by fracture of hyoid and laryngeal cartilages. (j) thyroid gland - beneath the capsule and within the substance (k) Salivary glands - (I) Seen in parotid, submaxillary and submandibular glands. (II) Site - Beneath the capsule and within the substance. (l) Lymphatic glands in the neck, especially in the anterior triangle (m) Tissues at back of neck - when counter pressure has been used.

(ii) Correspondence between external and internal bruising - (a) Extent, severity and situation of internal bruising - Usually matches with (I)the strength and degree of pressure used. (II) the external injuries. (b) Sometimes bruising is absent externally, although internally extensive bruising may be present (c) Rarely may be absent both externally and internally, especially if the victim was unconscious and amount of pressure was minimal. (d) Site of external bruising may not correspond exactly with that of internal, because of mobility of skin.

(3) **Tearing of muscles** - especially sternocleidomastoid

(4) **Carotid arteries** -

(i) Show intimal tears in 10-15% cases.

(ii) Site - at or near carotid sinus.

(5) **Laryngeal cartilages**.

(i) Epiglottis - very rarely shows fractures because of its protected position. Surface however may show a shower of petechial hemorrhages, or frank hemorrhage.

(ii) Thyroid cartilage - (a) Lamina [ala] - may show fractures, either vertically in the midline at the junction of laminae or obliquely or spirally across the left or right lamina, if pressure is much greater. **For trauma to front of neck** please see **ch 17**. (b) Horns [cornuae]- fracture of superior horns are much more common than that of laminae [**Fig 19.18**] because they are more

vulnerable and fragile than inferior horns. Frequently only the right superior horn is fractured, because it directly underlies the thumb. (c) Abnormality in the thyroid cartilage can be taken for fracture, and a small cartilage triticea within the lateral thyrohyoid ligament can be mixed up with a broken part of the superior cornu. (d) Like hyoid fractures, in general fractures of thyroid too are more common in older people [because of calcification of thyroid cartilages] (e) Because of greater force required to fracture them, thyroid cartilage fractures are more serious than those of hyoid bone.

(iii) Cricoid cartilage - (a) may show fractures. (b) Almost exclusively fractured in throttling. (c) its fractures are almost always associated with fatality. (d) May fracture across the front, midline or laterally.

(iv) Fractures are especially common in old persons with calcified cartilages

(v) Demonstration of laryngeal fractures - best done by first stripping the larynx of its attached muscles and ligaments.

(6) **Trachea:**

(i) Tracheal cartilages may be fractured rarely, especially if the victim is old [having calcified cartilages]

(ii) Trachea may show frank lacerations.

(7) **Hyoid bone** - Of all forms of compression of neck, fracture of hyoid is commonest in throttling. Occurs in 30-50% of cases, with inward driving of its distal fragments [inward compression fracture.

(8) **Lungs** -

(i) Congested with subpleural petechial hemorrhages.

(ii) Pulmonary edema.

(iii) Blood stained fine froth in bronchi

(iv) Microscopy - Some areas overdistended with rupture of alveolar walls and some areas are collapsed; hemorrhages in the interstices under the pleurae

(9) **Brain** - Congested. Petechial hemorrhages in the white matter. Subarachnoid hemorrhages.

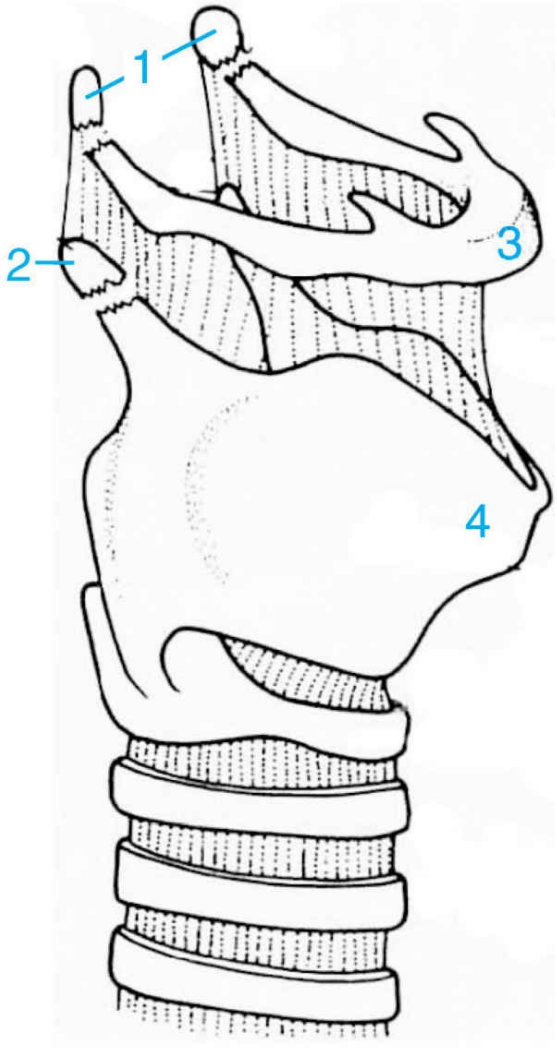


Fig. 19.18: Thyrohyoid complex and various fractures seen in throttling. (1) Fracture of greater cornu of hyoid (2) Fracture of superior thyroid horn (3) Hyoid (4) Thyroid cartilage

b. Medicolegal questions

i. Whether death was caused by throttling?

(1) **PM findings** - typical of throttling would be present

(2) **If victim was first killed by throttling and then hanged** -

(i) Suspended body shows extensive injuries to neck structures

(ii) Signs of violence on body

(iii) Signs of sexual assault and rape may be present on women victims.

ii. Can PM findings similar to those of throttling be produced by other causes?

Yes. In hard compression of neck eg air choke, blood choke [two common choke holds], flying kicks, jumping on neck, karate chops and kicking or stamping. These can in fact cause much greater damage [eg bilateral comminuted fractures of larynx] than are usually seen in throttling.

iii. For how long must the pressure be applied to cause death?

For **2 min** or more.

iv. Whether throttling was suicidal, homicidal or accidental?

(a) Suicidal throttling

(1) Suicidal throttling is not possible. Any attempt at suicidal throttling would inevitably produce unconsciousness first. As unconsciousness sets in, fingers relax.

(2) A person not realizing that he cannot throttle himself may first attempt throttling failing which may use ligature. In such cases some fingernail marks may be seen over the neck.

(b) Homicidal throttling

(1) **Incidence** - Because of non-requirement of any weapon whatsoever and immediate availability of hands, homicidal throttling is quite common. In all cases of fingernail marks and contusions over neck, the presumption must be of homicidal throttling.

(2) **Method of choice** - for killing weak individuals who cannot resist eg infants, children and women

(3) **Healthy adults can be throttled easily** when

(i) sleeping or unconscious

(ii) stunned

(iii) taken unawares

(iv) under influence of drinks or drugs or

(v) weakened by disease or poison

(4) **If a normal healthy adult is throttled** -

(i) Signs of struggle would be present on both victim and accused as well as at

the scene of crime.

(ii) Examination of accused may show abrasions, bruising and bitemarks at face, arms, back of hands, fingers etc.

(iii) Fingernail scrapings of the assailant should be taken to compare tissue type of victim.

(5) **Sexual assault and rape** - victim is often throttled during sexual assault and rape. Motive is usually not to kill but to stifle cries. If victim dies, the accused cannot take plea of accidental throttling.

(6) **Defending party** - may often allege that accused was actually trying to save the falling victim by applying his hands over the neck. This is highly improbable.

(c) Accidental throttling

(1) Sudden application of one or both hands or momentary grip on another person's throat may occur during demonstration of affection, joke, physiological experiments, playful activity and sports [eg wrestling]. It cause death from cardiac inhibition, which results from compression of carotid sinus.

(2) **Presence of injuries**

(i) Bruises on neck - death could not have died instantaneously, because formation of bruise requires a beating heart.

(ii) fracture of hyoid or thyroid - death cannot be due to accidental throttling, because a mere touch cannot produce these injuries

(iii) minor damage or absence of damage"indicates accidental throttling, but rarely can be seen even in homicidal throttling.

v. How much force was used by assailant?

Extent of damage - to neck structures. -damage indicates -force"indicates intent to injure if not to kill.

vi. How much time must the victim have taken to die?

Very difficult to ascertain from PM findings. If very little or no injuries are present, death might have been quick indicating less survival period. Severe and extensive injuries indicate longer survival.

vii. Could it have been a case of throttling in a person about to die from other causes?

In throttling during perimortem period [**ch 11**], nail marks may appear much the same. Thus it is difficult to say whether deceased was alive or dead at the time.

2. Palmar strangulation

Palmar strangulation is a type of strangulation, in which one palm is placed horizontally across the mouth and nostrils, and the other palm placed perpendicular across the first in such a way, that its heel presses upon the front of neck.

Salient features:

(1) **Mixed with smothering** - There is an element of smothering also (please see below). The lower palm smothers, while the upper strangulates.

(2) **Motive** - Generally used to stifle cries from the victim. The upper palm is used mainly to reinforce the lower palm used to stifle cry. Strangulation may not be the primary motive of assailant.

(3) **Victim** – Easier to accomplish if victim is intoxicated

(4) **PM findings** – Same as that in ligature strangulation, except ligature mark.

3. Mugging

Mugging [syn, arm lock, choke hold] is strangulation caused by holding the neck of the victim in the bend of the elbow.

Salient features:

(1) **Two types**. (i) Air choke [syn, bar arm control, choke hold] – Pressure is exerted on front of larynx (ii) Blood choke [syn, carotid restraint, carotid sleeper, lateral vascular neck restraint, sleeper holds] – pressure on both sides of neck.

(2) **Attack** - in both is made from behind

(3) **Mugging by lower limbs** – rare. Neck pressed by foot or knee. It may be repeatedly stamped upon

(4) **Cause of death** - (i) asphyxia (ii) reflex cardiac arrest

(5) **PM appearances:** (i) those of ligature strangulation with a broad object, i.e. minimal signs. (ii) Diffuse abrasion along the jaw margin – [due to friction of forearm] (iii) Diffuse bruising – behind larynx and in strap muscles of the neck (iv) fracture of hyoid or thyroid is rare (v) in repeated stampings by foot – (a) crushing of larynx and trachea (b) frank bleeding in soft tissues and muscles of the neck (c) swelling (d) intima of carotid damaged (e) damage to vagus and phrenic nerves.

4. Garroting

Garroting is a method of killing by asphyxia, where the victim is attacked from behind without warning, and his throat is constricted.

Salient features:

(1) **When used as judicial Execution:** (i) It was a form of judicial executing once practiced in Spain and Turkey. Sometimes there may be an iron collar around the neck which is tightened by a lever. (ii) In one variation there is a sharp spike at the back of the execution chair. As the ligature is tightened, the neck is forced against the spike which penetrates the spinal cord and immediate kills victim.

(2) **When used as a method of homicide:** (i) Results in sudden loss of consciousness and collapse. The assailant is then able to tie a ligature around neck without resistance. Even a relatively weak assailant can kill a healthy adult. (ii) **When used** - by robbers at lonely places to attack and rob travelers.

5. Bansdola

Bansdola (doing away with bamboos) is a form of strangulation, where compression of neck is achieved by bamboos.

Salient features:

(1) **Method** – Three procedures are usually employed (i) One bamboo is placed over the front of neck and the other at the back. Both ends are tied to achieve compression of neck (ii) victim is forced against ground"Bamboo is placed across the front of neck"Assailant stands with a foot on each end of the stick. (iii) Victim is pinned against a wall. Bamboo placed across the front of his neck and pressed hard towards the wall with both hands.

(2) **What is used:** (i) Bamboo - most commonl (ii) sometimes a supple branch of a tree is also used

(3) **PM findings:** (i) Same as that in ligature strangulation. (ii) In place of ligature mark, bruises on front of neck corresponding to the placement of bamboos are seen (iii) If 2 sticks are used, similar bruises over back of beck (iv) If used against chest, similar bruises on chest. Other findings of traumatic asphyxia.

V. HYOID BONE FRACTURES

Hyoid bone fractures are commonly seen in compression of the neck, eg hanging, strangulation and throttling. Lacerations of pharynx may occur following fractures of hyoid bone. Hyoid bone fractures are of **2** types - **inward compression fractures** and **outward compression fractures**.

A. Types of Hyoid Fractures

1. According to displacement of fractured ends

a. Inward (lateral) compression fractures

(1) Occur due to inward compressive force as occurs in **throttling**.

(2) **Mechanism** - Fingers of the grasping hand squeeze the greater horns towards each other"posterior [distal] fragment is displaced inwards [**Fig 19.19(a)**].

(3) **Fracture** - may be unilateral or bilateral. May occur at 2 places on either side

(i) Just at the base of distal fragment

(ii) At the joint between the greater horn and body of hyoid. Great care must be taken to ascertain it, because a natural joint may be confused with a fracture.

(4) **Periosteum** - torn on the outer side of the bone which is overstretched. Not torn on the inner side [**Fig 19.19**].

(5) **Complete detachment of smaller fragment from the hyoid** - may occur if the compression is severe. The smaller fragment may seen to be lying medially to the rest of the bone, if neck structures have not been disturbed during dissection. This can best be done by an in situ examination of hyoid.

(6) **Preservation of hyoid as exhibit** - Ideally hyoid should be preserved as an exhibit. After preservation, sealing and labeling, it should be handed over to the IO.

b. Outward (Anteroposterior) compression fractures

(1) **Outward fracture** - In case of **hanging**, as ligature presses on the neck, the hyoid is forced directly backwards"-es divergence of greater horns"Outward displacement of posterior fragment occurs, with rupture of periosteum on the inside [**Fig 19.19(b)**].

(2) **Nature of fracture** - Like inward compression fractures, it may be unilateral or bilateral, and like it may occur at 2 places. Similarly complete detachment of smaller fragment from the hyoid may occur if the compression is severe. The smaller fragment may seen to be lying laterally to the rest of the bone.

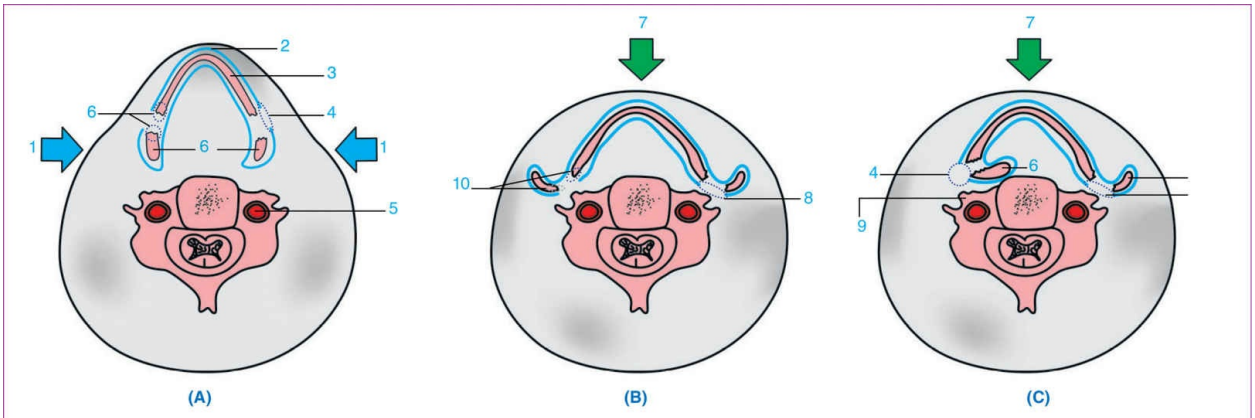


Fig. 19.19: Types of hyoid fractures. **(A) Inward compression fractures** [*seen in throttling*] **(B) Outward compression fractures** [*seen in hanging*] **(C)** One cornu **fractured inwards** and the **other outwards** [*seen in hanging, when one end is caught up against a bony ridge or sides of the vertebrae*].(1) Inward compressive force as occurs in throttling (2) periosteum over the hyoid (3) hyoid (4) periosteum torn on the outside (5) vertebral artery (6) Inward fractures of greater cornu of the hyoid (7) Backward force on hyoid as occurs in hanging due to ligature (8) periosteum torn on the inside (9) Bony ridge of the vertebra, in which greater cornu may get caught causing inward fracture (10) Outward fractures of greater cornu of the hyoid

B. Demonstration of Fractures

(1) **Palpatory method:**

(i) Hyoid body is grasped in one hand

(ii) Distal fragment [tip] is held between the finger and thumb of other hand

(iii) A gentle attempt is made of bend the distal fragment both on the inside and outside

(iv) In case of **inward compression fracture**, it can be easily bent in an inward direction [because of torn periosteum outside]; but outward movement is limited to the normal position only [because of intact periosteum on inside]. Exactly the opposite happens in **anteroposterior compression fractures**.

(2) **Toluidine blue with stereomicroscopy** - 1% Toluidine blue sol^{n} is applied to suspect areas, left for 15 s and cleaned off with pure water. Examine bone with a stereomicroscope [5-18 times magnification]. fractured sites would appear bluish.

(3) **X-ray and CT** - before start of autopsy are the best and most certain method to ascertain fractures. If for some reason, could not be done before start of autopsy, the thyrohyoid complex should be carefully dissected out and x-rayed separately. The x-ray or CT plates should be preserved as evidence.

VI. SUFFOCATION

Suffocation is exclusion of air from lungs from any means other than ligature [**Fig 19.20**].

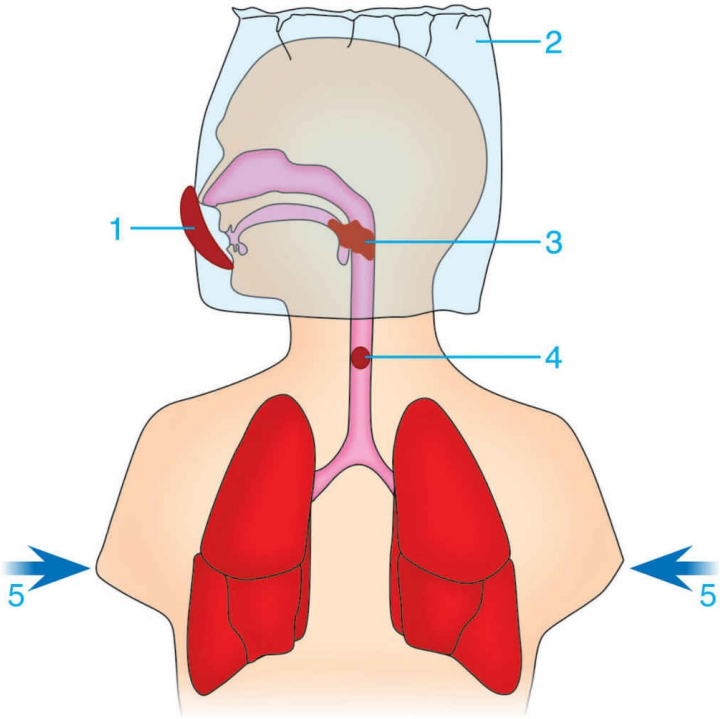


Fig. 19.20: Various forms of suffocation. **(1)** Covering of nose and mouth (smothering) **(2)** Enveloping plastic (smothering) **(3)** Gagging **(4)** Choking **(5)** Traumatic asphyxia

A. Environmental Suffocation

Environmental suffocation is deprivation of oxygen due to lack of oxygen in the environment.

Salient features:

(1) There may be deficiency of O_2 in the environment, or it may be replaced by an inert gas as N_2. Replacement of O_2 by a poisonous gas such as Cl_2 or H_2S would not cause death primarily by asphyxia, but by poisonous effects.

(2) Conc of O_2 in air is 21%. Its fall to 16% or less is dangerous. With 5% conc consciousness is lost rapidly and death occurs within a few minutes.

1. Causes

(1) **During play** - children may get locked in large boxes, trunks, or old disused refrigerators

(2) **Glue sniffing** [inhalant abuse]- [**ch 46**]. Smothering may also be involved [please see below]

(3) In the vicinity of **lime kilns** and wells or excavations in chalk rock

(4) **In confined spaces** – eg deep tanks of a ship, fermenters, grain bins, sewers, silos, tanning vats and unused wells, dusts, hazardous gases, fumes and vapors accumulate and oxygen may become deficient. A person may be suffocated on entering such a confined space

(5) **Cabin failure of aircraft** - at high altitudes

(6) **Natural upheavals** - eg CO_2 produced as a result of **limnic eruptions**. [please see ch 44 for details – under the heading "MLI of CO_2"].

2. PM findings

(1) As in asphyxia. (2) Petechial hgs are however absent (3) Head and face – pale

B. Smothering

Smothering is caused by closing the external respiratory orifices by hands or other means [eg a pillow].

1. Accidental smothering

(1) **Prevalence** - Most common manner of smothering

(2) **Circumstances causing AS**-

(i) Accidental fall - in large quantity of semisolid or finely divided material eg ashes, coal dust, grain, hay, mud, sand etc so that his mouth and nose are obstructed. Victim may struggle and try to breathe"inhales some material in air passages, and swallows some material in his stomach. PM examination will show matching material in air passages, lung, esophagus and stomach. Partially the death is due to choking also.

(ii) Autoerotic asphyxia - Please see ***sexual hanging*** above.

(iii) Children – playing with plastic bags over their faces. May get accidentally suffocated. It is not necessary for the open end [mouth] of bag to be tied around neck. Even with bag open, enough CO_2 may accumulate to cause CO_2 narcosis. Even a flat plastic sheet may adhere to face due to electrostatic forces and block mouth and nose. More efforts to breathe, or trying to move sheet away can generate more electrostatic forces causing sheet to stick harder

(iv) Epileptic or intoxicated person"buries his face in pillow or mattress

(v) Infants - (a) Infants covered with heavy bedding, blankets or sheets - may or may not die of suffocation, depending upon the force with which bedding etc obstructed the air passag es. (b) Infant not covered with heavy sheets, but overturns his face in bed"buries it in soft pillow or mattress"2 situations may occur now (I) complete blockage"Smothers immediately (II) incomplete blockage"does not smothers immediately"gradually face becomes more congested because it is dependent; furthermore blood traces, edema fluid, mucus and saliva keep accumulating over the pillow"blockage is complete"smothering occurs

(vi) Glue sniffing [inhalant abuse].

(vii) Newborns - Membranes covering the head during birth etc [**ch 27**].

(viii) Person sexually assaulting female"covers her face and mouth with hands or pillow to stifle her cries"when the act is complete he finds that the female has died of AS.

2. Homicidal smothering

(1) **Possible** when

(i) victim is incapacitated (a) from drink or drugs (b) by a blow to the head

(ii) victim is very young, weak or old

(2) **In children** – easily achieved by either covering face with pillow, or pinching nostrils by one mouth and closing mouth with the other.

3. Suicidal smothering

(1) **Suicidal smothering by hands** - impossible

(2) **Possible** – by

(i) Burying face in a mattress

(ii) Lying against the bed clothing to obstruct nose and mouth

(iii) tying a polythene or similar bag over the head.

(iv) Usually seen in mentally retarded persons.

(3) **Cut throat wounds** – if trachea is completely cut and soft parts obstruct the trachea, it is smothering.

4. Examination of suspect

(1) Saliva, blood, squamous epithelium, tissue cells on assailant’s hands or

pillow and other smothering material etc used to cause blockage

(2) Other Struggle marks

5. Autopsy findings

(1) **Slow deaths -** All classical asphyxial signs and symptoms are severe, if death occurs by slow asphyxia [more common]

(2) **Rapid deaths -** If death is rapid due to reflex cardiac arrest"asphyxial signs may be absent. They are classically absent in plastic bag suffocation.

a. External

(1) Obstruction by Bed clothing, cushion, Pillow etc applied with skill may not leave any external signs of violence, especially if the victim is too young or old.

(2) **Head and face:**

(i) Pale

(ii) Plastic bag – may be found over face. It may contain (a) Moisture – in case of plain **plastic bag suffocation** (b) inhalants – in case of **inhalant abuse**. Chemical examination is vital

(3) **Nose and mouth:**

(i) Oozing of blood

(ii) Skin around nose and mouth is pale white due to application of pressure

(iii) Scratches, nail marks, lacerations around nose and mouth

(4) **Congestion, petechiae**"rare. May be present if victim fights and struggles for breath

(5) **Nasal cartilage**"fractured

(6) **Frenulum of upper lip**"Lacerated

(7) **Lips, gums, tongue:**

(i) bruised, lacerated. In doubt, confirm by microscopy

(ii) Tongue - may be bitten and protruded

(8) **Anterior teeth**"broken.

b. Internal

(1) **Back of throat** – copious mucus

(2) **Air passages:**

(i) show blood stained frothy fluid, containing RBCs and desquamated respiratory epithelial cells.

(ii) If person is buried alive, show earth and sand.

(3) **Lungs** – Congested, edematous, show petechiae, areas of hemorrhage and collapse [atelectasis]. Some areas show emphysema

(4) **Internal organs** – deeply congested. Show petechiae.

C. Overlaying

Overlaying is accidental death by smothering caused by a larger individual sleeping on top of a smaller individual, usually an infant.

Salient features:

(1) **Occurrence** - Most commonly it occurs when mother shares bed with an infant and during sleep rolls over the infant and crushes him. In many cases, the mother is found to be under the effect of drugs and alcohol.

(2) **PM Findings** - (i) Signs of asphyxia, (ii) flattening and paleness of face and nose, (iii) nostrils filled with blood stained froth, (iv) pillows and garments [of both mother and child] stained with such blood stained froth.

D. Gagging

Gagging is a form of asphyxia which results from closure of airways by forcing a gag material [rolled up cloth, paper balls etc] in the mouth.

Salient features:

(1) **Gag** - The material which obstructs airways.

(2) **Position**- The gag must be pushed in the mouth sufficiently deeply, so that it touches the posterior wall of the pharynx (complete gagging). In incomplete gagging, when the gag is not pushed up to the back, oral secretions may cause the gag to swell and cause death some time later.

(3) **Manner** - (i) Suicidal (ii) Homicidal – (a) Unwanted infants - may be killed by this method [**ch 27**] (b) In adults - Robbers may stuff a cloth inside mouth of a chowkidar to keep him silent. There is simultaneous tying of hands (to prevent removal of gag) and legs (to prevent running away). The cloth may gradually swell up and cause gagging. Strictly speaking it is not homicidal, as the intention of robbers was only to keep him silent. Generally more than one person is required to gag a healthy struggling adult individual [one needed to restrain, and another to gag]. (iii) Accidental – (a) False dentures – may impact throat during anesthesia (b) Dental procedures - Gagging [mostly nonfatal] commonly occurs during dental procedures, such as making a

maxillary impression. Causes (I) Tactile stimuli - dentist's fingers or instruments contacting the oral mucosa or even by (II) Non tactile stimuli - Patient seeing the dentist or remembering a previous dental experience. (c) Injuries to nose and mouth – Blood may seep to the back of the throat and may clot, producing an "artificial gag" of clotted blood. [if it trickles down trachea, it would be choking] (d) Breast pushed too far in the mouth of a suckling baby (e) During sexual activities [Fellatio]

(4) **Cause of death:** (i) Asphyxia [most common] (ii) Reflex vagal inhibition [rarely]

(5) **PM appearances:** (i) classical signs of asphyxia (ii) Cloth must be removed and sent for forensic examination [fibre, manufacturer or laundry's mark, saliva etc].

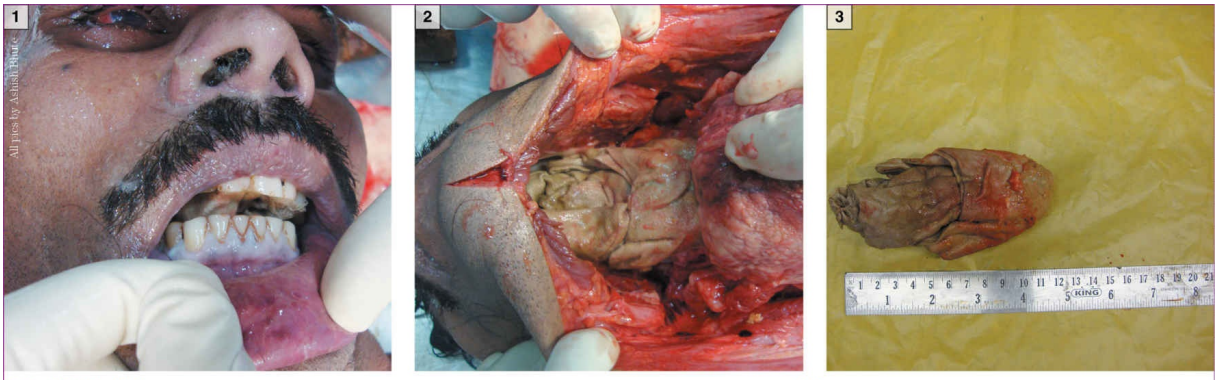


Fig. 19.21: (1) Case of accidental gagging. Gauze piece can be seen in the mouth behind teeth **(2)** On reflecting the tongue, the piece can be seen extending back upto epiglottis, completely blocking airways **(3)** Gauze piece taken out. It is a large piece measuring 12x6 cm.

E. Choking

Choking is a form of asphyxia caused by an obstruction within the air-passages.

Salient features:

(1) **Manner of death** - Choking is almost always accidental.

(2) **Level of blockage** – foreign body gets arrested at or just below the vocal cords. May produce an inflammatory reaction with edema.

(3) **Seen most commonly in** – People whose power to swallow or masticate is severely impaired, eg (i) very young (ii) very old (iii) psychiatric patients (iv) sick and infirm

(4) **Common circumstances** – (i) laughing or crying simultaneously with food intake (ii) ingestion of alcohol

(5) **Objects causing choking** – Any small object which can enter trachea can

cause choking. Most common objects are (i) Balloon [when trying to inflate it] (ii) Battery (iii) Button (iv) Cloth [esp after ENT operations, eg tonsillectomy] (v) Coin (vi) Corn (vii) Cotton (viii) Dentures (artificial) (ix) Fish (live) [as during some unorthodox treatments] (x) Fish bone (xi) Food [eg loaf, meat, milk] (xii) Fruit stone (xiii) hairpin (xiv) Leaves (xv) Marbles (xvi) Mud (xvii) nails [during biting](xviii) Onion (xix) Peanut butter, (xx) pen cap (xxi) Potato (xxii) Rag (xxiii) Rice [**Fig. 19.22**] (xxiv) ring (xxv) Rubber balloons (xxvi) Rubber teat (xxvii) Seed (xxviii) Tooth (broken, extracted by a dentist).

(6) **Choking by vomit** or **regurgitated food** - (i) during anesthesia (ii) insensibility from any cause eg poisoning (iii) during rape or violent sexual intercourse(iv) Head injury – irritation of brain causes vomiting, which may be inhaled due to unconsciousness (v) during fit of epilepsy (vi) Infants – usually regurgitate clotted milk after a meal and choke on it (vii) Tranquilizing drugs – may suppress gag reflex and may cause choking especially psychiatric patients

(7) **Choking by Blood** – (i) Facial injuries during blunt trauma as in a fall, fight, vehicular accident etc [broken nose, dislodged teeth, laceration of lips and gums] (ii) ENT operations [in Dr. Suresh Gupta vs. Government of NCT of Delhi 2004, the patient choked during an ENT operation – **ch 2**].

(8) **Choking by Gauze piece** - during operations

(9) **Natural diseases** - (i) Diptheria (ii) growths in epiglottis larynx and bronchus (iii) H. influenzae infection in children (iv) hemoptysis in pulmonary tuberculosis (v) hemorrhage into trachea (vi) infectious mononucleosis (vii) pharyngeal abscess (viii) rupture of aortic aneurysm in air passages and (ix) a tuberculous gland eroding in a bronchus and prolapsing in its lumen.

(10) Inhalation of steam and ingestion of irritant substances

(11) **Allergic reactions** – (i) Drug reactions [eg penicillin] (ii) Insect bites [bees, wasps, hornets (**ch 38**)]. May cause swelling of laryngeal mucosa and cause a quick death by choking.

(12) **Blow to the front of neck** – may cause hemorrhage and severe laryngeal edema resulting in choking. Sometimes death may occur due to reflex vagal inhibition.

(13) Very rarely pins, safety pins, small bone pieces may be lodged in the air passages for relatively long periods without causing any serious problems.

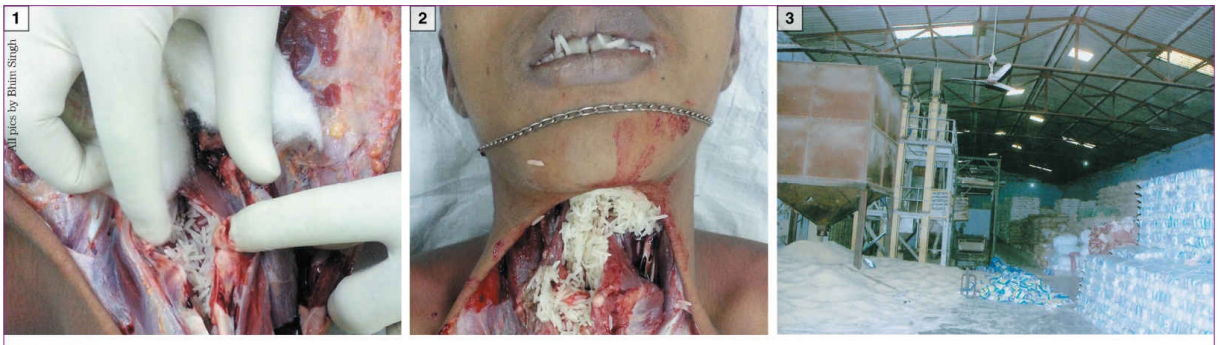


Fig. 19.22: Choking on rice. This 18 y old laborer was working on a rice husker - an agricultural machine used to automate the process of removing chaff (the outer husks) from rice grains. He accidentally fell in the machine and choked on rice. **(1)** Please note rice grains within the trachea. **(2)** Some rice are seen on face and mouth between the teeth also. **(3)** The tank of rice husker in which the victim fell. Its capacity was 10 tons, but at the time victim fell in it, it had 6 tons in it. Please note rice on the floor, which emanated from the tank after getting husked.

1. Course

If patient regurgitates object and survives, complications may develop after a latent interval.

2. Mechanism of death

Death results from anoxic anoxia. There may be

(1) Complete obstruction of larynx

(2) Incomplete obstruction of larynx resulting in laryngeal spasm. Sometimes laryngeal spasm passes off before hypoxia becomes fatal.

3. Cause of death

(1) **Immediate** – Asphyxia

(2) **Late** – Complications eg pneumonia, lung abscess, bronchiectasis.

4. PM findings

(1) Typical of asphyxia.

(2) **Air passages** – Larynx and finer branches of bronchi show pieces of food. Air passages show excessive secretion of mucus in which food is embedded. Mucosa is deeply congested.

(3) **Microscopically** – Lungs – intense interalveolar edema. Collection of desquamated respiratory tract epithelium.

5. MLI

(1) **Accidental** – Most common

(2) **Suicidal** – rare. Victims are usually psychiatric patients [dementia, psychosis] or prisoners. A foreign body is forcibly thrust inside the throat.

(3) **Homicidal:**

(i) **Infants** – Infanticide is practiced by stuffing a wad of paper or cloth into the larynx [if it remains in mouth, it is gagging]

(ii) **Adults** – rarely employed because of resistance offered by a healthy conscious adult. May be practiced in old frail persons, and persons suffering from some debility, disease, paralysis or in drunk and intoxicated persons.

6. Café coronary

Café coronary [syn, bolus death] is a condition in which a healthy person who begins a meal, suddenly collapses and dies without any further distress. The syndrome was first described by **Haugen** in **1963**, when he described it in 9 cases.

Salient features:

(1) **Predisposing conditions:** (i) Age - middle aged or elderly (ii) Gross inebriation, or use of sedative drugs. (iii) Drugs – especially anticholinergic and antidopaminergic drugs. They are known to interfere with swallowing mechanisms through reduction of salivation, .ed esophageal motility, delayed gastric emptying, and aggravation of gastroesophageal reflux. Drugs alone do not - the risk of café coronary, but combined with old age and/or psychiatric conditions, the risk is significantly -ed. Patients receiving high dosage of both categories of drugs also have -ed risk of choking. They must be monitored closely. (iv) Institutionalization - in long-term medical care facilities (v) Natural diseases - most notably Parkinsonism, clinically significant neuromuscular dysfunction, dementia or mental retardation (vi) Poorly chewed food – One of the factors responsible for it is the individual's dental status. They either have several teeth missing, are edentulous or have artificial dentures. Children who have inadequate dentition are known to choke on food too [**crèche coronary**]. (vii) Quick gulping. Haugen called it "the obvious lack of ordinary table manners". Combination of two or more conditions make the person more liable to café coronary.

(2) **Size of food bolus** – usually very large. It could be large size of bolus, which impinges upon the sides of larynx and stimulates the vagus nerve.

(3) **Incidence** - Overall incidence is 0.66/100,000 population/y. It -es with age beginning at about the fourth decade [30-40 y] of life. The peak incidence of 10-14/100,000/y occurs at the 7^{th} decade [60-70 y].

(4) Overall **male: female ratio** is 1:1.6.

(5) **Place of occurrence** – Despite the name café coronary [Fr, coronary attack occurring in a café or restaurant], only about $1/3^{rd}$ cases occur in a restaurant. Other places are private homes, nursing homes, or mental institutions. In **85%**

of cases, other people are present at the time of the fatal incident.

(6) **Type of food** - The type of food most often associated with fatal café coronary is **meat** [74%]. Most often it is steak. **Feces** have been found in mentally ill [**coprophagic café coronary**].

(7) **Level of obstruction** - supraglottic in 1/3rd; infraglottic in 2/3rd.

(8) **Signs of choking** – are absent both during life [clinically] and at autopsy [pathologically].

(9) **Cause of death** – reflex vagal inhibition of the heart. Suddenness of death is most marked.

(10) **Mechanism** – old age, gross inebriation and/or neuromuscular incoordination causes malfunctioning of deglutition mechanism"food slips within the larynx, bifurcation of trachea or within the bronchus"Gross inebriation and/or neuromuscular disorder inhibit gag reflex"food continues to remain in windpipe"stimulates recurrent laryngeal nerve, a branch of vagus nerve" vagus nerve stimulated"Cardiac standstill.

a. PM appearances

(1) **Larynx** – A large piece of poorly chewed piece of food is found. Sometimes nothing is found, as the obstructing piece of food has been removed by bystanders.

(2) Food in finer branches of bronchi is typically absent, because death occurs immediately and no forced inspirations occur.

(3) Findings typical of asphyxia are absent [**Table 6**].

Table 6: Differences between choking and café coronary

S.No	Feature	Choking	Café coronary
1.	**Air hunger**	**present**	**No**
2.	**Gag reflex**	**present**	**Absent**
3.	**Violent coughing**	**yes**	**No**
4.	Association with food	No particular association. Can choke on anything	Yes
5.	Association with alcoholism	Usually no	yes
6.	Associated neuromuscular disorder	No	May be present
7.	Time of death in relation of obstruction	Takes 3-6 min	Immediate.
8.	Age	Any age. However most common are very young and very old	Most often in old age
9.	Cause of death	Asphyxia	Reflex vagal inhibition of the heart
10.	PM findings	Typical of asphyxia. At the level of obstruction, there is inflammatory reaction with edema of the larynx	No such findings. Because of immediate death, there is no time for development of such findings

F. Traumatic asphyxia

Traumatic asphyxia is a form of asphyxia where exclusion of air from lungs is effected by mechanical fixation of chest in such a way that normal respiratory movements are prevented. Also known as **crush asphyxia**. It is mostly accidental, but may be homicidal and suicidal as well.

1. Causes

a. Accidental

(1) **Riot crush** - When large crowds gather at a small place, and due to some disturbance (eg fire, explosion, electricity failure etc), everyone starts running hither and thither in a completely disorganized way resulting in a stampede. Chest of each individual is pressed violently by the mass of the crowd. Some individuals may fall down, and others may trample on their chests (**human pile deaths**).

(2) **Restraint asphyxia** - When a policeman forcibly restrains a criminal, especially by pinning him on the ground in a prone position, with his hands pinned backwards. The death may also be due to excited delirium syndrome (**ch 11**).

(3) **Falling of earth or stone** – usually in a coal mine or during tunneling or in a building collapse

(4) **During car repair** - Mechanic repairing car positioned underneath a car"Jack slips"Vehicle falls on top of his chest

(5) **Industrial accident** – Machinery falling on victim's chest
(6) **During natural disasters** - earthquakes and landslides
(7) **During railway shunting of carriages** – victim may be pressed between two railway carriages
(8) **Heavy tree** or branch of tree falling on a person sleeping underneath
(9) Some snakes [Boas and pythons] kill by winding around chest, constricting it [**ch 38**].

b. Homicidal

(1) Assailant **jumps on** or **steps upon** the chest of victim
(2) **Jack-knifing** – Victim's thighs and knees are forcibly hyperflexed against his own chest and abdomen, so he can't breathe [indirect compression of chest]
(3) **Traumatic asphyxia by bansdola** – Chest is squeezed forcibly between two sticks placed across front and back of upper part of chest.

c. Suicidal

Victim allows a heavy weight to fall upon himself. Seen mostly in mentally ill.

2. PM appearances

a. External

(1) **Face, lips, scalp** - Swollen, congested.
(2) **Head, neck, upper chest above the level of compression:**
(i) Intense congestion, petechial and confluent hemorrhages and deep purple or purple red cyanosis. This is due to retrograde displacement of blood from the SVC into the subclavian veins and head and neck veins, due to sudden compression of chest. The level of compression is often very clearly marked, below which the skin is pale [**Fig 19.23**] or sometimes mildly cyanosed.
(ii) The spread of blood to the veins of upper limbs is prevented by valves, but it can easily go in the valveless veins of head and neck, where it can rupture distal venules and capillaries"petechial hemorrhages of skin, eyelids, conjunctivae, mucus membranes of mouth. Also bleeding from nose, mouth and ears. May cause "blood blisters" underneath the sclera [due to hemorrhages] which bulge through the eye.
(iii) The purple red color is both due to dilated bv as well as hemorrhages in the tissues around them.

(iv) If patient recovers, the purple color gradually disappears in 10-14 days without the color changes seen in bruises.

(3) **Pallor** - At the level of shirt collar or folds or creases in the garments, braces, buttons etc.

Fig. 19.23: Intense congestion of chest in a case of traumatic asphyxia. This man got crushed in a house collapse. Note the sharp line of demarcation between congested and pale skin on the chest.

b. Internal

(1) **Fractures** - of ribs, sternum, vertebrae, and other bones depending on how the body was crushed.

(2) **Brain** - Congested. Shows petechial hemorrhages

(3) **CVS** - Right side of heart markedly distended. Subendocardial petechial hemorrhages. All veins above aorta are markedly distended.

(4) **Lungs** - Dark, heavy. Show subpleural petechial hemorrhages.

(5) **Internal organs** - Congested.

G. Burking

During the years 1827-8, two Irish immigrants **William Burke** (1792-1829) and **William Hare** (1792-1858) killed **17 persons** by a special method now known as **Burking**, and sold their corpses for dissection to Dr **Robert Knox**, a private

anatomy lecturer, who used to teach students from the Edinburgh Medical School. They used to invite a beggar to their house, give him food and alcohol, and when the victim was drunk, throw him on the ground. Burke used to sit on the chest [traumatic asphyxia] and close victim's nose and mouth with his hands [smothering]. Hare used to pull the victim round the room by the feet.

(1) ***Burking*** is a method of homicidal asphyxia where two asphyxia methods – smothering and traumatic asphyxia – are simultaneously used.

(2) **Other combinations** – are known eg

(i) traumatic asphyxia and strangulation,

(ii) smothering and strangulation etc; but traditionally they are not called burking.

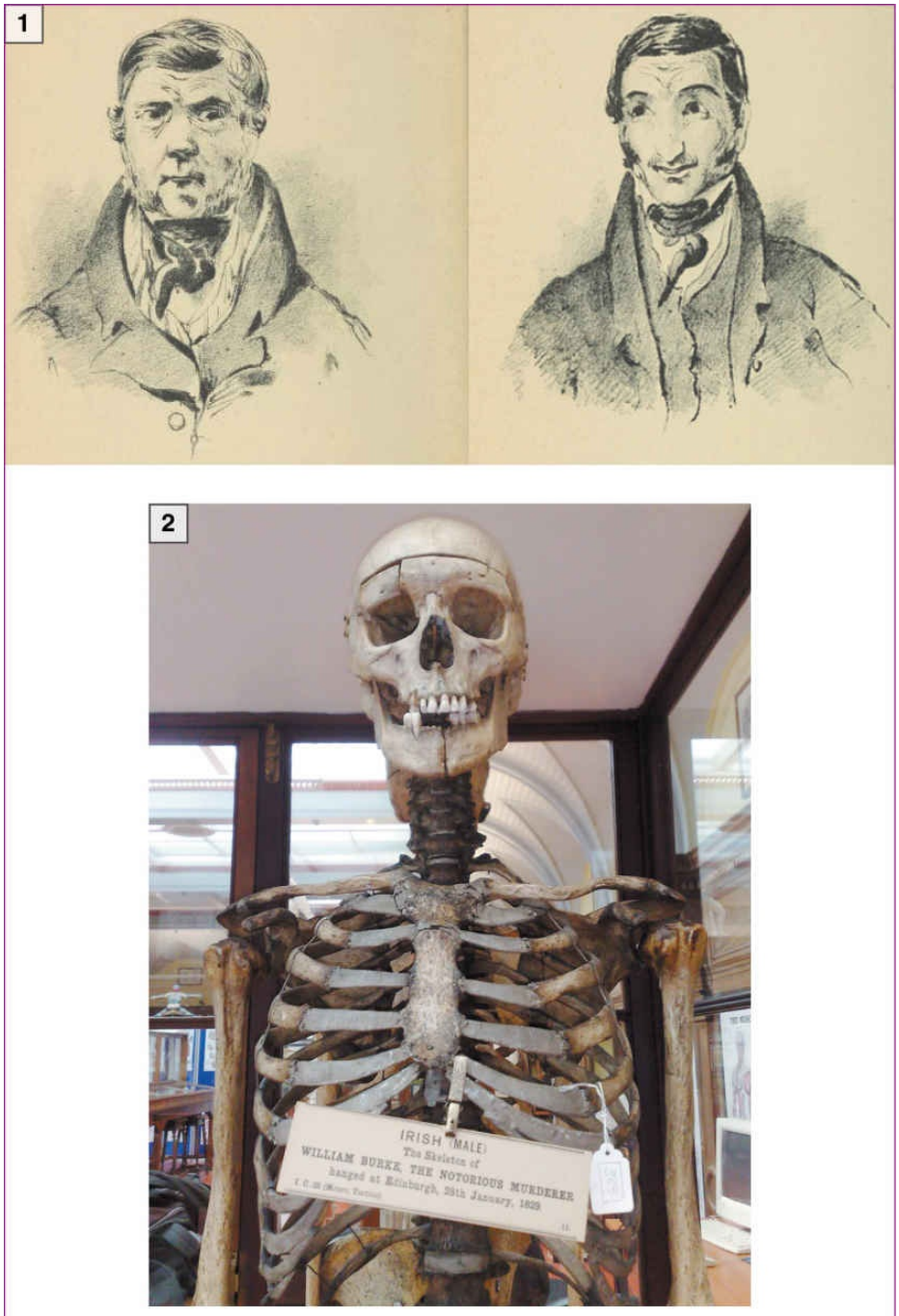


Fig. 19.24: **(1)** Burke and Hare **(2)** Burke's skeleton can be seen to this day in the anatomy department of Edinburgh Medical School. The presiding judge decreed that after the execution, his body should be dissected, and finally his skeleton should be preserved, otherwise subsequent generations may not believe such a person actually existed.

H. Positional Asphyxia

Positional asphyxia [syn, postural asphyxia] is exclusion of air from the lungs

due to abnormal position of the body.

Salient features:

(1) **Causes:** (i) When chest is prevented from expansion by internal pressure - eg. body is placed in such a position that abdominal organs press upon the diaphragm from below upwards. (ii) Falling in confined spaces - eg a well"victim gets wedged between the walls [may be considered a case of wedging also] (iii) Forcible flexion - of neck on the chest. (iv) Indirect compression of chest - Body is in a jack-knife position, i.e. victim's thighs and knees are driven against his own chest. (v) Intoxicated person - Slides out of bed"Head and adjoining region hang down from the edge, and remaining body rests at a relatively upper level.

(2) **Circumstances favoring positional asphyxia** - Victim is incapacitated due to some reason so that he cannot voluntarily make efforts to respire (i) strong intoxication (ii) Unconsciousness due to trauma, stroke or other natural disease.

1. PM findings

Same as that in traumatic asphyxia, except fractures and blunt injuries.

2. MLI

Almost always accidental. But may be homicidal, if assailant "doubles up" victims body in a jack-knife position.

VII. DROWNING

Drowning is a form of asphyxia caused by aspiration of fluid into air-passages, caused by complete or partial submersion in water or other fluid.

Salient features:

(1) Sp gr of body is **1.08**. Other sp gr in increasing order are (i) **Fat**-0.92 (ii) **Brain**-1.04 (iii) **Soft organs**-1.05 (iv) **Muscle**-1.08 (v) **Bone**-2.01. Natural tendency of the body is thus to sink down.

(2) Complete submersion is not necessary; submersion of only nose and mouth can cause drowning, as is seen in alcoholics passing out in shallow streams and getting drowned.

(3) **Current concepts** - According to current thinking the term "drowning" should include both fatal and nonfatal drowning. Accordingly a new definition of drowning has been suggested. It states, "***Drowning*** is the process of

experiencing respiratory impairment from submersion/immersion in liquid." The definition includes cases of drowning in all kinds of liquid, except body fluids [amniotic fluid, milk, saliva, vomitus].

A. Classification

1. Wet drowning

Wet drowning [syn, classical drowning, primary drowning] involves inhalation of water into lungs.

Salient features:

(1) Enormous quantities of liquid is inhaled into lungs

(2) The victim suffers from severe chest pain.

(3) **Fatal period** - Death occurs within minutes of submersion.

(4) **Cause of death** – (i) Cardiac arrest (ii) ventricular fibrillation (iii) Hyperkalemia.

2. Dry drowning

In ***dry drowning***, there is no significant presence of liquid in the lungs.

Salient features:

(1) **Mechanism:** (i) Laryngospasm - Just a few drops of liquids enter the larynx"elicit a violent laryngospasm"death due to asphyxia without entry of water in lungs. (ii) Absorption of liquid in circulation – victim brought out of water"resuscitated"water absorbed in hypertonic plasma.

(2) **Incidence** - About 10-15% of all drownings.

(3) **Victimology** – Commonly seen in (i) children (ii) adults under the influence of alcohol, sedatives or hypnotics.

(4) **Resuscitated victims** display (i) panoramic views of past life (ii) pleasant dreams with no distress.

(5) **PM Appearances** – Diagnosis is mainly of inference and exclusion as no classical signs are there (i) Signs of asphyxia – prominent (ii) Trachea – may be congested. May contain water (iii) lungs – virtually dry. Do not contain water.

3. Near drowning and secondary drowning

a. Near drowning

Near Drowning is defined as initial survival at least beyond 24 hours of an individual after asphyxiation due to submersion in fluid.

Salient features:

It is associated with **secondary complications**, which require further medical management.

b. Secondary drowning

Secondary drowning is ***death*** following near drowning after a period of relative wellbeing.

Salient features:

(1) **Causes** - (i) posthypoxic encephalopathy [most common], (ii) inadequate alveolar gas exchange due to primary alveolar membrane dysfunction and (iii) loss of surfactant.

(2) It can happen after immersion both in fresh and salt water.

(3) **Incidence** - 2-5% of all drownings.

Table 7: Differences between fresh water and sea water drowning

S.No	Criteria	Fresh water drowning	Sea water drowning
1. Lungs			
1.	Size and weight	Ballooned but light	Ballooned, very distended and somewhat heavier than in fresh water drowning
2.	Color	Pale pink	Purplish or bluish
3.	Consistency	More emphysematous	Less emphysematous. Contain more water. Soft and jelly like
4.	Pleural effusions	Generally absent	Generally present
5.	Shape after removal from body	Retained. Do not collapse	Tend to flatten out, perhaps due to greater weight
6.	Sectioning	Releases frothy, often blood tinged fluid. Crepitus is heard on sectioning	Greater quantities of frothy fluid are released. No crepitus on sectioning
7.	Pulmonary surfactant	Pulmonary surfactant is composed of phospholipids 1. phosphatidyl choline (PC) [80% of the total phospholipid], 2. phosphatidyl glycerol (PG) [11%] and 3. Phosphatidyl ethanolamine (PE) and other minor components [9%]. In both fresh water and salt water drowning, pulmonary surfactant is washed away, but PG and PE show greater reduction in fresh water drowning.	PC is reduced more in salt water drowning.
8.	General extent of injury to the lungs	Lesser	Greater because of presence of salt. This causes PC to reduce more severely.
2. Blood			
1.	Ca^{++}, Cl^{-}, Mg^{++} and Na^{+} levels in left heart blood	No increase	Appreciable increase [due to their diffusion from sea water]
2.	General circulation	Hemodilution	Hemoconcentration
3. Cause of death			
1	Cause of death	Ventricular fibrillation, hypoxia of heart muscles, cerebral hypoxia	Pulmonary edema, electrolyte imbalance

4. Immersion syndrome

Immersion syndrome [syn ***hydrocution***, ***submersion inhibition***] is death resulting from cardiac arrest due to vagal inhibition, subsequent to immersion in liquid. **Cardiac arrhythmias**, particularly those associated with the **long-QT syndrome** may be responsible in some cases. Molecular autopsy is required to diagnose such conditions [please see ch 5].

Salient features:

(1) **Incidence** – 1-2% cases of drowning

(2) **Causes:** (i) icy cold water stimulating the nerve endings at the surface of the body (ii) cold water entering ear drums, nasal passages, pharynx and larynx, stimulating nerve endings at mucosal surfaces (iii) Water striking the

epigastrium

(3) **Circumstances:** (i) Diving into the water feet first (ii) Diving with horizontal entry in water [with consequent blow on the abdomen] (iii) ”Duck diving” by the inexperienced [“Duck diving” is a surfing term in which the surfer pushes the surfboard underwater.

(4) **Aggravating factors:** (i) Alcohol -s incidence because of (a) general vasodilatation of skin (b) central effects on vasomotor center (ii) Emotional tension – because nervous reflex circuits are more active [eg during suicidal attempts]. **Imp** – Deprivation of oxygen may be a contributing factor in virtually all types of drowning, especially as the time of immersion lengthens.

B. Causes of Death Associated with Drowning

(1) **Asphyxia:**

(i) Obstruction of airways by fluid. Associated causes are

(ii) Circulatory failure [due to myocardial anoxia] and

(iii) Respiratory failure [due to respiratory center anoxia]. Now it is thought other factors also play a part

(2) **Laryngeal spasm** – due to entry of water into larynx

(3) **Vagal inhibition** – Please see under “immersion syndrome” above

(4) **Injuries** – due to body striking hard objects within water or head striking the base of swimming pool etc. May cause (i) concussion (ii) Fracture dislocation of cervical vertebrae (iii) Fracture of skull

(5) **Cramps** - occurring within water, disallowing victim to swim himself to safety

(6) **Exhaustion** – due to continued struggle

(7) **Long QT syndrome** – rarely

(8) **Ventricular fibrillation**

(9) **Hypoxemia and metabolic acidosis**

(10) **posthypoxic encephalopathy**

(11) **Resetting of hypothalamic thermostat at a lower temperature** – For details of last 4 causes, please see above under “pathophysiology”.

(12) **Electrolyte disturbances** – Rejected now.

C. Fatal Period

Four minutes in **F**resh water and **S**ix to **S**even minutes in **S**ea water [**Memory Aid - 3**].

D. PM Appearances [Wet Drowning]

Autopsy must be started as soon as possible, otherwise classical signs eg foam and froth at mouth and in lungs, overdistension of lungs may become less pronounced or may disappear altogether.

1. External

(1) **Clothing** - Wet. Small aquatic animals may be found trapped.

(2) **Skin** – (A) General - wet, cold, moist, pale [vascular contraction] (B) Cutis anserina [CA] [syn, goose flesh, goose skin] – (i) Appearance – granular and puckered (ii) Mechanism – spasm of **erector pilae** muscles, attached to hair follicles. (iii) Conditions in which cutis anserina is seen (a) during life - when skin comes in contact with cold water [drowning]. In India since water is usually warm CA due to drowning is rarely seen. (b) after death - (I) immediate onset - submersion of body in cold water immediately after death, when muscles are still warm and irritable and have ATP supply intact [agonal contraction] (II) Late onset - by rigor mortis of same muscles [postmortem artifact **ch 10**]. (C) petechial hemorrhages - rarely

(3) **Face** – (a) color – cyanotic (b) conjunctiva – congested; show petechial hemorrhages, especially in lower eyelids (c) pupils – dilated (d) Tongue – swollen and protruded (e) Foam or froth – (i) Time of appearance - Appears within 2 min of drowning (ii) Quantity – varies directly with the length of submersion and violent respiratory efforts (iii) Characteristics - fine, white, lathery foam at mouth and nostrils [one of the most characteristic external signs of drowning; **Fig 19.25**]. (iv) Color - usually white, but may be blood stained [from intrapulmonary bleeding] (v) More commonly seen in sea water drowning (vi) shape - projects as a small balloon or mushroom like mass [champignon de mousse], from mouth and nostrils (vii) On wiping – reappears, especially when chest is compressed (viii) Bubbles - do not readily collapse when touched with the point of a knife (ix) Other conditions where froth is seen [pseudofoam]– (a) electrocution, (b) epilepsy, (c) opium poisoning, (d) pulmonary edema, (e) putrefaction, (f) strangulation. Froth from these conditions can be differentiated from that in drowning by the fact that in drowning froth is finer, much more in volume and quantity and

reappears on wiping. In addition, each condition will have its own specific signs. For example, in putrefaction, the body will be bloated, discolored greenish black and have foul smell. (x) Situations when foam is not seen in drowning – (a) There were no violent respiratory efforts (b) Victim died immediately of immersion syndrome (c) dry drowning, when water could not enter lungs (d) Artificial respiration was performed, especially with respirator (e) Putrefaction - Foam disappears when putrefaction sets in. But putrefaction produces its own characteristic foam [pseudofoam].

Fig. 19.25: Froth in nostrils and mouth in wet drowning. PM lividity is pronounced over face.

(4) **Body and natural orifices** [mouth, nostrils, ears]: coated with algae [including diatoms], grit, mud, sand, seaweed, silt and waterweed.

(5) **PM staining** - (a) Location – (I) Standing water [eg pond]- confined to head

[Fig 19.25], neck and front of chest, lower arms and legs, because the body in water floats face down and buttocks up, with legs and arms hanging down [Fig 19.27] (II) Rapidly moving water [eg river]– PM staining may not develop because of constant change in body's position. (b) color – bright pink [may often be confused with that seen in CO, but CO is not found in blood].

(6) **Rigor mortis** – appears early, especially when the victim struggles violently just before death to save himself.

(7) Signs of **vomiting, micturition, defecation and seminal emissions** – as these phenomena occur agonally.

(8) **Hands** – (a) show cadaveric spasm - Grass, gravel, leaves, moss, sticks, twigs and weeds may be firmly grasped in the hands ["drowning man clutching at the straw"]. It is the surest sign of antemortem drowning. (b) Fingers – abraded (c) Nails – damaged. Show sand, mud and other foreign material under the nails. (d) Washerwoman's hands - (i) Appearance – bleached (white), sodden, thickened and wrinkled (corrugated) [like the soggy hands of a washerwoman]. Changes are seen both in hands and feet [Fig 19.26], but if shoes are worn, it may take twice as long for same changes to appear. ***Washerwoman's skin*** is merely a sign of immersion and is ***not a sign of antemortem drowning***. (ii) Mechanism - absorption of water into outer layers of skin (iii) Progression : Exact calculation of time is not possible, because seen early in warm water. Average durations are (A) **2-4 hours**"wrinkling and soddening [sodden"thoroughly soaked, soggy, heavy] of fingertips (B) **4-8 hours**"bleaching of epidermis (C) **24 hours**"entire hand shows bleached, wrinkled and sodden appearance (D) **48 hours**"Peeling of cuticle from palm and sole (E) **3-4 days**"Skin can be peeled off like a glove

Fig. 19.26: (1) Washerwoman skin over foot (early stage). Note just a bit of wrinkling, especially at the heel (2) and (3) Washerwoman skin seen over fingers.

(9) **Scrotum and penis** – May be retracted due to coming in contact with cold water [etiology similar to that of cutis anserina]. This phenomenon has been called “reaction phenomenon”.

(10) I**njuries:**

(i) Antemortem – (a) before drowning episode - Abrasions and contusions produced before drowning episode [eg during struggle] may get masked due to soggy skin. After skin is allowed to dry, they become visible. (b) during drowning episode – injuries due to striking on sides and bottom of wells, stones at bottom of lakes, rivers etc may be found

(ii) Postmortem – due to (a) damage by underwater flora or fauna [fish bites etc] (b) propeller of boats – May be cut right into half (c) by breakers off shore (d) against uneven surface or rocks at the bottom [**Fig 19.27**]

(11) **Putrefactive changes** – (a) **Early -** appear early after body is brought to the surface; within a few hours after being brought out from water, the body may start showing putrefactive changes. Skin may acquire a green or bronze color or may be exceptionally dark, presenting the appearance of a “negro’s head” [tête de nègre]. (b) **Where not seen** – changes such as greenish black discoloration may not be seen over areas which are tightly wrapped in clothing, shoes etc, or surfaces which are in close contact with each other [eg arm pits, upper limbs, lower limbs if they are tied together].

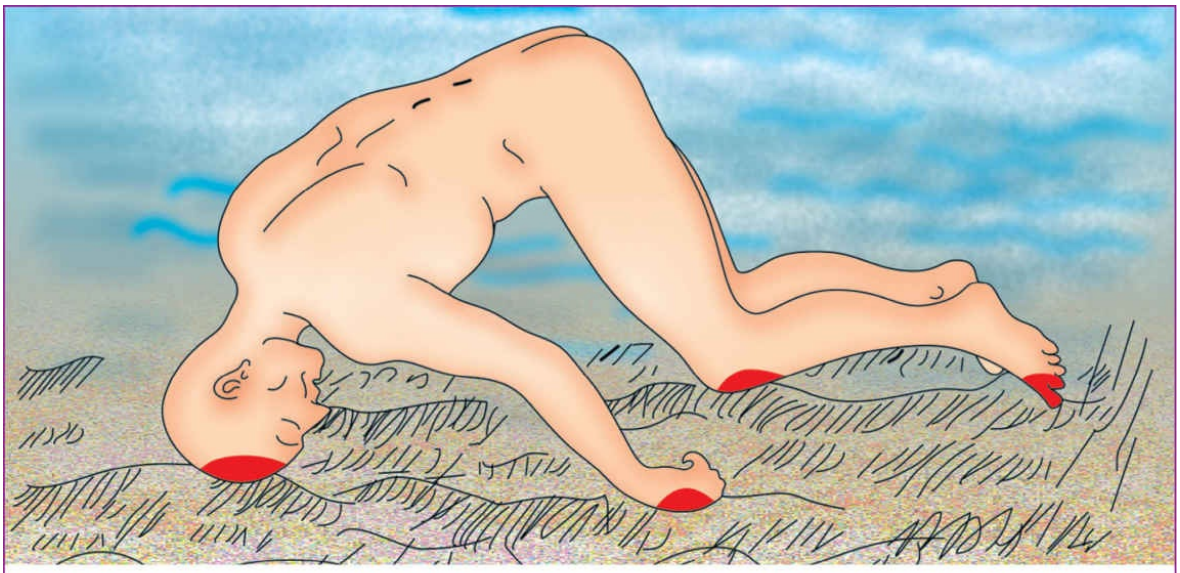


Fig. 19.27: This is how a dead body comes to lie under water. Because of this, postmortem injuries occurs on bony prominences (shown in red) when it scrapes against rough or stony bottom. This is also the reason for peculiar postmortem staining seen in drowning.

2. Internal

a. Air passages

(1) **Trachea and bronchi:**

(i) full of foam, froth [**Fig 19.28**], water

(ii) May contain algae [including diatoms], grit, mud, sand, seaweed, silt and waterweed, which would match with that present in water. Presence in terminal bronchioles is more significant than presence in trachea, which may be due to passive entry even in PM drowning.

(iii) May contain stomach contents – due to vomiting and subsequent aspiration during the unconscious gasping phase of drowning

(2) **Mucosa** – congested.

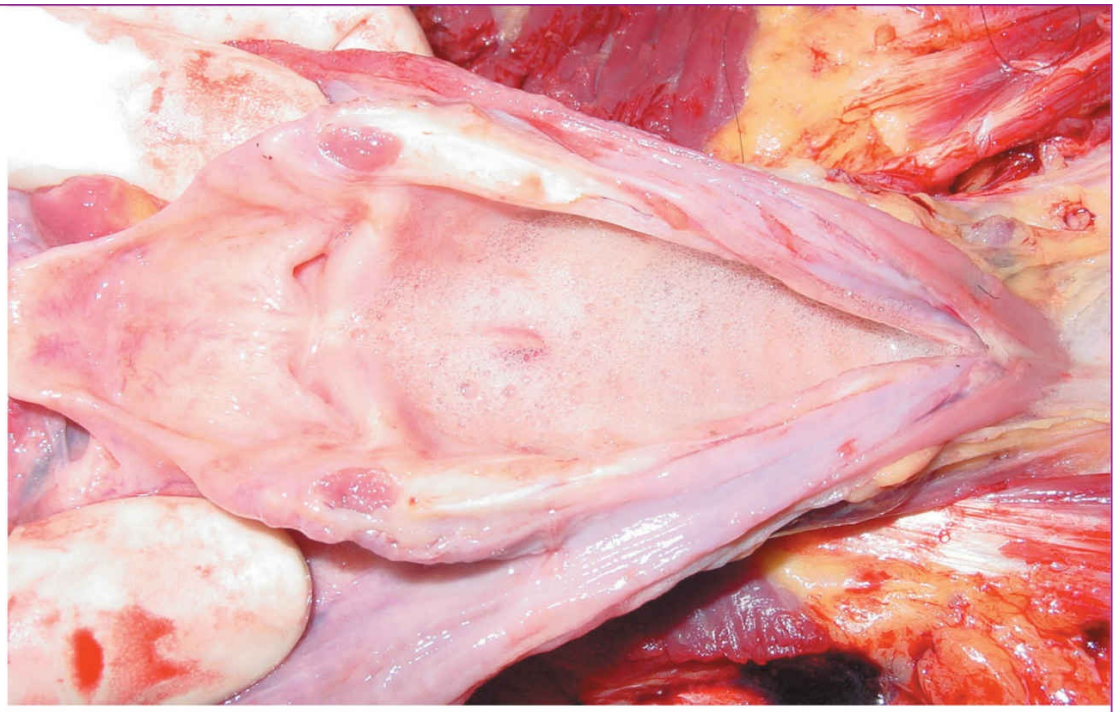


Fig. 19.28 Respiratory tract full of froth.

b. Lungs

(1) **Radiography** – Chest may be radiographed before incision. Alternatively only lungs may be dissected out and radiographed before sectioning.

(i) Pulmonary edema, seen as **Kerley B-lines** [short parallel lines at the lung periphery seen on chest radiographs with interstitial pulmonary edema].

(ii) extravascular lung water

(iii) mud, silt etc can be seen. Silica is radiopaque

(2) Size -

(i) voluminous, overdistended. Medial aspect of each lung approaches the midline.

(ii) may completely cover heart;

(iii) bulge out of chest as soon as sternum is removed [ballooning]. (B) Ballooning is .ed in - (I) pulmonary diseases eg extensive pleural adhesions, fibrosis etc.

(3) Weight – May be up to 1000 g each, especially in salt water drowning [normal – app 400 g (appendix 4)].

(4) Surface – Shows rib markings [grooves]. Have a doughy feel and pit on

pressure

(5) On section – watery, frothy, blood stained fluid oozes out of cut lung surfaces.

(6) **Acute pulmonary edema** – Pathophysiology - (a) - in ambient hydrostatic pressure (b) peripheral vasoconstriction from ambient cold (c) -ed pulmonary blood flow resulting from exercise [struggle] "- pulmonary capillary pressures"extravasation of fluid into the interstitium.

(7) Congestion:

(i) usually moderately congested.

(ii) may be pale. Mechanism of paleness- (I) forcing out of blood from lungs (II) Compression of vessels in the inter-alveolar septa by the air and water trapped in the alveoli.

(8) Paltauf's hemorrhages [syn, Paltauf's spots, signum Paltaufi]-

(i) Appearance - Shining, pale pink or bluish red hemorrhages, immediately beneath the pleurae.

(ii) Size – minute or up to 3-5 cm in diameter

(iii) Incidence – 50%.

(iv) Location – lower lobes of lungs [most common]. May also be on interlobar surfaces or anterior surfaces of entire lungs.

(v) Mechanism – Tearing of the interalveolar partitions due to -ed pressure during forced expirations

(9) Petechial hemorrhages – Absent or extremely rare

(10) Appearances in fresh water and seawater drowning - **Table 7**. The differences in appearances tend to become less clear as body remain in water for longer period

(11) Histologically –

(i) Edema – protein rich edema fluid in interstitial spaces

(ii) Interstitial congestion

(iii) alveoli - (a) Acute dilatation of alveoli with extension, elongation and thinning of the septa (b) alveolar walls are torn. Drowning water penetrates alveolar walls to enter tissues and blood vessels [this is the basis of diatom test]. (c) compression of alveolar capillaries (d) alveolar hemorrhages (e) alveolar macrophages (f) May contain stomach contents [vomiting and subsequent aspiration]

(12) **Hydrostatic lung:**

(i) Seen in PM drowning.

(ii) Due to sheer hydrostatic pressure water enters inside the lung and may simulate true “drowning lung”.

(iii) A dead body immersed for 20 hours at a depth of 2 meters will show “hydrostatic lung”.

(iv) Differentiating feature is absence of froth in trachea, bronchi and at mouth.

c. Heart and great vessels

(1) **Rights side of heart** –

(i) dilated.

(ii) Mechanism - Inhalation of water caused obstruction of pulmonary circulation by obscure mechanisms"back pressure"dilatation of right side of heart.

(iii) Right side of heart and large veins contain dark fluid blood.

(2) **Petechial hemorrhages** - in subepicardial region posteriorly

(3) **Aorta** – intima is stained red.

d. Blood

Fluid - due to dilution by

(i) inhaled fluid and

(ii) release of plasminogen activator from damaged endothelium of pulmonary capillaries.

Table 8: Differences between Emphysema aquosum and Edema aquosum

S.No	Criteria	Emphysema aquosum	Edema aquosum
1.	Condition of victim when went in water	Live and conscious	Live and unconscious
2.	Efforts to breathe	Intense, violent	No efforts
3.	Churning of water within air passages	Yes	No
4.	Creation of foam within air passages	Yes	No
5.	Passive entry of water in lungs	Not excessive, because of constant coughing, spasms of air passages	Excessive because of absent cough reflex. Copious water enters with each inspiration
6.	Appearance of lung	Foamy. Contains excessive froth, but relatively less water	Watery. Contains excessive water, but relatively less, or virtually no froth
7.	Alveolar walls	Ruptured due to violent efforts at breathing	Intact

e. Stomach and intestines

(1) **Gastric mucosa** – soft and heavy

(2) **Water** -

(i) Present in stomach in 3/4th and in intestines in 1/4th of all cases.

(ii) Amount should be measured and examined for foreign substances [naked eye, microscopic]. Also chemically for composition

(iii) Diagnostic value – (a) If chemical and microscopic nature of water is same as that of drowning medium, it could be a great confirmatory evidence of drowning. But it could also be due to victim having drunk the same water before drowning. (b) If the submersion medium was of a filthy or disagreeable nature, eg contaminated with debris, feces or other undesirable material, liquid manure or muddy water, then it is of great diagnostic value, as such water is unlikely to be drunk voluntarily. (c) Water in intestines - indicate peristaltic movements [a vital phenomenon], but this could again be due to antemortem ingestion of water.

(iv) Water is absent – if person died due to immersion syndrome, syncope, shock and in putrefaction.

(3) **Foreign matter** - May contain algae [including diatoms], grit, mud, sand, seaweed, silt and waterweed, which would match with that present in water.

(4) **Sehrt's sign** - Micro-ruptures of gastric mucosa [rupturae parvulae mucosae curvaturae ventriculi minoris] due to overstretching of stomach due to swallowed liquid. Named after German pathologist **Sehrt Ernst** [born 1879] who first described them in **1932**.

(5) **Wydler's sign** - Entire gastric content is collected in a beaker and allowed to stand for an hour. The contents divide into 3 layers — foam on the top, liquid in the middle and a solid component at the bottom [**Fig 19.29**]. First described in **1869** by **Ferdinand Wydler** in his book Zur Diagnose des Ertrinkungstodes.

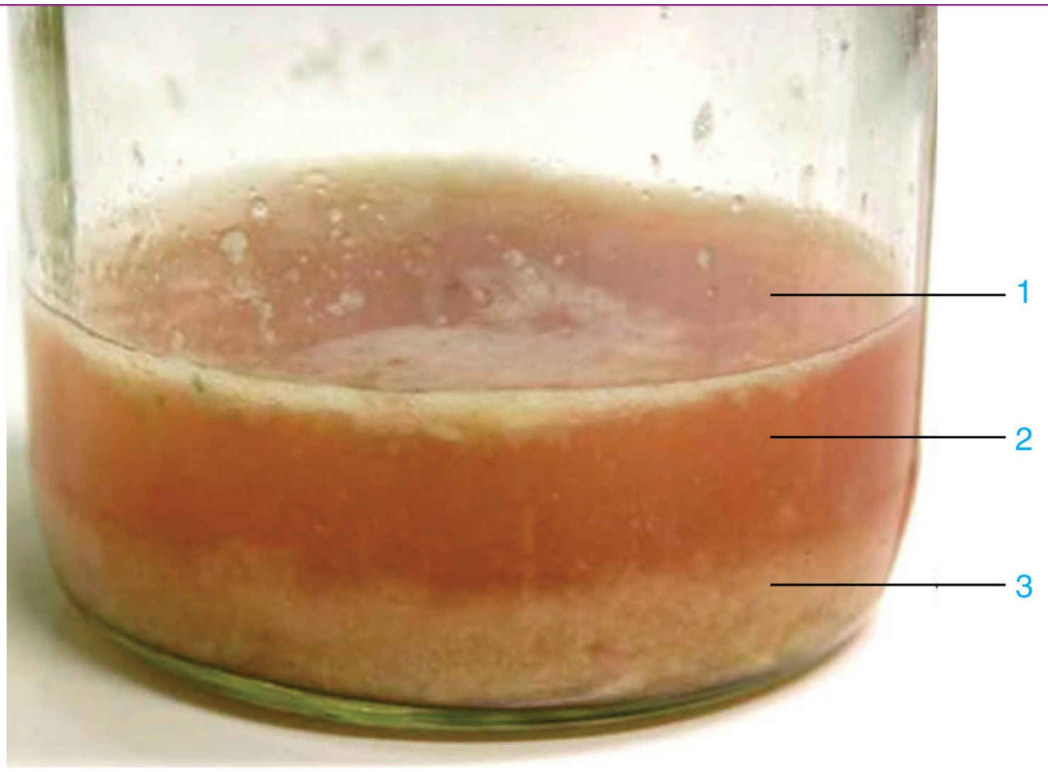


Fig. 19.29: Wydler's sign — 3 basic levels of the gastric content after sedimentation (1) foam on the top, (2) liquid in the middle and (3) a solid component at the bottom.

f. Brain

Swollen with flattening of gyri.

g. Temporal bone and middle ear

(1) **Hemorrhages** - in the **petrous** and **mastoid regions** of the temporal bone. The hemorrhages are localized in the mucosa of the middle ear and mastoid cells. Incidence – 50%.

(2) **Mechanisms:**

(i) - **venous and capillary pressure** - owing to respiratory efforts against a closed glottis

(ii) **penetration into the middle ear** of inhaled liquid via the eustachian tube – due to violent efforts at inhalation

(iii) **barotrauma**, i.e. -ed pressure.

(iv) Conditions in which temporal bone hemorrhages are seen – (a) CO poisoning (b) drug overdose (c) Hanging (d) Strangulation (e) MI (f) Head

injury. Thus they are not pathognomonic of drowning.

h. Paranasal sinuses

There is free liquid in the paranasal sinuses - most commonly in maxillary and sphenoid [**Sveshnikov's Sign**]. Named after Russian pathologist **Sveˇšnikov Vjacˇeslav Alexandrovicˇ** [1918-1988], who first described it in **1965**.

i. Spleen

(1) Small (<0.2% of body weight). Due to sympathetic stimulation with vasoconstriction and contraction of the spleen capsule and trabeculae. Contraction of the spleen during breath-hold apneas and diving has been observed in humans. Victims of drowning tend to hold their breath"splenic contraction.

(2) **Drowning index** [DI] -

$$D.I. = \frac{\text{Weight of both lungs and pleural effusion}}{\text{Weight of spleen}}$$

Values are ≥14.1 in drowning and much below this figure in non-drowning deaths.

(3) **Sabinsky's sign** [splenodystrophia anemica] - Small, anemic spleen seen in cases of mechanical asphyxia, especially in drowning. First described by Russian pathologist **Z J Sabinsky** in **1865**. He directly observed spleen in animals being killed by asphyxia, and found that with the onset of asphyxia, the spleen began to shrink.

j. Muscles

Bruising and rupture – especially of those around shoulder joint [due to violent efforts].

k. Other organs

Congested.

3. Immunohistochemistry

Microtubule-associated protein 2 (MAP2) – .ed expression in cerebral neurons [**ch 5**].

E. Laboratory Tests (of Drowning)

1. Diatoms

Diatoms were first discovered in lungs in a case of drowning by **Hofmann** in **1896**.

a. Description

Diatoms (phylum Bacillariophyta) are microscopic, unicellular, radially or axially symmetrical algae having a unique cell wall made of silica (frustule) (**Fig 19.30**).

(1) **Pigments** - They contain "**chlorophyll-a**" and carotenoid **fucoxanthin**.

(2) **Habitat** - (a) They live in both **fresh water** and **sea water**. (b) They also occur on **moist soil** and **rocks** and in (c) **atmosphere** (d) The vary from place to place, and there is seasonal variation at the same place.

(3) **Size** - ranges from **2µ to 2 mm**. Most species are from 10-80**µ**. During drowning diatoms of <20µ are able to pass through pulmonary circulation into systemic circulation.

(4) **Shape** – Boat shaped, circular, crescentic, linear, oval, rectangular or triangular. Diatom skeletons are readily recognizable as radially or axially symmetrical structures.

(5) **Number** - About 200 genera and 100,000 species are known.

(6) **Resistant to heat and acid** – because of silica walls.

Fig. 19.30: Diatoms as seen under the microscope

b. Principle

(1) **Antemortem drowning** (**Fig 19.31**): Person is respiring"Water enters lungs along with diatoms" violent respiratory efforts cause rupture of alveolar walls"diatoms <20µ pass through pulmonary veins and lymph channels"Reach left ventricle"From here pumped to the entire body"diatoms reach peripheral organs eg, bone, brain, liver, skeletal muscle, spleen etc, and ***can be*** demonstrated there. Also found in body fluids eg bile and urine.

(2) **Postmortem drowning:** Person is **NOT** respiring"Water passively enters lungs along with diatoms"No violent respiratory efforts"**No** rupture of alveolar walls"diatoms found in lungs but **NOT** in peripheral organs. Diatoms ***can not*** be demonstrated in peripheral organs.

(3) **Time of drowning** – Different species of diatoms are found in same body of water in different seasons (summer, winter, rainy season etc).

(4) **Site of drowning** – In a free flowing body of water, different species of

diatoms are found at different places. By examining the species of diatoms, one could opine, where exactly the person was drowned. This new field, where legally relevant information can be derived from diatoms is known as **forensic diatomology**.

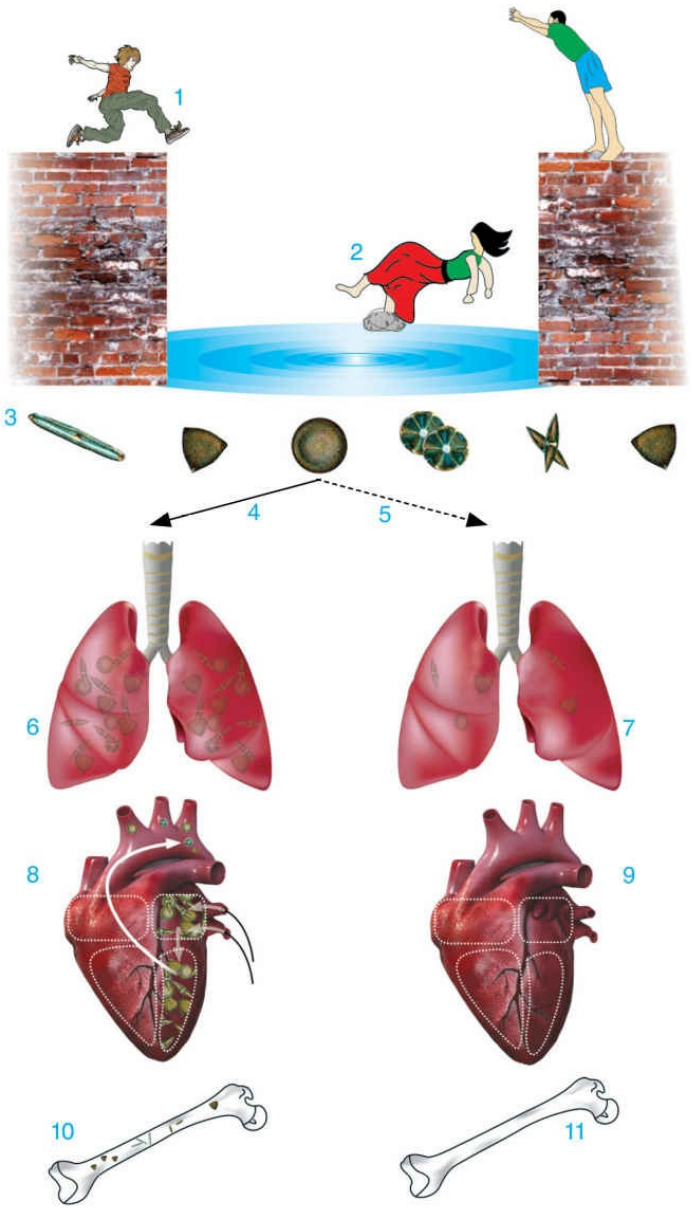


Fig. 19.31: Principle of diatom test. (1) In **live entry** (usually **suicidal**), diatoms within the water (3) enter the lungs along with respiration (4). These diatoms enter the ruptured alveolar walls (6) and are carried to the left side of the heart (8). Since the heart is beating in a live subject, these diatoms are then pumped to all organs of the body (10), where they can be detected. In **postmortem drowning** (always **homicidal**) (2) very few diatoms enter the lungs (5, 7) because the subject is not respiring. Diatoms do not enter the heart (9) and are not carried away to organs of the body (11). Thus they can not be detected in the organs.

c. Controversies

Diatoms may be found in bone marrow and organs due to mechanisms other than

drowning.

(1) **Air borne diatoms** – Diatoms are present in air too, which all persons keep respiring during life. They can enter circulation through pulmonary capillaries

(2) **Ingested diatoms** – Diatoms are present in drinkable water such as from wells and that which comes in tap water. They can enter circulation through frank ulcers or micro abrasions in the GIT.

d. Methods of extraction

(1) **Direct digestion of material:**

(i) With corrosives [HNO_3, H_2SO_4]

(ii) Enzymatic digestion with proteases such as proteinase-K - -es the yield, as the method is less destructive. Even soft bodied algae and protozoa can be recovered.

(iii) strong anionic detergents such as sodium dodecyl sulfate (SDS) and hydrogen peroxide - advantages as in enzymatic digestion

(iv) simply heating soft tissues in a water bath overnight

(2) **Indirect digestion of material** –

(i) incinerate material in electric oven"dissolve the ashes with corrosives as above

(3) **Ultrasonic radiation** - to solubilize tissue

(4) **Direct microscopic examination:**

(i) Lungs – Water is squeezed from lungs"squeezed water centrifuged"sediment examined. Diatoms found in other tissues must be of same species as seen here

(ii) Other tissues – microscopic examination shows optically empty sections.

(5) **Membrane filtering method (blood samples)** – Obsolete now.

e. Reference water samples

(1) About 2000 ml of water sample should be collected from the suspected site of drowning and stored in the refrigerator as a reference sample.

(2) 15 mL of iodine sol^n is added to kill microorganisms

(3) Allowed to settle overnight

(4) Next day most of the top layer of water is poured off. Remainder centrifuged to recover diatoms.

f. Technique

(1) Bone marrow of long bones - is highly reliable and suitable for demonstration of diatoms. Femur and sternum are the most useful and least

contaminated samples for this test; If not available for some reason tibia, humerus, radius, ulna etc can be used.

(2) **Removal of marrow:**

(i) The bone is washed in distilled water

(ii) Periosteum removed

(iii) A piece of rectangular bone is removed with a sharp and clean knife

(iv) Marrow is curetted out from the gutter [**Fig 19.32**]

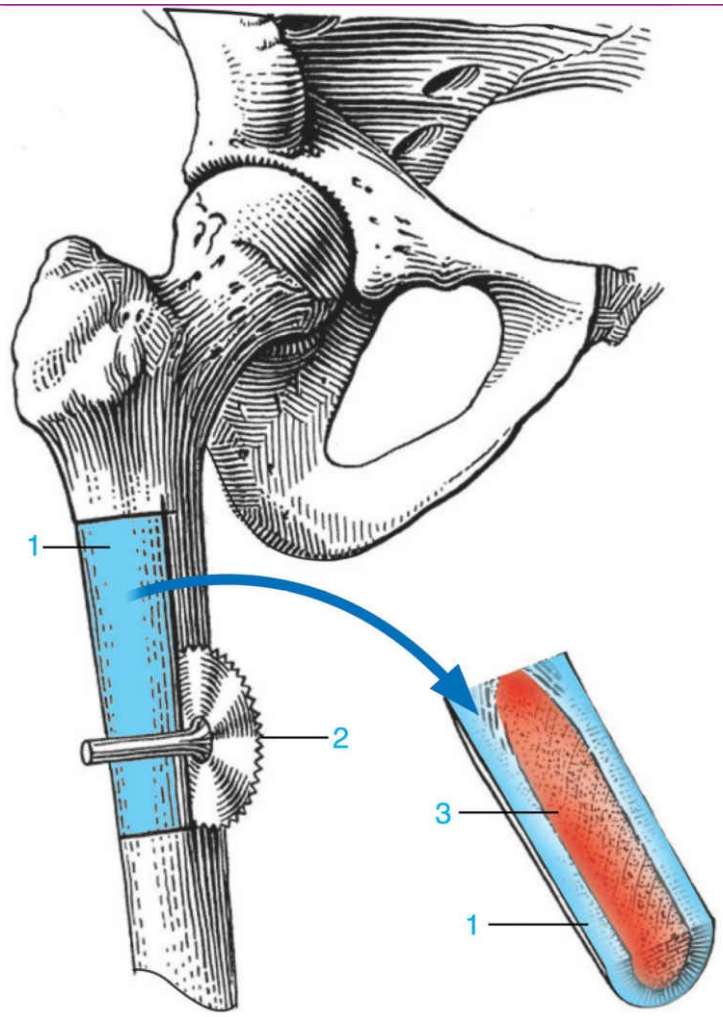


Fig. 19.32: Method of securing a specimen of bone marrow. A segment of bone (1), preferably from femur is removed with a rotating saw (2). The bone marrow (3) is then scooped up from the bone segment to test for diatoms.

(3) **Soft tissues** – brain, kidney, liver are washed and 1x1 cm pieces cut from deeper tissues.

(4) **Digestion:**

(i) 5 g of bone marrow or soft tissue is put in separate test tubes
(ii) Covered with 5 times the volume of Conc HNO_3.
(iii) Left at room temp for 1-2 days
(iv) All organic tissues are dissolved but not diatoms as they have silica shells
(5) **Centrifugation:**
(i) Tube containing digested tissues is centrifuged
(ii) Supernatant acid poured off
(iii) Replaced with distilled water
(iv) The process repeated 2-3 times to dilute the acid
(6) **Examination** – Deposit examined under phase contrast microscope or dark ground illumination. Diatoms examined. If they match those recovered from water, antemortem drowning is established.

g. MLI

(1) **Controversial cases** - Diatom test is not conducted routinely. In controversial cases, when a body is sent for 2nd postmortem examination, it is usual for the 2nd pathologist to conduct diatom test.
(2) Diatom test assumes importance when **only bones** or **isolated parts**, eg a limb is recovered from water. In such cases despite the fact that soft tissues [lungs etc] are not available, death by antemortem drowning can still be determined.
(3) Rarely when entire body is available but PM signs are ambiguous, diatom test is employed.

2. Examination of blood

Principle: Blood tests used in the diagnosis of drowning depend on the fact that if the person was respiring during drowning (antemortem drowning), water will enter lungs, and through ruptured alveolar capillaries reach the left heart. The blood in ***left heart*** will thus reflect several **physical** and **chemical** changes, depending upon the constitution of water (**Fig 19.33**). ***Right heart*** would ***not show*** corresponding changes. **Objections to blood tests**: In the presence of a **patent foramen ovale** (seen in about $1/3^{rd}$ of all people), blood in the two chambers would mix and differences would not be seen.

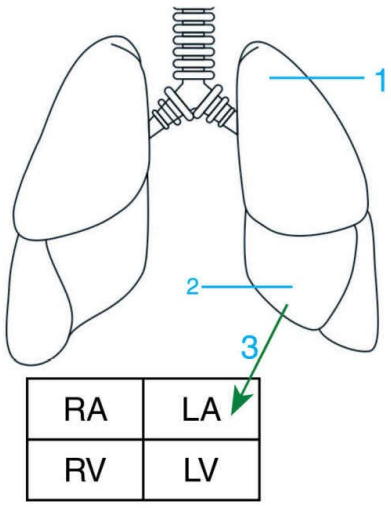


Fig. 19.33: General principle of blood tests used in the diagnosis of drowning. (1) Water enters lungs on submersion (2) If person is alive and making violent efforts at respiration, alveoli would rupture, causing water to enter alveolar capillaries (3) If person is alive, circulation would be active, and water would be transported to the left side of the heart (green arrow). Blood in the left side of the heart would show physico-chemical changes which blood in the right heart would not.

a. Physical tests

(1) **Specific gravity** - In all cases of drowning, irrespective of the salinity, the SG of the left atrial plasma is < SG of the right atrial plasma. Difference of 0·003 and above is significant.

(2) **freezing point** and **electrical conductivity** of blood in the left side of heart would be different from that in the right side, due to hemodilution. First used in 1902 by **Carrara**.

b. Chemical tests

(1) **Chloride estimation (Gettler's test)-** Sea water contains **1940 mg% NaCl**. Human blood contains **340-370 mg%**. Fresh water contains close to 0 mg%. Drowning in **sea water** (having -Cl^- than blood) will -chloride levels in left heart. Drowning in **fresh water** (having .Cl^- than blood) will .chloride levels in left heart. Difference of Cl^- levels in the two sides of heart should be **>20 mg%** to arrive at a diagnosis of drowning. First used in 1921 by **Alexander O. Gettler** (1883-1968), Chief Medical Examiner of New York City.

(2) **Magnesium** - Sea water contains more Mg than in blood. Just like Chloride, Mg^+ also shows same differences. First used in 1944 by **Moritz**.

(3) **Strontium** - Same principle. Strontium levels in sea water are 8000 µg/L compared to that in blood (30 µg/L). Fresh water contains virtually same amount as that in blood. Strontium level - in the left heart in sea water drowning. A difference of >20 µg/L denotes drowning in sea water.

(4) **Other components** - (a) Bromide - The bromide conc of sea water is 65–68 µg/mL, approximately 7500 to 10,000 times higher than that of river water or

blood from normal healthy individuals. Has been used in diagnosis of drowning. (b) Industrial waste – Chemical compounds discharged as industrial waste [oils, hydrocarbons] into waterways may enter lungs during course of drowning and may act as a diagnostic guide.

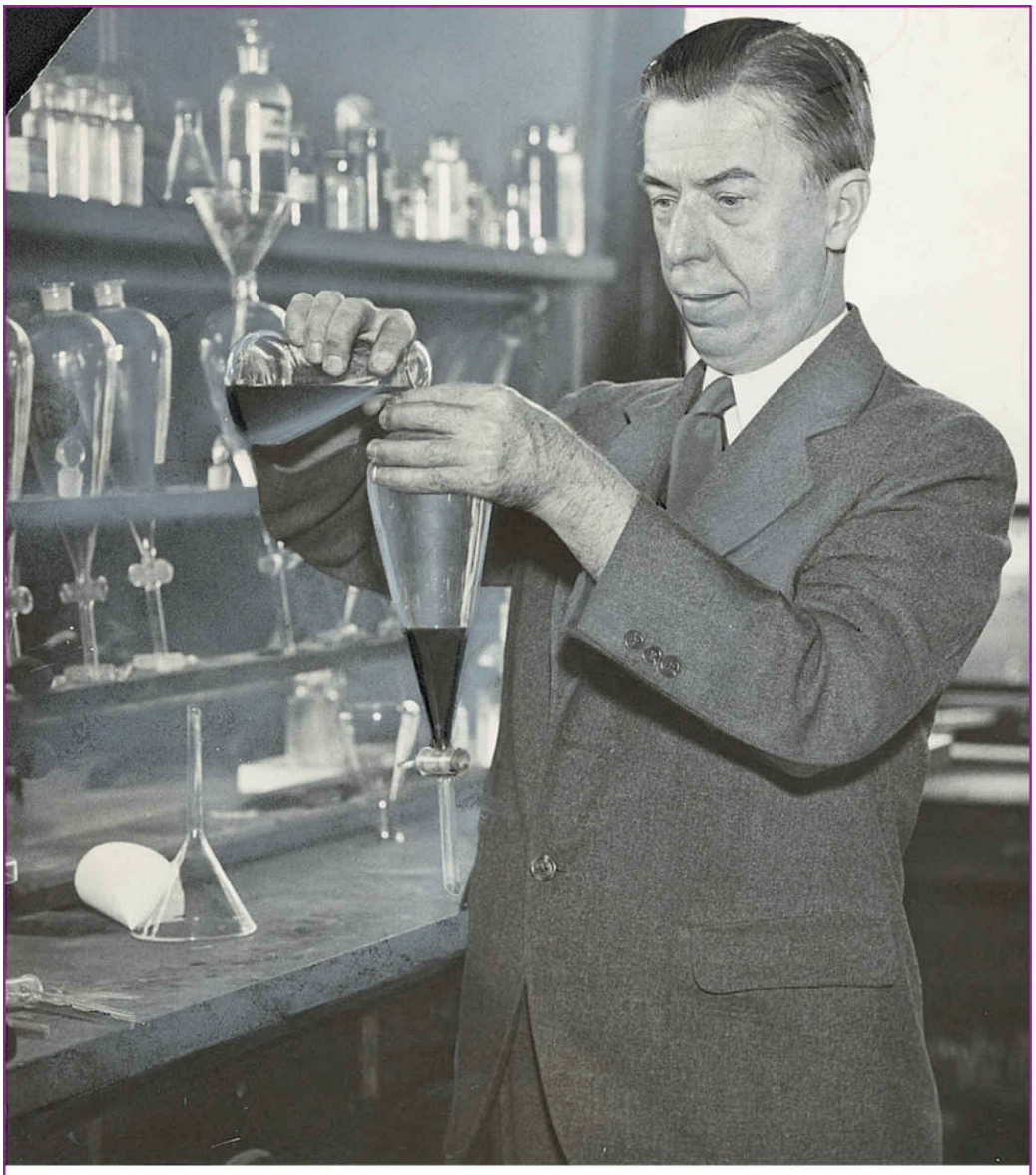


Fig. 19.34: Alexander O. Gettler

F. The Circumstances of Drowning

Manner of death cannot be interpreted from autopsy alone. History, scene of

crime examination, autopsy and toxicology examination all must be pieced together to reach a conclusion.

1. Accidental drowning

Drowning accounts for more accidental deaths in children and adolescents than all other causes except motor vehicle accidents. **Causes** – (1) accidental fall of non-swimmer in water, (2) alcohol or drug use, (3) hypothermia (4) lack of supervision, (5) Natural diseases, eg cardiac arrhythmias, (6) seizures (7) trauma [before or during submersion].

2. Suicidal drowning

(1) **Prevalence** - Common in India, among women, in localities nearby water reservoirs [Canal, Lake, River, Sea]

(2) **Clothed or unclothed:**

(i) Women suicides are generally body fully dressed [Nude female bodies often indicate homicide]

(ii) In a non-swimmer, naked body suggests suicide (iii) Suicides usually remove some of their outer clothing or shoes before leaping into water

(3) **General behavior:**

(i) Multiple suicidal methods – sometimes a person may resort to other suicidal methods [cut throat, poisoning, stab] before drowning himself, either to hasten death, or if the first method failed

(ii) Body found with heavy weights attached to it – homicide or suicide. But in children homicide alone.

(iii) Nature of weights, whether tied by ligature for fixed in clothing or found in the pockets are important

(iv) Sometimes suicides tie their hands or legs together. In such cases manner of tying, knot of rope or ligature should be examined to determine if such knots could be tied by the person himself [***Forensic knot analysis*** - ch 30]. Complicated knots may be within a particular profession only [eg sailor's knot]

(4) **Shallow water:**

(i) Generally drowning in shallow water [from where victim could have easily retrieved himself] indicates homicide.

(ii) Rarely mentally unsound, drunk or drugged persons may accidentally drown in shallow water.

(iii) Still rarely suicides may drown themselves in shallow water; sometimes

even by putting the head in a pail or cistern.

(5) **Case study** - English writer **Virginia Woolf** [1882 –1941] committed suicide on 28 Mar 1941 by drowning. She put on her overcoat, filled its pockets with stones, and walked into the River Ouse near her home and drowned herself. Her body was found 3 weeks later, on 18 Apr 1941.

3. Homicidal drowning

(1) **Homicidal drowning:**

(i) Victim is forcibly pushed under water to drown him.

(ii) Infants and children can be easily pushed under water.

(iii) If the victim is an accomplished swimmer, a heavy weight may be tied to his body.

(iv) An adult may first be made unconscious [eg by hitting him over the head].

(2) **Postmortem submersion** -Person is first killed by some other means [poison, strangulation, head injury etc]"Dead body disposed off in water, in the hope that police would think it was a case of accidental drowning [**Table 9**].

(3) **Drowning in shallow water** - eg ankle deep rivulet strongly suggests homicide, although rarely it may be suicidal [please see above]. If head injury is found, both accidental and homicidal drowning are possible

(i) Accidental - (a) with head injury - the victim slipped"sustained head injury"lost consciousness"drowned in shallow water. (b) with or without head injury - victim under influence of alcohol, drugs, or collapsed from natural disease [MI, epilepsy]"drowned

(ii) Homicidal - (a) Victim’s head forcibly held under water by assailants till he drowned (b) victim was struck on the head homicidally"lost consciousness"dumped in water to drown. For differentiation, other features must be looked for eg signs of struggle [torn clothes, scratches, bruises etc], circumstantial evidence etc.

Table 9: Differences between accidental drowning, suicidal drowning, homicidal drowning and postmortem submersion

S.No	Feature	Accidental drowning (antemortem)	Suicidal drowning (antemortem)	Homicidal drowning (antemortem)	Postmortem submersion
1.	All typical signs of drowning [froth, cadaveric spasm, algae and mud in finer branches of bronchioles, water in lungs etc]	Yes	Yes	Yes	No
2.	Hands tied behind back	No	No	Yes	No
3.	Heavy weight tied to body	No	Extremely rare	More common	Yes. To prevent the body from surfacing due to putrefaction
4.	Intoxicating drugs	Sometimes, as when an intoxicated person falls in water	Rare	common	Common
5.	Gettler test	+ve	+ve	+ve	-ve
6.	Diatom test	+ve	+ve	+ve	-ve
7.	Injuries [please also see "Injuries on drowned persons" – discussed as a separate heading]	Compatible with scene of drowning eg hitting against the walls of a well etc	No injuries. If at all present, they should be compatible with scene of drowning	Injuries on head, if person was rendered unconscious by hitting on head. Marks of strangulation, throttling etc. Multiple bruises are strong indicators	Injuries may be present, if killed by blunt force
8.	Drowning in Shallow water, eg shallow ankle high streams	Possible only if victim was drunk or under the influence of drugs	Virtually never seen	Strongly indicates homicidal drowning, eg when assailant forcibly holds victim's head below water	Virtually never seen
9.	Circumstantial evidence, eg suicide note, signs of struggle, psychological autopsy etc	Findings in favor of accident	Findings in favor of suicide	Findings in favor of homicide	In favor of homicide
10.	Presence of footwear (*chappals etc*) by the side of well	No	Yes	No	no

G. Duration of Submersion

(1) **Temp** - Body heat is lost about twice as rapidly in water than in air

(2) **Putrefactive changes** -

(i) 2 hours"Wrinkling of skin starts

(ii) 12 hours" Bleaching of cuticle

(iii) 24 hours"Bleaching, corrugation and soddening of cuticle becomes very much pronounced

(iv) 48 hours"Cuticle begins to separate from palm of hand and sole of foot

(v) 3-4 days"Cuticle can be peeled off like a glove

(3) **Rate of putrefactive changes:**

(i) twice as slow in water as in air - as long as body remains in water [lesser number of putrefactive microbes in water; no availability of air for putrefactive processes] [**Casper's dictum – ch 9].**

(ii) twice as fast as in air - when body is retrieved and exposed to open air [excessive water soaked in tissues facilitates microbial activity].

(4) **Time of floatation:**

(i) Mechanism - As putrefaction advances, gases of decomposition make the sp gr of body less than of water and it comes up.

(ii) ***Time of floatation*** is the period in which a body [untied with any weights] would come up to the surface of water.

(iii) Time of floatation depends on (a) Salinity of water – In salt water bodies float earlier (b) Sp gr of body – infants and obese persons float earlier [more fat".sp gr] than thin and heavily framed persons

(iv) Position at the time of floatation - (a) Normally – spine uppermost (b) Obese persons and women – face up. Due to fat and gas in breast and abdomen

(v) Place of floatation – (a) generally same as the place of drowning. Body does not move much under water, unless there are strong currents (b) When body comes up and is not discovered immediately, then it may move away considerably.

(vi) Time period in India - floatation occurs in (a) 12-18 h in summer (b) 18-36 h in winters.

(vii) In colder countries - the period is 2-7 days depending on temperature of water.

20. Starvation

I. INTRODUCTION

Starvation is a severe reduction in nutrient, vitamin and energy intake that occurs either from withholding of food or from administration of unsuitable food.

Salient features:

(1) **Types:** (i) **Acute starvation** - withholding of food is sudden and complete. (ii) **Chronic starvation** - withholding of food is gradual. Chronic malnutrition, as occurs in poor, deprived sections of society.

(2) **Inanition** - Symptoms and effects of starvation.

II. CAUSES

A. According to Etiology

1. Circumstantial causes

(1) Poverty [most common cause in India]

(2) Fasting

(3) **Accidental:** (i) earthquakes (ii) famine [failure of crops, overpopulation, war] (iii) landslides (iv) lost in desert or jungle (v) marooned on island (vi) shipwrecks (vii) trapped in mines and pits etc.

2. Medical causes

(1) Ankylosis of jaw (2) Alcohol and drug addicts [partial starvation only. Food is ignored due to overwhelming desire for drug. In alcoholics calories are supplied by alcohol, so no food intake " protein malnutrition] (3) Anorexia nervosa (4) Bulimia nervosa (5) cancer and stricture of esophagus (6) Coma (7) Diabetes mellitus (8) Digestive diseases (9) Mental illness [Major depressive disorder, paranoid schizophrenia, senile dementia].

3. Miscellaneous

(1) Eccentrics may refuse to eat food for no reason.

(2) Ignorance, witchcraft etc – parents either do not provide food, or do not provide food of right kind.

B. According to Manner

1. Accidental starvation

Same as mentioned above [circumstantial causes].

2. Suicidal starvation

(1) **Fasting** - (i) political reasons [fast unto death] (ii) purely exhibition.

(2) **Mentally ill and hysterical persons** - often do not take food.

3. Homicidal starvation

Withholding of food from unwanted children, step children, illegitimate children, child abuse, elderly people, feeble minded, jail inmates etc.

III. SYMPTOMS AND SIGNS

A. Acute Starvation

Starvation is an acute severe form of primary PEU [Protein-Energy Undernutrition].

1. Clinical

a. 30-48 hours

(1) Feeling of hunger

(2) Pain in epigastrium, which is relieved by pressure.

b. 4-5 days

(1) **General:** (i) Temp – subnormal (ii) Voice – weak, whispering (iii) Absorption of s/c fat and emaciation – (a) Cheeks - sunken (b) Eyes – sunken, glistening. Pupils dilated (c) Bony prominences - become visible (d) Chest – ribs prominent, concavities in intercostal spaces, [**Fig 20.1**] supraclavicular fossae sunken (e) Abdomen - concave and scaphoid [boat shaped] [**Fig 20.1**] (f) Limbs – thin, flaccid, loss of muscular power (g) Muscular weakness – progressive, severe (h) Loss of wt – Marked. (iv) Odor – offensive, especially towards death.

(2) **Dermatologic:** (i) Skin - dry, inelastic, pigmented, rough, thin, wrinkled. Shows follicular hyperkeratosis and trophic skin changes (ii) Hair and nails – brittle, dry, lusterless, hair loss.

(3) **CVS:** (i) Cardiac insufficiency (ii) Pulse – slow at rest; paroxysmal tachycardia on exertion

(4) **GIT:** (i) Lips – cracked, dry (ii) Tongue – coated, dirty (iii) Thirst – intolerable (iv) Saliva – Scanty, thick (v) Motility - Initially constipation. Diarrhea and dysentery towards death.

(5) **Genitourinary:** Urine – acidosis, highly concentrated, scanty, turbid

(6) **Intellect:** (i) Generally clear till death (ii) In some cases – delusions and hallucinations of sight and hearing.

c. >5 days

(1) **1st stage** - Initially rapid mobilization of protein stores " converted by liver to glucose to maintain energy supply to brain [which can utilize only glucose]

(2) **2nd stage** – . in the utilization of proteins; shift to fat metabolism

(3) **3rd stage** – As complete depletion of fat stores occur, metabolism is again shifted to proteins.

Fig. 20.1: Death due to starvation. Note the general emaciation, especially sunken eyes and cheeks

2. Lab investigations

Same as in chronic starvation below.

B. Chronic Starvation

1. Clinical

Changes occur in a constant order (1) . ed resistance to infection " diarrhea, dysentery, tuberculosis (2) - ing cachexia (3) Anemia (4) Edema – begins first in lower limbs and feet (5) Fatigue, lethargy [mental and physical] (6) Hypotension, peripheral vascular stasis (7) Loss of hunger, hunger pains, well-being (8) Loss of wt – progressive; rapid in first 6 months (9) Mental faculties – gross mental retardation, loss of self-respect (10) Pigmentation (11) Polyuria (12) Temp ..

2. Lab investigations

(1) **Blood:**

(i) Alkaline phosphatase, Angiotensin Converting Enzyme [ACE], calcium, chlorides, cholesterol, glucose, iron binding capacity [transferrin levels], Mg, phosphate, Proteins, Sugar, T_3 - .

(ii) Ketones [acetone], NPN, urea - -

(2) **Urine** – Ketones present, pH ..

IV. CAUSE OF DEATH

(1) **Causes of death** are: (i) Circulatory failure due to brown atrophy of heart (ii) Exhaustion (iii) Intercurrent infections

(2) **Contributing factors** are: (i) Dehydration (ii) Hypothermia.

V. FATAL PERIOD

(1) **In number of days:**

(i) Both water and food are completely withdrawn " 10 days [both adults and newborns]

(ii) Only food withdrawn " 60 days

(iii) Only water withdrawn " 10 days [happens rarely when a camper is lost in desert, has good food supply but no water].

(2) **By % loss of body reserves** – Loss of

(i) 20% protein (ii) 40% total body wt and (iii) 80% fat is fatal.

A. Factors Influencing the Fatal Period

All following conditions [**Table 1**] can be understood by the fact that - ed fat reserves and . ed expenditure mean more survival and vice versa.

Table 1: Factors influencing fatal period in starvation

S.No	Criteria	Withstand less	Withstand more
1.	Age	very young, very old	Fatty, muscular
2.	Sex	Males	Females
3.	Condition of body	Lean, thin, emaciated	Fatty, healthy
4.	Temp	Excessive cold or hot environment	Normal environment
5.	Physical exertion	More	Less

VI. PM APPEARANCES

(1) **General:**

(i) Ascites [in 10% cases]

(ii) Edema - under the eyelids, cheeks and chin, inside the thigh and around ankles

(iii) loss of s/c fat and emaciation – extreme. Gives rise to (a) sunken eyes and cheeks, (b) bony prominences [eg clavicles, tips of shoulders, ribs, anterior superior iliac spine] are more prominent. (c) Chest - ribs prominent, concavities in intercostal spaces, supraclavicular fossae sunken (d) Abdomen - concave and scaphoid (e) Limbs – thin, flaccid (f) Wt - . ed more than 40%

(iv) Intercurrent diseases, superadded infections due to loss of vitality (e) Odor – offensive, especially towards death

(2) **Rigor mortis** – sets in and disappears early

(3) **Face** – pale

(4) **Dermatological:**

(i) Skin – dry, inelastic, pigmented, rough, thin, wrinkled. Shows follicular hyperkeratosis and trophic skin changes

(ii) hair and nails– brittle, dry, lusterless

(5) **Fat:**

(i) Complete absent from around internal organs, and in mesentery, omentum, and s/c tissues. This is never seen in wasting diseases eg cachexia and tuberculosis.

(ii) disappears late from – fat of female breast, around orbit

(iii) subepicardial fat – replaced by watery gelatinous material

(6) **Muscular system:**

(i) Muscles - atrophied and dark due to - in lipochrome

(ii) Fibres – (a) lose striations, (b) become more uniform due to granular degeneration

(7) **Skeletal system:**

(i) Children - (a) Dental defects [eg caries, decalcification] (b) Rickets (c) Spinal curvature

(ii) Adults - (a) Osteomalacia

(iii) in both – (a) Demineralization of bone (b) stress fractures

(8) **Viscera** [**general**]:

(i) Great . in size and wt of all organs, except brain [changes similar to those in senility]

(9) **Heart:**

(i) small [due to brown atrophy]

(ii) chambers empty

(10) **Lungs:** (i) collapsed (ii) edematous (iii) pale (iv) on cut section – exude very little blood, show hypostatic basal congestion

(11) **GIT:** (i) Lips – cracked, dry (ii) Tongue – coated, dirty (iii) stomach and intestines show atrophy of all layers (iv) intestines – (a) walls appear like tissue paper, very friable to touch and break easily (b) mucosa – shows extensive non-specific ulceration as seen in ulcerative colitis (c) contents – offensive watery fluid and gas (v) mucosa – stained with bile

(12) **Liver:** (i) atrophied (ii) shows necrosis due to protein deficiency

(13) **Gall bladder** – distended with bile [no food in duodenum " no cholecystokinin " no contraction of GB].

(14) **Spleen** – shrunken

(15) **Renal system:** (i) kidneys – atrophied (ii) urinary bladder – empty [cf gall bladder].

(16) **Brain** – wt retained [unlike other organs], pale, soft

(17) **Blood:** (i) Volume – marked reduced (ii) anemia – hypochromic microcytic

(18) **Special investigations** – Blood and urine findings same as given in ch starvation above.

VII. D/D AT AUTOPSY

(1) **Diseases which cause loss of wt** - Addison's disease, AIDS, anorexia nervosa, chronic diarrhea, diabetes mellitus, food intolerance, hyperthyroidism, malabsorption syndromes, malignancy, pernicious anemia, progressive muscular atrophy, pyloric stenosis and tuberculosis.

Differentiation - (a) In none of the above diseases, marked absence of fat [especially around orbits and from female breast] as in starvation would be noted. (b) Specific findings of each disease would be evident

(2) Sometimes disease is both the cause and effect, setting up a vicious cycle, eg TB " malnutrition " accentuation of TB. Primary cause of death may be given as that which shows more marked features.

VIII. MEDICOLEGAL ASPECTS

Hunger strikes – Medicolegal issues arise when a political prisoner goes on a hunger strike and the jailor requests the jail doctor to force-feed the prisoner in order to save his life. (i) **The current legal situation in India** appears ambiguous according to **IPC** (a) **Force feeding** - doctor may be sued for using criminal force **u/s 350, 352, IPC**. (b) **No force feeding** - doctor may be sued for abetment of suicide **(u/s 306, IPC)**. (ii) **Indian constitution – Article 21** ensures right to life; there is no equivalent right to die. Since Indian Constitution is supreme, force feeding of hunger-strikers is **lawful in India**. (iii) Criteria for force feeding – appearance of ketones in urine (iv) **World Medical Association Guidelines** – The situation is reverse. **WMA** has given guidelines to doctors in its **Declaration of Tokyo** (adopted in 1975; amended in 2005) and **Declaration of Malta** (adopted in 1991; amended in 1992). Both ***respect the autonomy of the***

patient, and suggest that the ***doctor should NOT force feed*** in such cases. Feeding is allowed, when patient has lapsed into coma.

21. Deaths Associated with Anaesthesia and Surgery

I. FATAL BLOOD CONCENTRATIONS

(1) Chloroform - Pl see **ch 40**
(2) Diethyl ether - 180 mg%
(3) Divinyl ether - 50 mg%
(4) Ethyl chloride - 40 mg%
(5) Halothane - 20 mg%
(6) Trichloroethylene - 50 mg%

Memory Aid 1: Fatal blood concentrations of anesthetics

It is useful to remember fatal blood concentrations of anesthetics in relation to that of alcohol, which is **500 mg%.** Now remember following **2 mnemonics**:

1. The following mnemonic outlines **3 imp anesthetics** which have **1/10th** of this concentration, i.e. **50 mg%**.

Three can form diving ethics

Three can = **trichloroethylene**

Can form = **chloroform**

Diving ethics = **divinyl ether**

2. **Hell = halothane**. Hell should be worst. So it has lowest fatal concentration [**20 mg%**], i.e. 1/25th of that of alcohol.

II. CAUSES OF DEATHS ASSOCIATED WITH ANAESTHESIA AND SURGERY

Anesthesia may be general, or of some specified type. The causes of general anesthetic and operative deaths are (**Table 1**):

Table 1: Causes of General Anesthetic and operative deaths

1. Deaths associated with **anesthesia**
2. Deaths associated with **surgical operation** (a) Surgeon going through the sternum, inadvertently
3. Deaths by **diseases** (a) Those for which Anesthesia was given or operation performed: (i) **Emergency operation -** A highly serious condition for which a last-ditch attempt was made through surgery, e.g. explosive injuries, with extreme fragmentation. (ii) **High risk operations –** e.g. Coronary bypass surgery, Heart transplant operation (b) Other than those for which anesthesia was given or peration performed: (i) Hypertension, IHD, lung disease

A. Deaths Associated with Anesthesia

(1) **Inexperience** – Many deaths occur due to inexperience and failure to adopt precautions when clearly indicated

(i) **Inadequate depth of anesthesia** – Intubation and bronchoscopy may cause vagal inhibition if depth of anesthesia is inadequate

(ii) **Inadequate ventilation** – Hypoxia, sudden death due to heart failure

(iii) **Post-operative respiratory obstructions** – by tubing or swabs.

(iv) Breathing circuit disconnections

(v) Between staff

(vi) **Haste**

(vii) **Distraction**

(viii) **Excessive pressure on airway** – rupture of lungs

(ix) Positive pressure ventilation – converts a simple pneumothorax into tension pneumothorax

(x) **Intubation** of esophagus instead of trachea

(2) **Clinical factors** -

(i) Underventilation

(ii) Low blood volume

(iii) Inadequate transfusion

(iv) Anoxia (causes cerebral damage)

(v) Inadvertent hypothermia

(vi) Hyperpyrexia

(vii) Inhalation of regurgitated material (vomit), especially during the post-operative period

(3) **Technical mishaps** -

(i) Administration of **incompatible blood**

(ii) Infusion of **wrong drug or fluid** (a) sodium citrate for normal saline. They are both colorless and have virtually similar labels. To avoid such errors, use distinctive labels and keep in separate cupboards (b) Anesthetic of greater strength than required (c) ether injected in error for a local anesthetic. Label should be checked as well as odor to prevent such incidents (d) Administration of adrenaline in error for cocaine

(iii) **equipment failure**

(iv) **Inadvertent inhalation** - of gauze and other swabs, dentures

(v) **Mislabeling** of Oxygen and anesthetic gases

(vi) **Explosions and fires** – Ignition of inflammable anesthetic vapor by an electrical spark. Most dangerous inflammable mixtures are cyclopropane-oxygen and ether-oxygen. The spark may arise from a faulty electrical appliance, X-ray apparatus, static electricity or from a surgical diathermy electrode.

Common adverse reactions associated with anesthetic agents

Malignant hyperpyrexia (Malignant Hyperthermia)

Malignant hyperpyrexia, ***malignant hyperthermia*** (MH) or malignant hyperthermia syndrome (MHS) is a rare life-threatening condition triggered by exposure to certain general anesthetics (specifically all volatile anesthetics), nearly all gas anesthetics, and the neuromuscular blocking agent succinylcholine.

III. INVESTIGATION OF DEATHS ASSOCIATED WITH

ANAESTHESIA AND SURGERY

A. Preliminary Investigations

1. Visit to the operation room

Check all equipment, preferably in consultation with an independent anesthetist from a different hospital. Check all valves and containers to ensure that there was correct mixing of percentages of gases.

2. History

(1) Obtain **history of exposure** to relevant and potentially toxic chemicals during (a) the period prior to hospitalization(b) hospital stay (c) Preanesthetic preparation (d) Anesthesia

(2) **Obtain list** – of relevant and potentially toxic chemicals associated with each of these periods

(3) Thorough review of hospital chart

(4) Hold discussions with the surgical and anesthetic team.

3. Existing diseases

Some surgical conditions are high risk per se eg resection of the aortic aneurysm and repair [surgeon may not be able to control bleeding]

4. Anesthesia

(1) Get information about anesthetic agents used

(2) Check if method of administration was correct

(3) Inadvertent wrong mixing of anesthetic agents may have occurred.

(4) Note duration of time the patient remained under anesthesia. Was it excessive?

5. Equipment

(1) All equipment including containers, valves etc must be checked in association with appropriate qualified personnel.

(2) Check is correct mixing of gases was ensured.

B. Autopsy

(1) **Examine all devices *in situ:***

(i) Devices - attached to and inserted into the body must not be removed, either by the operating surgeon or nurse before sending body for postmortem. Common indwelling devices encountered are airways, chest tubes,

indwelling catheters and needles, intravenous cannulae and wound drains.

(ii) Autopsy pathologist - should open cavities with devices in situ, to know where the other end was reaching [eg intubation device may be in the esophagus, thoracic drain pipe may be puncturing lung etc]. All devices must be checked for patency also.

(iii) Fluids in each cavity - Note presence, smell and volume etc. Preserve for analysis

(2) **Examination of operation site** – Must be thoroughly examined, although at times it may be difficult because of surgical alterations of the anatomy as well as because of the presence of hemorrhage, adhesions, sepsis and edema etc.

(3) Look out for **artifactual** findings - Sutures from stomach and intestine may appear to have broken down. It could however be an autolytic change

(4) **Surgical errors** -

(i) Ligation of a wrong vessel, eg ligation of coronary artery, while implanting a heart valve prosthesis.

(ii) Inadvertent ligation of ureter, bile duct

(iii) Perforation of large bv

(iv) Inadvertent removal of a vital organ

(5) **Brain:**

(i) Hippocampal gyrus and cerebellum - show hypoxic changes. The **Ammon's horns** [syn, Cornu Ammonis, CA, hippocampus] are particularly vulnerable, especially the **Sommer sector** [region CA1] and to a slightly less extent, the endfolium. The selective necrosis in the Sommer sector may be identifiable macroscopically if the patient survives for more than a few days.

(ii) Diffuse severe leukoencephalopathy of cerebral hemispheres with sparing of immediate subcortical connecting fibres

(iii) Demyelination and obliteration of axon

(iv) Infarction of basal ganglia

(v) Damage is limited to white matter.

Samples to collect

(1) Blood - For grouping, cross matching etc. Collect blood of deceased, transfused blood, empty and half empty blood bags etc

(2) For culture - exudates, pus

(3) Samples from all organs - Histopathology

(4) **For toxicology** - Both lungs must be tied and submitted for toxicological examination [**ch 5**]. Other viscera as in standard toxicology death.

(i) Alveolar air - collection procedure as given in ch 5.

(ii) Gases from cavities, heart and blood vessels - Fill body cavity with water"use a rubber dam to trap gases before cutting the organ.

IV. LEGAL ASPECTS OF ANESTHESIA

A. Legal Responsibilities of an Anesthetist

Role during illegal surgery:

(1) It is the duty of anesthetist to refuse anesthesia during illegal surgery [eg illegal MTP, organ retrieval, amputation of healthy limbs for making beggars etc].

(2) He cannot claim he did not know the nature of surgery because during preanesthetic check-up he must have discovered it.

(3) He must bring it to the notice of legal authorities.

(4) If he administers anesthesia during illegal surgery he is a party to crime.

22. Deaths Due to Cold and Heat

I. COLD

Hypothermia is a condition in which core (rectal) temperature drops below 35.0 °C (95.0 °F). This is the minimum temperature required for normal metabolism and body functions.

A. Stages of Hypothermia

B. Cause of Death

(1) **Failure of vital centers** - due to anoxia
(2) **Cessation of heart function** – due to atria1 and ventricular fibrillation.

Table 1: Stages of hypothermia and associated major effects on human body

Temp in Celsius	Temp in Fahrenheit	Major effects
35°C	95°F	Hypothermia
32°C	90°F	Shivering stops
30°C	86°F	Hypothalamus loses ability to regulate temperature. Body becomes poikilothermic
26°C	79°F	Death ensues (if temperature maintained for 24 hours)

C. Local Effects

Local effects are divided into two groups -

(1) those that occur **without any freezing** of the body tissues, e.g. **chilblains (pernio)** and **trench foot (immersion foot),** and
(2) those that result from the **freezing of the skin or a body part** (generally the fingers, toes, ears, nose and cheeks).

1. With tissue freezing

Frostbite

Frostbite (congelatio) is a cold-related injury characterized by freezing of tissues.

Salient features:

(1) It occurs at temperatures **below 0°C**.
(2) **Mechanism:** (i) Ice crystals form in the skin and deeper tissues. (ii) These

exert **osmotic force**, causing water to move from intracellular spaces. (iii) This in turn, causes **swelling of tissues** (filling up of intercellular spaces with fluid). (iv) At the same time there is cellular dehydration, hyperosmolarity, denaturation of proteins and destruction of enzymes.

(3) **Signs and Symptoms:** Skin - numb and discolored; it is **purple** in most severe cases. **Blisters** appear, which may become hemorrhagic. On touching the skin, it does not feel like normal "bouncy" skin; instead it feels just like a **rock, block of ice** or like a **piece of chicken from the freezer.**

2. Without tissue freezing

a. Trench foot (immersion foot)

Trench foot is a condition caused by prolonged exposure of the feet to damp, unsanitary and cold conditions.

Salient features:

(1) The word "trench" refers to trench warfare, used in World War I, where many soldiers suffered from this condition.

(2) **Predisposing factors:** (i) Temperature: **5-8°C.** (ii) Dampness

(3) **Signs and symptoms:** (i) In uncomplicated cases, there is no permanent injury. (ii) The affected feet become **numb** (iii) turn blue **(cyanosis)** or red **(erythrosis)** and (iv) may **swell.** (v) Advanced condition involves **blisters** and **open sores**, which lead to **fungal infections**; this is sometimes called **tropical ulcer [jungle rot]** (vi) If left untreated, trench foot usually results in gangrene, which may require amputation.

b. Chilblains

Also known as **pernio** and **perniosis**. There is **redness** and **inflammation** of skin. ***Acral ulcers*** (ulcers affecting digits and toes) form. These are due to constriction of capillary beds in the skin.

Table 2: Characteristics of cold injury

Hypothermic condition	Characteristics
Frostbite	Ice crystals form in tissues. Tissue feels like a block of ice. Hemorrhagic blisters appear.
Frostnip	Numbness and tingling. Skin is white or waxy
Trench foot	Swelling of feet. Feet blue or red. Sores leading to fungal infections. Entire foot affected.
Chilblains	Swelling not seen. Redness and inflammation. Acral ulcers form. Only fingers and toes affected.

D. Causes of Hypothermia

LPG - Liquefied petroleum gas [syn, autogas, LPG] is now a commonly used commercial vehicle fuel [it is different from CNG]. It consists of a mixture of propane [C_3H_8] and butane [C_4H_{10}] which when moderately compressed turns into a liquid at atmospheric temperature and then reverts back to a gas when the pressure is sufficiently reduced. It is cheaper and more environmental friendly than petrol or diesel. Motor vehicles can now be purchased to run on LPG or, more commonly, they are converted from running on petrol or diesel. Its operating temperature is extremely low. The boiling point is between -42° and 0°C, depending on the hydrocarbon mix. Owing to its rapid vaporization and consequent lowering of temperature it can cause severe cold injuries if brought into contact with the naked skin. Several cases of cold injury following skin contact with LPG at filling stations have been described.' Mostly the accident occurs due to vapor leak between the gun nozzle and fuel tank.

E. PM Appearances

1. External

(1) **Skin:**

(i) PM staining is reddish. Mechanism - OxyHb cannot give up O_2 because of excessive cold [The oxygen dissociation curve of Hb shifts to the left side at low temperatures] " Diffuse through skin " Causes reddish color

(ii) Even non dependent areas show pink, brown pink or violet patches with indistinct blurred margins, especially over and around joints [eg knees, elbows, hips]. Mechanism " These are hyperemic areas, which develop just prior to death as the skin is pressed against snow or cold ground. Mental confusion leads to uncoordinated movements eg crawling.

(iii) In some cases, skin may be completely white [“white death”]

(iv) Sometimes spots of bluish discoloration are seen on hands, elbows, knees and feet [represent minor frostbite lesions].

(v) Histology – edema and hyperemia of dermis, foci of inflammatory cell infiltration

(2) **Generalized edema** - Swelling of ears, hands noticeable. Mechanism - Cold injury " capillaries are damaged " leaking of plasma into the tissue

(3) **Cold stiffening** [syn cold rigor]:

(i) Body fluids, fats and tissues are frozen, solidified and stiff due to extreme cold, giving rise to a stiffened posture.

(ii) May be confused with rigor mortis

(iii) Differences from rigor – **Table 3**.

(iv) Freezing with subsequent hardening of s/c fat, especially in infants sometimes makes the skin folds rigid, which may be mistaken for ligature mark [pseudoligature mark - **ch 19**].

Table 3: Differences between rigor mortis and cold stiffening

S.No	Feature	Cold stiffening	Rigor mortis
1.	Temp	Body icy to touch	Body may be warm to cold, but not icy
2.	Suppleness	Body is stony hard due to complete freezing of fats and tissues	Body still has a fleshy and "bouncy" feeling
3.	Forcible flexing of joints	A crackling sound heard as particles of ice break within frozen synovial fluid in joints	No such crackling is heart
4.	Allow some time to pass	As body thaws, stiffness disappears and body becomes supple. Normal rigor mortis [RM] then starts developing if further time passes. Body becomes stiff again, but this time because of RM	Depending on the stage of rigor mortis, either it becomes stronger, or starts disappearing. Once body becomes supple, it would not become stiff again

2. Internal

(1) **Blood:** (i) Bright red (ii) Intravascular hemolysis [seen after freezing of blood]

(2) **Skull** – ice inside skull may cause separation of skull sutures.
(3) **Brain** – Cytotoxic cerebral edema [**ch 17**].
(4) **Heart:**
(i) Ice crystals present [also in bv and interstitial tissue spaces].
(ii) Right atrium and ventricle dilated.
(5) **Lungs:**
(i) Congested
(ii) Intra-alveolar, interstitial, and intrabronchial hemorrhages
(iii) Bronchopneumonia
(6) **Stomach:**
(i) Mucosa studded with numerous brown-black acute erosions.
(ii) Similar to those seen in stress.
(iii) First described by Wischnewski in **1895** [known as **Wischnewski erosions**, **points**, **spots** or **ulcers.**].
(iv) Mechanism - (a) stress (b) -ed tissue amines, histamine and serotonin. (c) - in capillary permeability (d) impairment of the blood flow in the GIT mucosa (e) . in O_2 dissociation.
(7) **Pancreas:**
(i) Fat necrosis [most constant finding]
(ii) Aseptic pancreatitis
(iii) Pancreatic hemorrhage.
(8) **Adrenals** – Lipid depletion from the adrenal cortex.

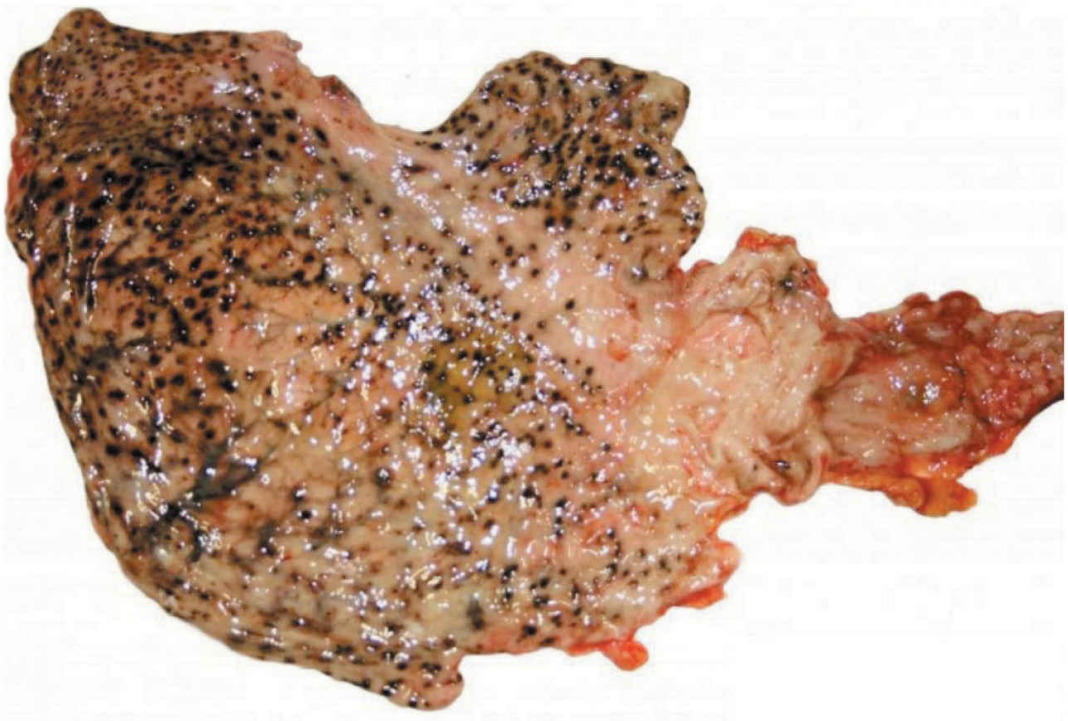


Fig. 22.1: Wischnewski spots.

3. PM chemistry

(1) **Blood** - Glucose -ed

(2) **Urine** - - ed catecholamine [adrenalin, noradrenalin], histamine and serotonin [quite valuable in diagnosing hypothermia]; - ed glucose

(3) **Vitreous:**

(i) - glucose [in nondiabetics].

(ii) -CO_2. Glucose conc and total CO_2 content vary inversely with temp.

F. ML Significance

1. Paradoxical undressing

(1) Def - Refers to a cold-exposed hypothermic person taking off his clothes just before death. He is found dead in an extremely cold environment, but paradoxically he is naked, with clothes strewn around.

(2) The phenomenon is rare

(3) It is a terminal event

(4) More common in old

(5) **Hide and die syndrome** - This may be seen sometimes. Characteristics are

(a) Person may hide himself in corners, in cupboards, under piles, furniture or other household goods (b) Naked or seminaked person is found amid a greatly disturbed scene, with furniture pulled over and drawers and cupboards emptied out. All disturbance is however at lower levels only. Tops of tables are not disturbed. May lead a police officer to believe a criminal act has taken place. Visit to scene of crime by an experienced pathologist is necessary.

(6) Appearance of the body implies a criminal act, especially rape, but no signs of foul play are there.

(7) Mechanism – (a) Hypothermia " (I) . sympathetic activity of the skin, and (II) Paralysis of the thermal regulatory mechanism " dilation of cutaneous bv " - skin temperature " spurious feeling of warmth "Undressing. (b) Terminal mental confusion, delirium and hallucinations.

II. HEAT

1. Heat edema

Heat edema [syn, Colombo flop, Deck ankles] is a very mild form of heat illness which appears as dependent soft tissue swelling, usually in the lower extremities, in a person lacking acclimatization.

2. Heat rash

Heat rash [syn lichen tropics, miliaria rubra, prickly heat] is another very mild form of heat disorder characterized by rash on anterior surface of elbows, posterior surface of knees, sternum, clavicle, waist, axillae. Palms and soles are not affected.

3. Heat cramps

Heat cramps [syn, Cane cutter’s cramps, Fireman’s cramps, Miner’s cramps, Stoker’s cramps] are severe muscle spasms resulting from a combination of prolonged exercise, heavy sweating, and excessive water replacement in extreme heat.

4. Heat syncope

Heat syncope [syn, heat collapse] is a condition resulting from intense peripheral vasodilatation leading to peripheral pooling and hypotension.

5. Heat exhaustion

Heat exhaustion [syn, heat prostration] is a condition resulting from severe dehydration after a huge amount of sweat has been lost.

6. Heatstroke

Heatstroke [syn, Coup de soleil (French, lit. stroke of the sun), heat hyperpyrexia, siriasis, sunstroke, thermic fever] is the most serious heat disorder characterized by a complete breakdown of thermoregulatory mechanism, complete loss of sweating and temperature rising >41°C [106°F].

Salient features:

(1) Terms “sunstroke” and “thermic fever” are more often used when there has been a direct exposure to the sun

(2) **Pathophysiology** - Heat " renal and splanchnic vasoconstriction with concomitant peripheral vasodilatation " shunting blood to the periphery [to dissipate heat] " If exposure to heat continues, the vasoconstriction needed to keep the blood in the periphery fails " cutaneous blood flow . es " less heat dissipation " hyperthermia " cerebral edema " ed ICP " .ed cerebral blood flow " CNS dysfunction. Subarachnoid hemorrhages in some cases.

(3) **Factors aggravating heatstroke:** (i) absence of a full acclimatization (ii) alcoholism (iii) drugs [major tranquillizers] (iv) dysentery with resulting dehydration (v) heavy and impervious clothing (vi) humid atmosphere [at 100% humidity, even a relatively low temp of 32°C may lead to heatstroke] (vii) infections (viii) muscular activity (ix) no breeze (x) obesity (xi) old age (xii) toxic pyrexia [recent or present]

(4) **Signs and symptoms** – onset is sudden with sudden collapse and loss of consciousness. (i) Prodromal symptoms - Cramps, dizziness, excessive thirst, faintness, giddiness, headache, general weakness, lack of energy, mental confusion, nausea & vomiting, purposeless movements, restlessness, staggering gait (ii) Skin – dry, hot, flushed, cessation of all sweating (iii) Temp – 42°C [108°F] is common; 45°C [113°F] has been recorded. When temp rises > 42°C, vasodilatation occurs with . in blood volume " circulator collapse " cardiac failure (iv) Respiration – rapid [>30/min], deep, Kussmaul type (v) CVS – (a) BP . (b) Tachycardia; pulse >130/min. Becomes irregular later (vi) CNS deficit – due to - ed temp (a) bizarre behavior (b) cerebellar dysfunction (c) Clouding of senses (d) convulsions [generalized or Jacksonian] (e) decerebrate rigidity (f) delirium (g) Eyes - fixed and dilated pupils, oculogyric crisis (h) hallucinations (i) muscular twitching (j) opisthotonus (k) speech difficulties (l) coma.

(5) **Prognosis** – bad; mortality is very high.

(6) **Diagnosis** - Based on classical triad of (i) - ed temperature (ii) dry skin (iii) neurological deficit

(7) **Differential diagnosis:** (i) Falciparum malaria (ii) Datura poisoning

(8) **Management:** (i) Move individual to a cooler place (ii) loosen clothes or undress (iii) allow air to circulate around the patient (iv) wrap individual in wet towels or clothing (v) Place ice packs in areas with the greatest blood supply [neck, under the arm and knees, groin] (vi) monitor body temp constantly to guard against overcooling. If hyperpyrexia continues for > 4 hours, prognosis is bad. Even if the condition is not fatal, the patient may have permanent neurological deficit. (vii) Monitor HR and RR (viii) IV fluids and electrolytes (ix) Iced gastric lavage (x) Anti-convulsant [if convulsions] (xi) After initial treatment, bed rest may be recommended for several days.

(9) **Fatal period** – 5 min-3 days

a. PM appearances [of heatstroke]

Not specific.

(1) **Temp** – remains high after death. PM caloricity may be seen.

(2) **CNS:**

(i) Brain – (a) congested and edematous. (b) Petechial hemorrhages seen in white matter (c) cerebral hemispheres - (I) show - in weight, (II) flattening of convolutions (III) Histopathology – [1] Chromatolytic changes [2] Degeneration of neurons [least in globus pallidus] [3] Microglia – show diffuse proliferation [4] Pyknotic nuclei [5] Swollen dendrites (d) Scattered petechiae all over the brain, especially in the walls of 3rd ventricle and floor of 4th ventricle. (e) Cerebellum – shows most striking and consistent changes. (I) Purkinje layer – [1] shows edema [2] Purkinje cells – show disintegration, swelling and reduction in number. [3] If survival is >24 h " complete degeneration of Purkinje layer and gliosis. (II) Rarefaction of granule cell layer (f) Hypothalamus – edema of nuclei

(ii) Meninges - congested.

(3) **Heart:**

(i) Dilatation of right auricle

(ii) Flabbiness of muscles

(iii) Myocardial degeneration

(iv) Petechial or confluent subepicardial and subendocardial petechial

hemorrhages

(4) **RS:**

(i) Trachea and bronchi – contain frothy hemorrhagic fluid

(ii) Lungs – Congestion, Edema, hemorrhages, lobular pneumonia [if survival is more]

(5) **Liver:**

(i) Centrilobular necrosis

(ii) Congestion

(6) **Kidneys:**

(i) Short survival - (a) -ed wt (b) Congestion (c) Edema

(ii) Longer survival - (a) Hemoglobinuric nephrosis (b) Tubular necrosis

(7) **Adrenals:**

(i) Cortical degeneration

(ii) Adrenal and pericapsular hemorrhages

(iii) Sinusoids – engorged

(8) **Bone marrow:** Degeneration of megakaryocytes [if survival >24h]

(9) **General:**

(i) Visceral hemorrhages - Mechanisms (a) - ed temperature " hepatic damage " prothrombin depletion (b) thrombocytopenia and . plasma fibrinogen (c) -ed capillary fragility due to anoxia

(ii) Disseminated intravascular coagulation.

23. Impotence and Sterility

I. INTRODUCTION

A. Impotence

(1) ***Impotence*** is inability of a person to perform sexual intercourse. Impotence in male is better termed as **Erectile Dysfunction (ED)**.

(2) Potentia copulandi – Power to copulate.

B. Sterility

(1) ***Sterility*** is inability to beget or conceive children (in the male and female respectively).

(2) **Infertility** is a preferred term. It is used in relation to a couple unable to beget children, and is defined as failure to conceive following ***twelve months*** of unprotected and regular intercourse.

(3) A person can be impotent without being sterile or vice-versa. Both conditions may co-exist too.

II. CAUSES OF IMPOTENCE IN THE MALE

A. Psychogenic

1. Generalized

(1) Age related decline in sexual arousability

(2) Generalized unresponsiveness

(3) Primary lack of sexual arousability

(4) Generalized inhibition

(5) Chronic disorder of sexual intimacy.

2. Situational

a. Partner related

(i) Lack of arousability in a specific relationship (ii) High central inhibition owing to partner conflict or threat (iii) Lack of arousability owing to sexual object preference (**impotence quoad hoc**)- Person is impotent towards one particular woman (say wife), but not towards the rest. This may lead to medicolegal problems. “Quoad hoc“ is a Latin phrase meaning “As to this” or “with respect to this”. Thus “impotent quoad hoc” literally means “impotence with respect to”.

b. Performance related

(1) Associated with other sexual dysfunction/s (eg, rapid ejaculation)

(2) Situational performance anxiety (eg, fear of failure).

c. Psychologic distress related or adjustment related

(1) Associated with negative mood state (eg, depression)

(2) Major life stress (eg, death of partner)

B. Organic

Memory Aid 1: Organic causes of impotence

I = Inflammatory (eg, Prostatitis)
M = Mechanical (eg, Peyronie's disease)
P = Postsurgical (eg, Radical prostatectomy)
O = Occlusive (eg, Vascular Atherosclerosis)
T = Traumatic (eg, Pelvic fracture)
E = Endurance factors (eg, Chronic renal failure)
N = Neurogenic (eg, Multiple sclerosis)
C = Chemicals (eg, Drug induced - antihypertensive drugs)
E = Endocrine (eg, Diabetes mellitus)

For differences in emasculation and castration and effects of castration before and after puberty, please see **ch 11**"Grievous hurt"Emasculation.

III. CAUSES OF IMPOTENCE IN THE FEMALE

[A] Impotence in the female is better termed as **Female sexual dysfunction [FSD]**. The American Foundation of Urologic Disease (AFUD) Consensus Panel in 2000, classified female sexual dysfunction in following 4 groups (1) **Sexual desire disorders** - (i) Hypoactive sexual desire disorder (HSDD)- [syn ***frigidity***] Absence of desire for sexual activity (ii) Sexual aversion disorder - Phobic aversion to and avoidance of sexual contact with a sexual partner (2) **Sexual arousal disorder** - Inability to attain sufficient sexual excitement. (3) **Orgasmic disorder** - No orgasm following sufficient sexual stimulation and arousal (4) **Sexual pain disorders:** (i) Dyspareunia - Persistent genital pain associated with sexual intercourse. (ii) Vaginismus - Persistent involuntary spasm of the musculature of the outer third of the vagina that interferes with vaginal penetration. (iii) Other sexual pain disorders (non-coital) - Genital pain induced by non-coital sexual stimulation. In some of the above cases [eg orgasmic disorders, dyspareunia], intercourse is physically possible; thus would not amount to **legal impotence**. Husband cannot sue for nullity if wife suffers from such conditions [Please also co-relate with male sexual dysfunction above]. [B] **Etiology of FSD** – (1) Abnormalities of genitals – eg vaginal atresia (2) cancers (3) Gynecologic problems (4) Long-term medical diseases (5) medications (6)

psychosocial difficulties, eg vaginismus (7) systemic ailments.

IV. CAUSES OF STERILITY IN THE MALE

(1) **Congenital** -
(i) Anorchia
(ii) Azoospermia and related conditions [**Table 1**] [could be acquired too].
(iii) Cryptorchidism
(iv) Congenital Absence of Vas Deferens
(v) Genetic abnormalities - (a) Karyotype anomalies including Klinefelter syndrome (b) Y chromosome microdeletions (c) Kallmann syndrome (d) mutations in genes involved in Hypothalamus–pituitary–gonadal axis (e) Partial/Mild Androgen Insensitivity syndrome)

Table 1: Nomenclature related to pathological semen quality according to WHO (2010)

S.No	Term	Criteria (Spermatozoa)
1.	Oligozoospermia	$<15x10^6$/ml [range 12-16]
2.	Asthenozoospermia	<32% motile
3.	Teratozoospermia	<4% morphologically normal
4.	Oligo-astheno-teratozoospermia	Disturbance of all three parameters
5.	Azoospermia	No spermatozoa in the ejaculate
6.	Cryptozoospermia	Spermatozoa absent from fresh preparation but observed in a centrifuged pellet
7.	Aspermia	No ejaculate
8.	Leucospermia (leucocytospermia)	$>1x10^6$ ml leucocytes in the ejaculate

(2) **Acquired** -
(i) Acquired hypogonadotrophic hypogonadism or endocrine factors
(ii) Erectile, ejaculatory dysfunction
(iii) Exogenous factors [medications, cytotoxic drugs, irradiation, heat]
(iv) Obstruction, subobstruction of proximal and/or distal urogenital tract
(v) Post-inflammatory forms [orchitis, epididymitis]
(vi) Recurrent urogenital infections [prostatitis, prostatovesiculitis]
(vii) Surgeries that can damage vascularization of the testes
(viii) Systemic diseases [liver cirrhosis, renal failure]
(ix) Testis trauma
(3) **Idiopathic** -Unknown etiology [about 50%].

Quantity of seminal fluid in a single emission is **1.5 -6.8 ml** [**ch 23**]**.** Of this **seminal vesicles** contribute most [70%]; **prostate** [25%]; **testes** [4%], and **bulbourethral glands** [1%].

Memory Aid 2: Quantity of semen

Some Professionals **Test Bulb**

Some – Seminal Vesicles

Professionals – Prostate

Test – Testes

Bulb – Bulbourethral glands

How much contributed

Seminal vesicles – **s**eventy %

Prostate – ***P***acchis [Hindi for 25] %

Test – 4 letters, so 4% by testes

Bulb – Bulbourethral glands – Last letter "l" looks like 1, so 1% by Bulbourethral

V. CAUSES OF STERILITY IN THE FEMALE

(1) **General** -

(i) Extremes of age

(ii) Tobacco smoking

(iii) Sexually transmitted disease

(iv) Body weight and eating disorders

(v) Chemotherapy - Alkylating drugs [busulfan, chlorambucil, chlormethine, cyclophosphamide, ifosfamide, elphalan, procarbazine]

(vi) Other general factors – Cannabis, Significant liver or kidney disease, Thrombophilia (hypercoagulability or a prothrombotic state)

(2) **Hypothalamic-pituitary:**

(i) Hypothalamic dysfunction

(ii) Hyperprolactinemia

(3) **Ovarian** –

(i) Anovulation [anovulatory infertility]

(ii) .ed ovarian reserve [number of eggs that can be successfully recruited for a possible pregnancy are .ed]

(iii) Gonadal dysgenesis [Turner syndrome]

(iv) Luteal dysfunction

(v) Ovarian cancer
(vi) Polycystic ovary syndrome
(vii) Premature menopause
(4) **Tubal/peritoneal** – (i) Endometriosis (ii) Pelvic adhesions (iii) PID, [usually due to chlamydia] (iv) Tubal dysfunction (v) Tubal occlusion
(5) **Uterine** - (i) Asherman's Syndrome (ii) Uterine fibroids [leiomyoma] (iii) Uterine malformations
(6) **Cervical** - (i) Antisperm antibodies (ii) Cervical stenosis (iii) Non-receptive cervical mucus
(7) **Vaginal** - (i) Vaginismus (ii) Vaginal obstruction
(8) **Genetic** -
(i) Intersex conditions [eg androgen insensitivity syndrome]
(ii) Mayer-Rokitansky-Küster-Hauser Syndrome [MRKH] - Primary amenorrhea and an underdeveloped uterus. Incidence - 1 in 5,000 females.
(iii) Turner's Syndrome.

VI. MEDICOLEGAL ASPECTS OF IMPOTENCE AND STERILITY

A. Law Relating to Marriage in India

1. Hindu Marriage Act, 1955 [HMA]

a. Conditions of a valid hindu marriage

According to **S.5** of the ***Hindu Marriage Act, 1955***, (**HMA**) a total of **five** conditions must be fulfilled for a proper legal Hindu marriage (i) **No living spouse** - At the time of marriage, neither party must have a living spouse (ii) **No mental illness** - At the time of marriage (a) neither party must be incapable of giving a valid consent to it due to unsoundness of mind (b) though capable of giving a valid consent, neither party must be suffering from a mental disorder of such a kind and degree, so as to be unfit for marriage and procreation of children (c) neither party must be subject to recurrent attacks of insanity. Earlier, there was a 4^{th} condition – epilepsy – also, but it has been repealed w.e.f. 29 Dec 1999. Currently if either party, or both are epileptic, it is a valid and legal marriage [**ch 28 –** Civil responsibilities of the mentally ill]. (iii) **Age** - The bridegroom must be above 21 years of age and bride of 18 years of age. (iv) **No**

prohibited relationship - Both parties must not be within degrees of prohibited relationship (eg, father and daughter; mother and son; brother and sister; uncle and niece; aunt and nephew), unless custom allows (v) **No *Sapindas*** - Both parties must not be sapindas [a Sanskrit term meaning "people connected by particles of one body"] of each other, unless custom allows [Sapinda relationship extends as far as the third generation (inclusive) in the line of ascent of mother, and fifth (inclusive) in the line of ascent through the father]. **Table 2** summarizes various conditions of a valid Hindu marriage and what happens when they are not met with.

Table 2: Five conditions of a valid Hindu marriage under the HMA, 1955 and consequences if they are not met with

S.No	Section of HMA, 1955	Condition	Result if condition not met with	Punishment/fine
1.	5(i)	Neither party must have a living spouse	Marriage is void u/s 11 HMA	(1) Bigamy [S.17, HMA] (2) Punishment 7 y + fine, if person having spouse told other person about it [S.494, IPC] (3) Punishment 10 y + fine, if person having spouse concealed information from other person about it [S.495, IPC]
2.	5(ii)	Mental illness	Marriage is voidable u/s 12(b), HMA	None
3.	5(iii)	Boy>21 y; Girl>18 y.	Voidable [S.3(1). The Prohibition of Child Marriage Act, 2006]	(1) 2 y rigorous imprisonment [RI] and/or one lakh Rs fine to a person so marrying [S18(a) HMA]. [NB. Before 2007, the punishment was 15 days simple imprisonment and $ 1000 fine] (2) 2 y [RI] and one lakh Rs fine to a person who performs, conducts, directs or abets any such marriage [S.10 The Prohibition of Child Marriage Act, 2006] – applicable to pujaris etc (3) parents or guardians of such children are also punished as above. But female parent or guardian can not be imprisoned [S.11, The Prohibition of Child Marriage Act, 2006]
4.	5(iv)	Degrees of Prohibited relationship	Marriage is void u/s 11, HMA	1 month simple imprisonment and/or $ 1000 fine [S18(b) HMA]
5.	5(v)	Sapindas	Marriage is void u/s 11, HMA	1 month simple imprisonment and/or $ 1000 fine [S18(b) HMA]

b. End of marriage

Under HMA, m**arriage can end in one of 3 ways** -(i) **Null and void** (ii) **voidable** (iii) **divorce**.

i. Null and void

(1) **Rationale behind null and void marriages** – Marriages are so morally degrading that their continuance may be damaging to the society fabric. Thus they are declared null and void ab initio (**S.11 HMA**), ie. they are never

deemed to have taken place legally

(2) **When null and void** - if conditions (i) , (iv) and (v) above are not met with.

(3) So-called "wife" does not have any right to property, nor is she entitled to alimony [support money given after divorce].

ii. Voidable

(1) **Rationale behind voidable marriages** - Law understands that under certain conditions, one partner may have difficulties living with the other. But the conditions may not be serious [or morally degrading] enough to cause it to declare marriage null and void ab initio.

(2) However if the affected party applies for nullity of marriage, nullity may be granted by the court. Partner is not entitled to alimony etc (**S.12 HMA).**

(3) **Conditions (a)** if the marriage has not been **consummated** (i.e. no intercourse has taken place after marriage) owing to the **impotence** of the other party. Consummation of marriage [also known as ***vera copula*** (L. vera, true; copula, union) - Full sexual intercourse, Real union] is considered an integral part of marriage **(b)** if the other party was of **unsound mind (c)** If the consent of the other party was taken by **force** or **fraud (d)** if the wife, at the time of marriage, was **pregnant by some person other than the husband.**

iii. Divorce

(1) **Divorce** is given after marriage is legally deemed to have taken place [**S.13(1)**, **HMA**].

(2) Wife is eligible for alimony.

(3) **Grounds - 9** grounds. (A) **Things which a spouse does to himself** (i) Has had voluntary sexual intercourse with any person other than his spouse (ii) Ceases to be a Hindu by conversion to any other religion (iii) Renounces the world by entering any religious order (B) **Things which a person does to spouse** (i) Treats spouse with **cruelty** [repeated MTPs without husband's consent have been accepted by courts as causing "cruelty to husband" (**ch 26**)] (ii) Deserts spouse for a continuous period of 2 years (C) **Diseases** (i) If the spouse is **incurably of unsound mind** (ii) If he has been suffering from a **virulent and incurable form of leprosy** (iii) If he has been suffering from **venereal disease in a communicable form** [eg HIV, AIDS] (D) **Others:** If he has not been heard of as being alive for a period of 7 years. In addition to the above, certain additional grounds are available only to wives **u/s 13(2), HMA**. These are: (a) If the husband has been guilty of **rape**, **sodomy** or **bestiality** (b)

If the marriage of the girl was solemnized before she was 15 years of age, and upon attaining that age, she repudiated the marriage. She can however repudiate the marriage only till she is 18 years of age; not after attaining that age.

2. Prohibition of Child Marriages Act, 2006

(1) Passed to overcome the shortcomings of Child Marriage Restraint Act, 1929.

(2) It received Presidential assent on 10 Jan 2007, and came into effect from 1 Nov 2007.

(3) It -ed punishment of marriage of girls<18 y and boys <21 y, and also made such marriages voidable. Before 2007 such marriages were neither void, nor voidable. Doctrine of Factum valet applied [what ought not to be done becomes valid when done].

B. Questions of Impotence and Sterility

The question of impotence and sterility arises in both civil and criminal matters.

1. Civil

a. Marriage and its termination

(1) **Impotence *at the time* of marriage**"

(i) **HMA -** Marriage is **voidable**, i.e. if the affected party presents a suit in court for nullity, the marriage is dissolved [**S.12, HMA**]. If no such suit is presented, the marriage may continue.

(ii) **SMA** – Marriage is void [**S.24, SMA**]. For a successful suit of nullity, the **impotence should be *incurable***. If impotence is curable, nullity would not be granted. In a British case S.v.S.(1962), the wife had **vaginal atresia** preventing full intercourse. An artificial vagina could be constructed by means of plastic surgery, but the husband asserted that it would not amount to normal sexual intercourse (***vera copula***). He filed a suit for nullity of marriage, but it was rejected.

(2) **Impotence *developing after* marriage**"Marriage is neither null & void, nor voidable, nor divorce can be obtained. Divorce may however be sought for causing cruelty to the partner (as not being able to perform sexual intercourse may be taken as equivalent to treating the partner with cruelty).

(3) **Sterility *at the time* of marriage**"Marriage is neither null & void, nor voidable, nor divorce may be obtained.

(4) **Sterility *developing after* marriage**" Same as (3) above.

b. Fecundatio ab extra

Fecundatio ab extra refers to conception occurring in a woman when the semen was deposited on her thighs or on the vulva.

Salient features:

(1) **Physiology** - Impotent person"unable to penetrate vagina"semen deposited on woman's thighs or on the vulva"Spermatozoa travel up the genital passage"pregnancy results

(2) If pregnancy results from fecundatio ab extra, marriage is not said to be consummated; it can still be annulled on grounds of impotency.

c. Disputed paternity and legitimacy

A sterile male may dispute the paternity and legitimacy of his child [also see **ch 24**]. Please see **ch 29** for solving disputed paternity.

d. Suits of adoption

e. Claim for damages

When loss of sexual function is claimed as the result of assault or accident.

2. Criminal

Rape and Unnatural offences

3. Both Civil and Criminal

Adultery

Adultery is sexual intercourse by a man ***M*** with a married woman ***W***, without the consent of her husband ***H***. Punishment – 5y or fine or both. Wife is not punishable [**S.497, IPC]**.

VII. STERILIZATION

Sterilization is a procedure to make a person sterile, without interfering with the potency. It is usually done by vasectomy in the males and tubal ligation in the females.

A. Classification

1. According to intent

Whether the intent was to produce sterilization.

a. Direct

The operation was intended to produce sterilization

b. Indirect

Sterilization was an unintended result of an operation, eg removal of a bleeding uterus to save life of a woman [syn, incidental].

2. According to sex

(1) Male sterilization

(2) Female sterilization

3. According to reversibility

a. Temporary [reversible] sterilization

Eg IUCD in females. But this is more appropriately called contraception, rather than temporary sterilization. Most active research currently is on non-hormonal male contraceptives.

b. Permanent [irreversible] sterilization

Eg hysterectomy.

4. According to consent

Whether the consent was present or not.

a. Voluntary

Voluntary or elective sterilization is done with the consent of the person. It is of following types:

(1) Contraceptive – to limit the size of family [family planning]

(2) Eugenic – To prevent conception of children who are likely to be physically or mentally defective. Done in order to improve race by preventing transmission of hereditable anomalies, defects and diseases.

(3) Incidental [syn, indirect]– unintended result of a different operation or

(4) Therapeutic – to prevent danger to the life or health of woman during unintended future pregnancy.

b. Compulsory

Compulsory or forced sterilization is done without the consent of the person, usually on the order of the State.

Salient features:

(1) When done - Usually done on mental defectives or sex offenders

(2) Legal provisions – There is no legal provision for compulsory sterilization in India. However during promulgation of Emergency between 1975 and 1977 a number of persons were compulsorily sterilized in a vain attempt to check rising population.

B. Methods

(1) **Chemical [pharmacological]** – There is no "sterilization pill" that causes permanent inability to reproduce. Antiandrogens [Cyproterone acetate, Leuprolide acetate, Medroxyprogesterone acetate] are used to reduce libido.

(2) **Electrical** - Electrocoagulation is used with a laparoscope to burn and block the tubes. No longer recommended because of risk for internal burns during the procedure and for ectopic pregnancy after the operation.

(3) **Mechanical** – A device (hinged or spring clips or a small ring or band made of silicone rubber) is placed to close and seal each fallopian tube or spermatic duct.

(4) **Radiological** – Radiological exposure of testes. Not recommended.

(5) **Surgical** -

(i) Females – Hysterectomy, Tubal ligation

(ii) Males – orchidectomy [syn, castration, frequently used for the sterilization of animals, but rarely for humans], vasectomy.

C. Guiding Principles

(1) **Consent** –

(i) of subject - required. Person should be >18 y

(ii) of subject's spouse – (a) Not legally required in India, but to avoid marital discord later, it is advisable for doctor to have written consent of subject's spouse also [**ch 2"consent of spouse**]. (b) Countries requiring spousal consent for sterilization - Brazil, Chile, Ecuador, Guatemala, Honduras, Japan, Niger, Rwanda, Taiwan and Turkey. (c) Countries requiring that spouse be informed - Finland, Hungary, and Switzerland.

(iii) Who cannot give consent - (a) Person <18y (b) Mentally unsound

(2) Legality in India – legal after informed consent

(3) Post vasectomy guidelines – Avoid intercourse for 3 months or until semen examination shows absence of spermatozoa on 2 consecutive occasions. Continuous monitoring for 3 months or longer if other methods [chemical,

electrical, radiological] are used.

D. Medicolegal Aspects

(1) **Wrongful birth and life suits:**

(i) Wrongful birth suit - If a sterilization operation fails, a person may suffer the "injury" of being born into the world and may claim damages. Such suits are generally unsuccessful.

(ii) Wrongful life suit - It is an action brought by or on behalf of a deformed child against the doctor [defendant], claiming that because of the negligence of the defendant he or she has to endure a deformed and unhappy existence. The child in a wrongful life claim does not allege that the defendant's negligence caused his or her deformity. Rather, the child alleges that the defendant's negligence, in failing adequately to inform the parents of the risk of a deformed birth, caused his birth. That is, had there been no negligence, the child would not have been born to experience the pain and suffering attributable to the deformity. In other words, he or she would have been better off not being born. [for a variation of wrongly life suit (illegitimacy), please see **ch 24**].

(2) Couple may claim damages for the cost of unsuccessful procedure, pain and suffering as a result of pregnancy, medical expenses of pregnancy, and the loss of comfort.

VIII. THE DELHI ARTIFICIAL INSEMINATION (HUMAN) ACT, 1995

The Delhi Artificial Insemination (human) Act, 1995 (Delhi Act no. 12 of 1996) was passed to provide for the regulation of donation, sale and supply of human semen and ovum for the purpose of artificial insemination. Its **main provisions** are (i) All semen banks in Delhi would have to be duly registered (**S.3**) (ii) Registration shall be renewed every year (**S.4**) (iii) On receipt of application for registration, the Supervisory Authority (Director of Health Services, Govt of NCT of Delhi) shall, if satisfied, register the applicant and issue him a certificate of registration (**S.5**) (iv) If semen bank is run without registration, there is fine of `5000 on first offence. On second offence or subsequent offence, there is imprisonment of 3 months or fine of `5000. (**S.6**) (v)

The semen bank before accepting semen for artificial insemination shall test the donor for **HIV 1** and **2 antibodies** by using ELISA kit. Donor shall be allowed to donate only when found HIV negative (**S.10**) (vi) The donated semen shall be stored by cryopreservation in liquid nitrogen or any other safe method for a minimum period of 3 months in order to exclude window period of HIV 1 and 2 (**S.11**) (vii) At the end of 3 months, a second ELISA test shall be done (**S.12**) (viii) **S.14** gives the **duties of qualified medical practitioner and Government hospitals performing artificial insemination**. They will (a) keep complete record of the bio-data including mark of identification of the donor and the recipient of the semen or ovum; (b) test the recipient for HIV 1 and 2 and sexually transmitted diseases before performing artificial insemination; (c) **seek the written consent of the husband and the wife, seeking artificial insemination**; (d) **seek the written consent of the donor and the recipient and their spouse**, in case of requests of semen or ovum from specified donor/recipient (e) not segregate the XX or XY chromosomes for artificial insemination (f) **maintain secrecy about the identity of the donor and the recipient** of the semen/ovum; (g) seek the written consent of the recipient for using the semen on the basis or only one ELISA Test being negative where facilities for cryopreservation and liquid nitrogen for semen are not available.

IX. ASSISTED REPRODUCTIVE TECHNOLOGY (ART)

(1) **Assisted reproductive technology (ART)** – Refers to the art and science of getting gametes together. May be done by artificial or partially artificial means. May or may not involve third party [eg IVF of parents' own gametes].

(2) **Third party reproduction** - Use of oocytes [oocyte donation], sperm [sperm donation], embryos [embryo donation], or uterus [surrogate motherhood] that have been provided by a third person (donor) to enable an infertile individual or couple (intended parent) to become parents. While the donors may be known or anonymous to the intended recipients depending on the process of recruitment, surrogates are usually known to the intended parents.

(3) **Guidelines for ART in India** – have been laid down by ICMR [***N***ational ***G***uidelines for Accreditation, Supervision and Regulation of ART Clinics in India, 2005 (abbreviated hereafter as - **NG, 2005**)]. No punishments have been mentioned. Not following guidelines may at most call for an action by MCI or

SMC [ch 1].

A. Techniques

1. Artificial insemination

Artificial insemination (AI) is artificial introduction of semen into the reproductive tract of a female to produce pregnancy.

a. Types

(1) Artificial Insemination Homologous (or Husband) [**AIH**] – semen of husband is used

(2) Artificial Insemination Donor [**AID**] – semen of some person other than husband is used

(3) Artificial Insemination Homologous and Donor [**AIHD**] – Pooled semen is used in which semen of both husband and donor are mixed.

b. Medical aspects

(1) **Donor** –

(i) advised not to ejaculate for 2-3 days before providing the sample in order to - the sperm count.

(ii) Tested for HIV, hepatitis B etc. Semen cannot be taken if donor suffering from these diseases.

(2) **Collection of semen** –

(i) through masturbation

(ii) use of an electrical stimulator

(iii) use of a special condom [collection condom] to collect semen during intercourse.

(3) **Quarantine** - The sperm is quarantined for a period of 6 months. Donor re-tested prior to AI.

(4) **Woman's menstrual cycle** - is closely observed, by tracking basal body temperature and changes in vaginal mucus, or using ovulation kits, ultrasounds or blood tests. The time of maximum fertility coincides with that of ovulation. Ovum can survive for a maximum of up to **24 h** without fertilization after it leaves the ovary. Similarly spermatozoa survive for a maximum of **24 h** within female genital tract. The take an average of **6-24 h** for traveling from vagina to tubes. Because of uncertainly of ovulation.

c. Indications

(1) **for AIH** -

(i) Male factors - (a) Husband has an ED [impotency] (b) Husband has a physical anomaly making him unable to deposit semen naturally [elephantiasis, epispadias, hypospadias, hydrocele, penile and scrotal tumors, retrograde ejaculation etc] (c) husband is dead, and his semen was harvested after death [Posthumous assisted reproduction].

(ii) Female factors – Cervical stenosis, Persistent cervicitis, Scant or unreceptive mucus.

(2) **for AID** -

(i) Husband is sterile

(ii) Husband suffers from hereditary disease

(iii) wife is allergic or iso-immunized to husband's sperm

(iv) husband suffering from HIV or hepatitis B

(v) woman is unmarried or a widow

(vi) lesbian couples

(vii) Rh incompatibility [in olden times; not now].

(3) **for AIHD** – Husband has oligozoospermia. By mixing semen with that of donor, sperm count is brought to normal with a possibility that husband's sperm would fertilize ovum.

d. Precautions

(1) **Consent** - of donor, recipient and their spouses is essential. As both donor and recipient identities are shielded from each other, physician is the only link between the two. It should be specifically stated that the physician has full discretion to chose the best possible donor.

(2) **Essential requisites of donor** -

(i) Blood group - No Rh incompatibility. Should have same blood group as that of husband

(ii) Family - Should have completed his own family

(iii) Health - Should be physically and mentally healthy. No familial, hereditary disease or STD [HIV etc]. Chromosomal studies - should be done on him for possible genetic defects

(iv) Physical characteristics, race etc - should match that of the husband of recipient as closely as possible.

(v) Relationship with the couple - Should have no relation to either spouse.

(vi) Single donor's semen should not be used to produce more than ten children. More children in different families from same donor would ultimately - the

possibility of consanguineous marriages.

(3) **Couple**-should be psychologically fit and emotionally stable

(4) **Witness** - must be present at the time of insemination

(5) **Delivery** - should not be done by the physician who performed AI. This avoids the necessity of either falsifying the records or disclosing true paternity in the records.

e. MLI

Please see ML aspects of ART below.

f. Complications

Please see complications of ART below.

2. Conventional in-vitro fertilization and embryo transfer [IVF-ET, test tube babies]

(1) In vitro fertilization is the ***most common ART*** procedure. Mythological IVF is well known. Gandhari bore 100 sons of Dhritarashtra without having any biological relation with him. World's first test tube baby **Louise Brown** was born on **25 July 1978**, in Oldham General Hospital, Greater Manchester, **UK**. Done by **Patrick Steptoe** and **Robert Edwards**. Use of **clomiphene citrate** and **human chorionic gonadotrophin (hCG)** enabled scientists to control and time oocyte maturation. This converted IVF to a viable form of clinical treatment. The world's second and India's first IVF baby, **Kanupriya**, alias **Durga**, was born 67 days later on Oct 3, 1978. Done by Subhas Mukherjee in Kolkata by an entirely different technique. Mukherjee was the first person in the world to use (a) gonadotropins for ovarian stimulation prior to ovum pick-up in an IVF treatment cycle; (b) the transvaginal route by colpotomy for harvesting oocytes; and (c) freezing and thawing of human embryos before transferring them into the uterus. Today there are an estimated 250 IVF clinics in India.

(2) **Procedure** - Controlled ovarian hyperstimulation with exogenous gonadotropins"oocyte retrieval via transvaginal ultrasonographic guided aspiration"Fertilization of oocytes with sperm in culture"formation of zygote"allowing zygote to develop for 3-5 days"Transcervical
Transfer of zygotes to uterine cavity under US guidance.

3. Surrogate motherhood

A surrogate mother is a woman who by contract agrees to bear a child for someone else.

Salient features:

(1) There are **two parties** to the contract – "**the commissioning couple**" (who makes the request) and the **surrogate mother**, who agrees to bear child on behalf of "the commissioning couple" either for money (**Commercial surrogacy, paid surrogacy**), or simply to help the couple out of love and affection [**Altruistic surrogacy**], eg a woman agreeing to bear child for her sister or even her daughter.

(2) **Types** - Surrogacy is of two types: (i) Traditional (Complete) surrogacy – Female of the "commissioning couple" is unable to bear children (ovarian, fallopian tube or uterine pathology). Sperm from the male of the "commissioning couple" is used to impregnate the surrogate mother. Impregnation is done via IUI (intrauterine insemination), or ICI (intracervical insemination) which is performed at a fertility clinic [as in **Baby M case** – please see below]. Rarely intercourse is used for sperm transfer. The couple thus "buys an ovum" from the surrogate mother and also hires her uterus. The surrogate mother returns the baby to the "commissioning couple" after delivery. (ii) Gestational surrogacy (uterine surrogacy)– The female of the "the commissioning couple" has a **functioning ovary**, but there is uterine pathology, so she can not carry on the pregnancy in her own uterus. The couple thus hires the uterus only. The baby is conceived in-vitro with the sperm and egg of "the commissioning couple", and transferred to the uterus of the surrogate mother, who returns the baby the couple after delivery. (iii) Ovarian surrogacy – A third form is also recognized by some. The female of the "the commissioning couple" has a **functioning uterus**, but there is ovarian pathology. She thus "buys only an ovum" from the surrogate mother. Pregnancy is carried out in her own uterus. It is not traditionally considered a case of surrogacy.

(3) **Other classifications** – Surrogacy can be classified in a number of other ways depending upon how it is looked at. (i) **International surrogacy** [syn, overseas surrogacy]- A surrogacy arrangement, involving an overseas country. Assumes significance when one country recognizes surrogacy, but the other does not [please see Jan Balaz and Susanne Anna Lohle case below]. (ii) **Same sex surrogacy** [syn, Gay surrogacy]- Same sex male couple hires a

surrogate mother; same sex female couple hires a sperm donor. In **1999**, **Barrie** and **Tony Drewitt-Barlow** became the first British same-sex couple to be named on their children's birth certificates. They hired a surrogate mother in California [international surrogacy], who gave birth to twin brothers Aspen and Saffron. Following a ruling by an American court, the twins became the first British children to be registered as having two fathers and no mother. In **2003**, they used the same egg donor and a different surrogate to have the 3rd child, and in **2010** another surrogate to have twin boys Dallas and Jasper. They now have five children – all surrogate babies. They set up the British Surrogacy Centre in Essex, in Feb 2011.

Table 3: Salient Differences between AIH and AID

S.No	Criteria	AIH	AID
1.	Donor	Husband	Person other than husband, but not a relative
2.	Indications	As in text	As in text
3.	Consent	Needed from both husband and wife	Needed from husband and wife as well as from donor and his wife
4.	Preconditions	None	Donor should have his own child
5.	Donor characteristics	None	Must be <45 y; should be of same race as husband; should closely resemble husband in physique etc
6.	Medical tests	Sperm count should be normal. Other routine tests	Sperm count should be normal. Other tests to exclude diseases like diabetes, endocrine dysfunction, epilepsy, hereditary and familial disorders, psychiatric disturbances, Rh incompatibility, tuberculosis, transmissible infections eg HIV, AIDS, Hepatitis B etc
7.	Disclosure of identity	Known to each other	Donor and recipient must not know each other
8.	Outcome of AI	Husband knows	Donor must not know
9.	Confidentiality	Not between donor and recipient; but confidentiality maintained from general public	Maintained between donor and recipient as well as from general public
10.	Doctor's role	May deliver the child who conducted AI	Doctor conducting AI should avoid delivering the child, as it would lead to disclosing the identity of father in birth records
11.	Legal issues	As in text	As in text

4. Other techniques

(1) **Embryo donation** [**ED]:**

(i) A form of third party reproduction [please see above].

(ii) It is donation of ***surplus embryos*** remaining after one couple's In vitro fertilization, or IVF treatments, to another person or couple.

(iii) The embryos are typically donated after the woman for whom they were originally created has successfully carried one or more pregnancies to term.

(iv) The resulting child is considered the child of the woman who carries it and

gives birth, and not the child of the donor.

(v) Same principle is followed in oocyte [egg] donation.

(vi) No compensation is generally asked for. It is done on altruistic basis.

(2) **Gamete intrafallopian transfer** (GIFT) – First reported in 1984. After collection of ovum as in conventional IVF, they are mixed with sperm and transferred back into fallopian tube with the hope that they would form a zygote. A fundamental problem with this procedure is that fertilization is not confirmed unless the patient conceives. Therefore other techniques like ZIFT and TEST were developed.

(3) **Intracytoplasmic Sperm Injection** [ICSI]:

(i) Procedure – (a) The ovum is injected with a single sperm, taken from the husband's sperm (after preparation in the lab). (b) A special needle is used to go through the wall of the egg and the sperm is introduced into the cytoplasm of the egg. (c) The fertilization is observed; if division occurs, the embryos are transferred into the woman's uterus.

(ii) Indications - (a) Cases who had vasectomy and surgical reversal failed. (b) Past Infections which result in surgically intractable obstructions. (c) severe problems in the seminal fluid [eg severe deficiency in number or motility of the sperms or both]. (d) when the man has no sperms in the seminal fluid but has sperms in his testicles, eg absence of vas deferens. One of the techniques of sperm retrieval is used [please see below].

(iii) Advantages - Only a single sperm is required, and it does not even have to be motile.

(4) **Oocyte Donation [OD] or egg donation** – Donors may be

(i) Anonymous - Donors unrelated to the recipients who do it for altruistic or monetary reasons. They are typically recruited by egg donor agencies.

(ii) Designated donors - a friend or relative brought by the patients to serve as a donor

(iii) Patients taking part in shared oocyte programmes. Women who go through IVF donate unused eggs to others.

(5) **Techniques of Sperm Retrieval** – These techniques are for sperm retrieval only. Must be followed by ICSI [please see above]

Techniques are (a) Microsurgical Epididymal Sperm Aspiration or MESA (b) Percutaneous Epididymal Sperm Aspiration or PESA (c) Testicular Microdissection (d) Testicular Perc-Biopsy (e) Testicular Sperm Aspiration or

TESA (f) Testicular Sperm Extraction or TESE (I) Indications – Same as ICSI above. The two procedures are used in combination

(6) **Tubal Embryo Stage Transfer** [TEST, embryo intrafallopian transfer, Tubal Embryo Transfer, TET] – Same as ZIFT (please see below), except that here pre-embryos are transferred on day 2 or 3 after fertilization. They are thus more mature. Its advantage over ZIFT is that it allows for the assessment of fertilization and embryo quality.

(7) **Zygote Intrafallopian transfer** (**ZIFT**) – Same as GIFT, except that fertilization occurs outside the body and zygote is transferred back to the woman. In both TEST and ZIFT, the embryos are placed in an environment which is expected to be optimal for further development and implantation.

(8) **Other miscellaneous procedures:**

(i) Controlled ovarian hyperstimulation only [COHS]

(ii) direct intraperitoneal insemination [DIPI]

(iii) fallopian tube sperm perfusion [FSP]

(iv) intrauterine insemination [IUI]

(v) combination of IUI and DIPI and

(vi) peritoneal oocyte and sperm transfer.

B. Semen Banking

A ***semen bank*** [syn, sperm bank] is a facility that collects and stores human sperm mainly from sperm donors, primarily for the purpose of achieving pregnancies through AI.

C. ML Aspects of ART

There is no law on artificial insemination applicable in the entire country [The Delhi Artificial Insemination (Human) Act, 1995 is applicable only in Delhi]. **NG, 2005** [please see above] are only the nature of guidelines.

(1) **Adultery:**

(i) ART used for married woman with the consent of the husband does not amount to adultery on part of the wife or the donor. AID without the husband's consent can, however, be a ground for divorce or judicial separation [**S.3.16.2, NG 2005**]

(ii) **u/s 497, IPC** – sexual intercourse is an essential element in adultery. Since

AID does not involve sexual intercourse, it does not amount to adultery. If husband consents to AID through actual intercourse with a third party, it cannot amount to adultery, since adultery can only be committed in the absence of husband's consent.

(2) **Confidentiality** - Any information about clients and donors must be kept confidential. [**S.3.2.3, NG 2005**]

(3) **Consummation of marriage** – Conception by ART, even in cases of AIH, does not amount to consummation of marriage, although the child conceived may be biologically that of husband (thus marriage remains voidable) [**S.3.16.3, NG 2005**]

(4) **Damages** – doctor can be sued for damages if

(i) He conducts AIH without husband's consent or conducts AID without donor or his wife's consent

(ii) Recipient contracts a transmissible infection like HIV.

(5) **Divorce and judicial separation** – please see "Adultery" above.

(6) **Incest** – Risk of inadvertent admixture of gametes between closely related couples is a distinct possibility because of confidentiality. It has been asserted that it is doctor's duty to check records and exclude possibility of close relations, eg by checking surnames etc, but as surnames change after marriage, such cross-checking on the part of doctor is impossible. Nevertheless in order to avoid damage suits later, doctor should specifically include the exclusionary clause in the contract making him free from such liabilities.

(7) **Legitimacy** - A child born through ART shall be presumed to be the legitimate child of the couple. He shall have a legal right to parental support, inheritance, and all other privileges of a child born to a couple through sexual intercourse [**S.3.12.1, NG 2005**]. In legal documents, eg birth certificate, school leaving certificate, passport etc, the name of the parents to whom he was born will be entered, even if the child was genetically unrelated to them. It is furthermore advisable for parents to adopt the child legally.

(8) **Minimum age for ART** - For a woman between 20 and 30 years, two years of cohabitation/marriage without the use of a contraceptive, excepting in cases where the man is infertile or the woman cannot physiologically conceive. For a woman over 30 years, one year of cohabitation/marriage without use of contraceptives. Normally, no ART procedure shall be used on a woman below 20 years [**S.3.14.1, NG 2005**]

(9) **Nullity of marriage** – please see c**onsummation of marriage** above

(10) **Posthumous AIH** - A child born to a woman artificially inseminated with the stored sperms of her deceased husband must be considered to be a legitimate child notwithstanding the existing law of presumptions **u/*s 112, IEA*** Indian Evidence Act, which says that child born after 280 days of death of husband is illegitimate [ch 24] [**S.3.16.5, NG 2005**].

(11) **Rights of a Child Born through ART Technologies** [**S.3.12, NG 2005**]:

(i) Children born through the use of donor gametes shall not have any right whatsoever to know the identity (such as name, address, parentage, etc.) of their genetic parent(s) [**S.3.12.3, NG 2005**]

(ii) In the case of a divorce during the gestation period the law as pertaining to a normal conception would apply [**S.3.12.4, NG 2005**]

(12) **Unmarried woman opting for AID** – There is no law barring an unmarried woman to AID. The child would inherit all property of the woman, if the woman dies intestate. However it is advisable for her to make a will immediately after birth of such a child and make him a beneficiary.

24. Virginity, Pregnancy and Delivery

I. VIRGINITY

A. Definitions

(1) A ***virgin*** (virgo intacta) [Latin virgo, "sexually inexperienced woman"] is a female who has not experienced sexual intercourse.

(2) **Defloration** [Latin de, away; flore, flower. A poetic likening of the rupture of hymen by sexual intercourse to the plucking of flowers] means loss of virginity with associated rupture of hymen. Such a woman is called a **deflorate woman**.

(3) Loss of virginity without associated rupture of hymen results not in a deflorate woman, but a false virgin.

(4) ***Apta viro*** [please aptae viris] is a woman who is "fit for a husband" or "a woman who has reached marriageable years".

B. Normal Female Genital Anatomy

1. External

The **labia majora** (1) [L. labia, lip] [**Fig 24.1**] are two elongated folds of skin projecting downwards and backwards from **mons pubis** (2) [Latin, "pubic mound". Also known as **mons veneris** ["mound of Venus"] or simply the **mons**]. It is a pad of fat lying in front of pubis. They meet in front at **anterior commissure** (3) and at the back at **posterior commissure** (4) in front of the **anus** (5) In a virgin the labia majora are thick, firm, elastic and rounded and lie in apposition so as to completely close the **vaginal orifice**. (6) The **labia minora** (8) are about 4 cm long; they are two soft, small, thin, pink and sensitive folds just within the labia majora. Between **Labia majora** and the **labia minora** is a groove called **interlabial sulcus**. (19) There are 2 such sulci-left and right. The lower portions of labia minora fuse in the midline and form a fold called **fourchette** (9) [Fr, "little fork"]. The depression between fourchette and the vaginal orifice is called **fossa navicularis** (10) [L fossa, depression; navicula, boat; a boat shaped depression]. **Clitoris** (11) [Gk, kleis, key; in reference to it being key to female genitals] is a small button-like organ located near the anterior junction of the labia minora, above the **urethral opening** (12) and vagina. It is covered with a fold of skin that surrounds and protects it [**clitoral prepuce**, clitoral hood, or preputium clitoridis). It develops as part of the labia minora and is homologous with the foreskin (also called prepuce) in male genitals. The **frenulum** (13) [also known as the Crus glandis clitoridis] is a small fold of tissue on the under surface of clitoris, created by the two medial parts of the labia minora. Urethral opening is 2.5 cm behind the clitoris and immediately in front of the vaginal orifice. **Vestibule** (14) is a narrow triangular area which extends from the clitoris above to the anterior margin of the hymen below, and laterally to the labia minora.

Memory Aid 1: Female genital anatomy

Fossa navicularis is in **F**ront of **F**ourchette; **V**estibule is **V**entral to **V**agina.

It usually remains concealed by the labia. **Vulva** includes mons veneris [which forms its anterior portion], labia major, labia minora, clitoris, vestibule, hymen and urethral opening. The **perineum** (15) is a wedge shaped area between the lower end of the posterior wall of vagina and the anterior anal wall. The cervical canal is nearly at right angles to the vagina when bladder and rectum are empty.

Bartholin's glands (16) secrete a mucoid fluid, which drains in vagina through Bartholin duct openings (17).

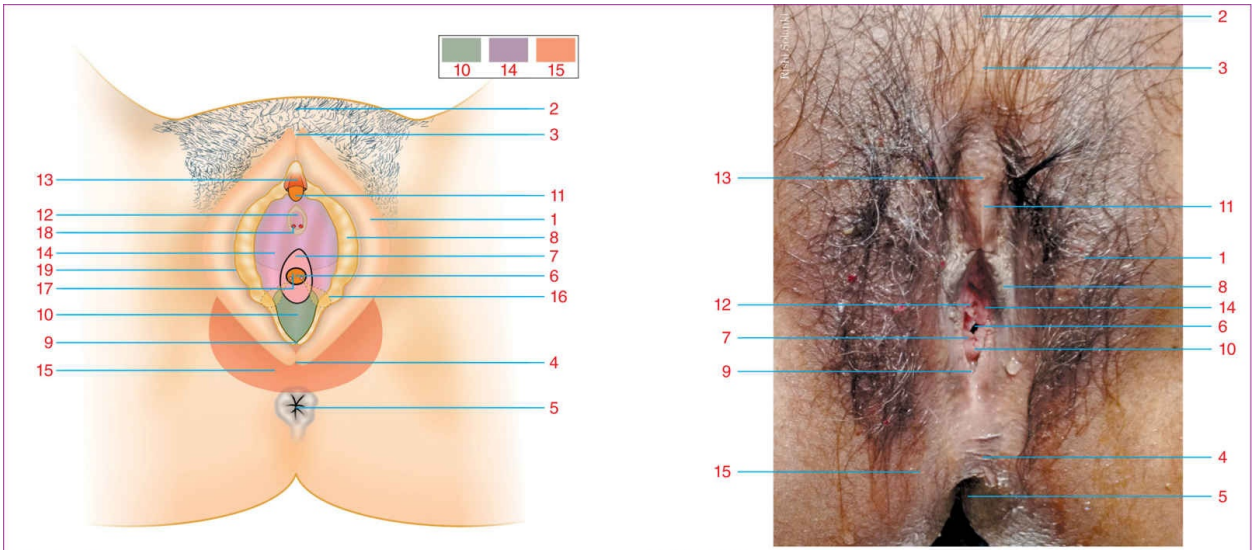


Fig. 24.1: Normal female genital anatomy (External). Please correlate nos (given in red). with text.

Hymen

Hymen [**Fig 24.1**, (7)] [Gk Hymenaios, God of marriage] is a fold of mucus membrane about 1 mm thick, situated at the vaginal outlet.

Salient features:

(1) The average adult hymen consists of folds of membrane having an annular or crescentic shape, the broadest part lying posteriorly

(2) **Diameter of hymenal orifice in children** – (i) 1 mm/y [eg 1 mm at 1 y; 2mm at 2 y and so on] till it reaches about 10 mm in a prepubertal girl. (ii) >10mm diameter in a prepubertal girl indicates sexually abused girl. [Please compare with hymenoscope ball sizes **(ch 25)**].

i. Types

Shown in **Fig 24.2**. Differences between fimbriated and ruptured hymen [**Table 1**].

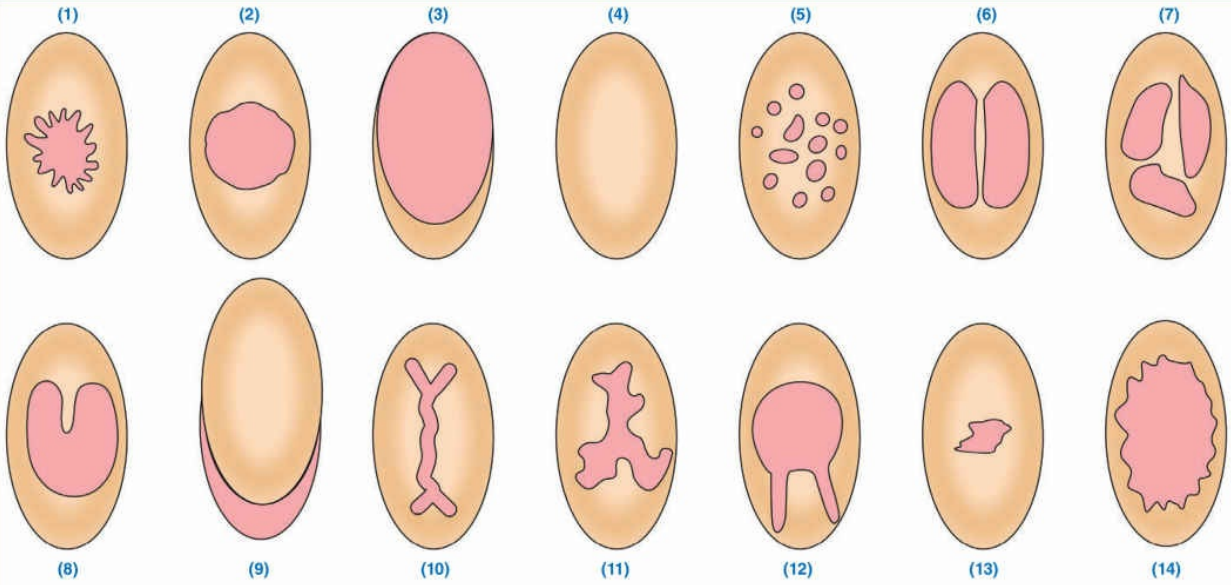


Fig. 24.2: Some common types of hymen: (1) Fimbriated or denticular – the free edges of hymen show indentations, but the indentations do not reach the peripheral margin [**Table 1**]. Compare it with a torn hymen (12), where tears are reaching up to the periphery. This is the usual criteria to distinguish between these two types of hymen (2) Annular or Circular [Hymen anularis]– the hymen has an almost circular central opening (3) Semilunar – The hymen looks like a crescent (4) Imperforate [Hymen imperforatus]– The hymen has no opening at all. In such cases, at the onset of menstrual cycle, the doctor needs to make a surgical opening to let menstrual blood out. If this is not done, it may lead to collection of blood within the distensible vagina (hematocolpos) or uterus (hematometra) (5) Cribriform or Fenestrated [Hymen cribriformis]- a hymen pierced by many small perforations. This type of hymen lets menstrual and other fluids out with no problem, but sexual activity and the insertion of tampons can be problematic (6) Septate [Hymen bifenestratus] - a hymen in which the opening is divided by a narrow septum. If the septum is somewhat broader, it has been called hymen bifenestratus or hymen biforis. (7) Hymen trifenestratus – three openings are seen (8) Subseptate - a hymen in which the opening is partially filled by a septum growing out of one wall, but not reaching the other. It almost reminds one of throat with the uvula hanging down (9) Falciform - hymen, leaving out a sickle-shaped opening (10) Vertical [Hymen labialis]– the opening is almost vertical slit like (11) Irregular – No regular pattern can be made out (12) Normal injury at coitus – hymen shows a tear. Note that the tears reach the peripheral margin. This is an important feature to note. Please look at fimbriated hymen (1). and see that the indentations in that case do not reach up to the peripheral margin (13) Hymen microperforatus-opening much smaller than normal (14) Carunculae myrtiformes.

Table 1: Differences between fimbriated and ruptured hymen [Fig 24.2]

S.No	Feature	Fimbriated hymen	Ruptured hymen
1.	Situation of notches	Present anteriorly. Do not extend to the base (periphery) of hymen	Present posteriorly [6 O′ clock position], or both sides of the median line [5 O′ clock and 7 O′ clock position]. Extend to the periphery of hymen [Fig 24.2] [for explanation of clock positions, please see ch 25]
2.	Shape & size of notches	Bilaterally symmetrical	Asymmetrical
3.	Mucus membrane over notches	Intact	Torn
4.	Cause	Occurs naturally	Sexual intercourse or introduction of large foreign bodies

ii. Rupture

Rupture of hymen during first sexual intercourse is known as ***defloration***; such a woman is termed a ***deflorate woman***.

Salient features:

Causes other than sexual intercourse – (i) Accident – (a) Straddle injuries eg

(I) Fall on a projecting object (II) Slipping on fence, furniture, pole etc (III) Playing at seesaw (b) Ch of accidental rupture - (I) Associated tearing of perineum (II) periurethral tissues, labia and mons usually involved (III) Injuries on other parts of the body are also seen, consistent with the history of accident (IV) tears generally not associated with abrasions or bruising of hymenal margins (c) Separation of thighs forcibly in children – generally will not rupture hymen. But if separated too far with tear of perineum, hymen may simultaneously be ruptured (d) Hymen does not usually rupture by (I) Dancing (II) Jumping (III) riding (IV) vigorous exercise (ii) Bad hygiene - causes irritation"scratching"torn hymen (iii) Foreign body – (a) Sola pith – Used by prostitutes to dilate the vagina of very young girls to make them suitable for sexual intercourse [aptae viris]. Sola is an Indian plant with stems containing woody pith [sola pith]. It is sponge wood with great absorptive powers. A piece of sola pith is inserted into the young girl's vagina. The piece is as large as the vagina can contain. The girl is asked to sit in a tub of water. The absorbent sponge wood absorbs water and swells up, enlarging the vagina. The process is repeated with bigger pieces of sola pith, till the vagina is roomy enough for sexual intercourse. The hymen is torn in this process. (iv) Gynecological examination (v) Masturbation – (a) especially with a large object (b) If manipulation is limited to parts anterior to hymen, it may be spared (c) Characteristics –(I) Clitoris and labia minora enlarged (II) Vaginal orifice dilated (III) Vagina roomy (IV) hymen thickened and shows scratches (vi) Sanitary tampons (vii) Surgical operation (viii) Ulceration – diphtheria, fungus, other causes.

2. Internal

a. The vaginal passage

(1) **Passage** - The vaginal passage is a roughly cylindrical tube, about 7.5 cm long.

(2) **Walls** - The tube's anterior wall is shorter [6 cm], and posterior wall longer [9 cm].

(3) **Cavity** - It is collapsed antero-posteriorly to form a crosswise slit. When distended, it looks like a gourd, wider at the top.

(4) **Width at the upper end** - (i) 3-4 cm in nullipara and (ii) 6-7 cm in parous women.

(5) **Fornix** -

(i) The fornices of the vagina (fornix vaginae) [Latin fornix, arch] are arch like recesses around the cervix.

(ii) They are the deepest portions of the vagina, created by the vaginal portion of cervix.

(iii) Number - There are four fornices in all - (a) the **posterior fornix** is the largest recess, and lies behind the cervix. It is close to the rectouterine pouch (b) the **anterior fornix** is shallower and lies close to the vesicouterine pouch (c) The two **lateral fornices** lie on each side of the cervix.

b. Relations

(1) **Anteriorly** – Bladder

(2) **Laterally** – Levator ani muscles

(3) **Posteriorly** – Perineal body and rectum

(4) **Superiorly** – Pouch of Douglas.

C. Signs of Virginity

1. Genital signs

(1) **Hymen** -

(i) Normal hymen is annular or crescentic in shape, but may be of other shapes [**Fig 24.2**]

(ii) Thickness - 1 mm

(iii) Broadest part - lies posteriorly

(iv) May be very fleshy or cartilaginous sometimes [may result in false virgin]

(v) At 10 years of age, the tip of small finger can be passed through it; at puberty, one finger can be passed [also see diameter of hymen above].

(2) **Posterior commissure and fourchette** – intact. They are usually torn after violent and forcible sexual intercourse [**ch 25**] or childbirth. A single act of intercourse does not cause much alteration in genitalia, except rupture of hymen.

2. Extragenital signs

Breasts

(1) **In a virgin** - the breasts are firm, elastic and hemispherical. Nipple is small

and undeveloped, surrounded by an areola which is pink in fair complexioned women and dark brown in dark women.

(2) **Breasts become large and flabby** - by frequent handling, manipulation, sexual intercourse, lactation and suckling

(3) **Single act of coitus** - does not enlarge breasts

(4) **Milk** – present only in lactating mothers. In some hormonal disorders, milk may be seen even in virgins.

D. Medicolegal Aspects of Virginity

1. True virgin v False virgin

(1) Classically presence of an unruptured hymen was considered a sign of virginity. But now it is known that this is not true. Because of the ancient and classical association of intact hymen with virginity, any woman with an intact hymen may be called a virgin by a layman or even an inexperienced doctor. But if she has experienced sexual intercourse, she would be a false virgin [i.e. one who appears a virgin on hymenal examination, but is not truly a virgin].

(2) **True virgin** is a woman who has never experienced sexual intercourse. Her hymen may or may not be intact.

(3) **False virgin** is a woman who has experienced sexual intercourse, but her hymen is intact.

(4) **Principal signs of virginity** - **Table 3**. Any single sign alone may not be taken as a sure sign of virginity, but all signs taken together are virtually diagnostic of virginity.

(5) **Rupture of hymen** – usually occurs at first intercourse.

(6) **Causes of intact hymen** even after 1st intercourse – If it is elastic, fleshy, folded, loose, thick or tough. Such a hymen permits displacement, distortion and stretching without rupture.

(7) Intact hymen has been seen in women having repeated sexual acts, women presenting for delivery and even prostitutes [although in some prostitutes, intact hymen may be due to hymenoplasty].

(8) Carunculae myrtiformes or Carunculae hymenales– In women used to intercourse [eg married women, prostitutes etc] the hymen almost disappears giving rise to just a few small, fleshy, minuscule, round projections or tags of tissue at the periphery [hymenal ring].

Table 2: Relationship between hymen and sexual intercourse

Hymen / Sexual Intercourse	Intact	Torn
Not Experienced	True virgin	Accident, bad hygiene etc [please see above for full list]
Experienced	False virgin	Deflorate woman. Normal case of sexual intercourse

2. Divorce

If the woman was not a virgin at the time of marriage, there is no provision either for nullity or divorce under ***HMA 1955*** [**ch 23**].

3. Defamation

A person may be charged for defamation [**S.499, IPC**] if he alleges loss of virginity of a woman, and she is found to be a virgin on examination.

4. Rape

Allegations of rape may be proved if loss of virginity can be shown, keeping all other factors in consideration.

5. Nullity of marriage

If a married woman wants her marriage to be declared **null and void** on account of impotency of husband (**S.12 of *Hindu Marriage Act, 1955*),** she must prove that she has been a virgin, and that her husband has been unable to consummate marriage [please see medicolegal aspects of impotence and sterility in previous chapter].

II. PREGNANCY

Pregnancy is the condition of having a developing embryo or fetus in the female, when an ovum is fertilized by a spermatozoon.

Table 3: Differences between True and a False virgin

S.No	Feature	True Virgin	False virgin
1.	Sexual intercourse	Not experienced	Experienced
Genital signs			
2.	Hymen	Intact, rigid, inelastic, edges distinct and regular. Admits tip of little finger only	Intact, but loose, elastic, thick, tough and fleshy. Easily admits two fingers
3.	Labia majora	Adjacent to each other and in opposition. Completely close the vaginal orifice	Separated, not opposed to each other, vaginal orifice may be seen between them
4.	Labia minora	Small, pinkish, In contact with each other; covered by labia majora	Enlarged, pigmented, not in contact with each other, not covered by labia majora
5.	Vagina	Narrow and tight; mucosa rugose and reddish; sensitive to touch; walls approximated	Large and roomy; vaginal rugae less marked [they only disappear completely after first birth]; vagina lengthened, especially towards the posterior fornix. Full length of examining finger can be passed into the posterior fornix.
6.	Fossa navicularis	Intact	Disappears
7.	Fourchette	Intact	Torn. May show a healed scar
8.	Vestibule	Narrow	Gaping, spacious, wide
9.	Clitoris	Small	Enlarged
10.	Posterior commissure	Intact	Generally torn
Extragenital signs [Breasts]			
11.	Size, shape, feel	Small, hemispherical, firm	Large, pendulous, flabby
12.	Areola	Pink	Pigmented
13.	Nipples	Small and pink	Enlarged, pigmented

Salient features:

Age – occurs between 14-45 y, but has been recorded beyond either extreme.

A. Diagnosis of Pregnancy

1. Signs of pregnancy in the living

a. Presumptive signs of pregnancy

Probability of pregnancy is about **25%**.

i. Amenorrhea

(1) Earliest sign

(2) Other reasons for amenorrhea

(i) endocrine or local pathology, environmental and nutritional causes, severe anemia, tuberculosis etc

(ii) fear or nervousness [due to illicit intercourse in an unmarried woman]
(iii) intense desire for pregnancy [in married women]
(3) Pregnancy during already existing amenorrhea – may occur
(i) in women who have never menstruated
(ii) during amenorrhea of lactation.

ii. Breast changes

Quite characteristic in primigravida but are of less value in multigravida
(1) Up to 8 weeks – tenseness and tingling in breasts
(2) After 2nd month -
(i) -in size
(ii) become nodular [due to hypertrophy of mammary alveoli]
(iii) Superficial veins - enlarged and more distinct
(iv) Nipples – deeply pigmented and more erectile
(v) Areola – becomes dark brown [it is pink in the virgin]. **Montgomery's tubercles** are seen. These are small rounded dark colored tubercles seen on areola [represent enlarged sebaceous glands].
(3) 3rd month – Colostrum is secreted.
(i) It is a thin, yellowish fluid consisting of fat globules and large phagocytic cells filled with droplets of fat.
(ii) It can be expressed from the breasts by gentle massage.
(4) 6th month - Striae [silvery lines] are seen on the breast due to stretching of the skin. More prominent in primiparae.

iii. Fatigue

(1) Women in the first trimester of pregnancy experience significantly greater fatigue; they spend more hours sleeping.
(2) Could be due to neuroactive steroids.

iv. Morning sickness

(1) Comprises of nausea and vomiting, seen usually in the morning. Passes off in a few hours
(2) Appears by the end of 4 wks and ends by 12 weeks
(3) Varies greatly in severity.

v. Hyperpigmentation

(1) **Incidence** - Develops in up to 90% of women.
(2) Usually more accentuated in those with a darker complexion.

(3) **Main changes** are

(i) Linea nigra – [Latin, “black line”] Pigmentation of the midline of the abdominal skin. Extends from pubis to beyond the umbilicus

(ii) Melasma gravidarum - Irregular brownish patches of varying size on the face and neck. Also known as chloasma or **mask of pregnancy**.

(iii) Pigmentation of the areolae and genital skin may be accentuated.

(4) **Etiology** -

(i) Melanocyte-stimulating hormone which is -ed during pregnancy

(ii) Estrogen and progesterone also have melanocyte-stimulating effects.

(5) **D/d** - Oral contraceptives may cause similar pigmentation.

(6) These pigmentary changes usually disappear, or regress considerably, after delivery.

vi. Quickening

(1) ***Quickening*** is slight fluttering movements felt by the mother from about 16^{th} -20^{th} wks of pregnancy.

(2) Due to movements of the fetus.

vii. Sympathetic disturbances

Irritable temper, Perverted appetite, Salivation.

viii. Urinary disturbances

(1) During early weeks of pregnancy, the enlarging uterus exerts pressure on the bladder"Frequent micturition.

(2) As pregnancy advances, uterus rises up into abdomen"Frequency returns to normal

(3) Few weeks before term"head descends in pelvis"frequency -es once again.

ix. Vaginal changes

(1) **Color change in the vulvovaginal mucosa** - changes from rose color in the nonpregnant state to pale violet in early pregnancy [**Chadwick’s sign, Jacquemier’s sign, Jacquemin’s sign**]. First seen at 4^{th} week as a result of venous obstruction. Other changes at this period

(2) Anterior wall of vagina – flattens

(3) Vulval and vaginal tissues – become softer

(4) Secretion of mucus -es

(5) Vaginal pulsation - can be felt.

x. Vascular changes

Angiomas [**vascular spiders**], develop in about 65% of white women and 10%

of colored women. These are minute, red elevations on the skin, particularly common on the face, neck, upper chest, and arms, with radicles branching out from a central lesion. The condition is often designated as nevus, angioma, or telangiectasis. **Palmar erythema** is seen in about 65% of white women and 33% of colored women.

b. Probable signs of pregnancy

Probability of pregnancy is about **50%**.

i. Enlargement of abdomen

(1) End of 12^{th} wk [3^{rd} m] – uterus fills up the pelvis

(2) >12^{th} wk – uterus appears over the brim. This causes abdomen to show visible enlargement in size [**Table 4**].

(3) end of 20^{th} wk [5^{th} m] – Uterus is midway between symphysis and umbilicus

(4) end of 6^{th} m –

(i) Uterus at umbilicus

(ii) Striae gravidarum appear. (a) These are bluish or slightly pinkish, curved irregular, depressed lines arranged more or less concentrically, sometimes radially around umbilicus. (b) They gradually become broader and deeper near inguinal ligament. (c) Locations - abdomen (most common; 48%), breasts (25%), thighs (25%), hips (d) Etiology - Rupture of subcuticular elastic fibres, due to gradual distension of abdomen. Hormonal changes also play a role (e) Most women experience striae gravidarum during their first pregnancy. Some who do not develop striae in their initial pregnancy develop them in subsequent pregnancies. Some women do not develop them at all. Genetics may play a role. (f) Progression - Become paler with time. After 1 y, appear as white scars [linea albicantes]. These are irregular, white or silvery s/c scars. They do not develop in all cases, and in some cases they disappear after sometime.

(5) end of 7^{th} m –

(i) Uterus midway between umbilicus and xiphisternum

(ii) umbilicus becomes level with the skin

(6) end of 8^{th} m – at xiphoid cartilage

(7) 9^{th} -10^{th} m –

(i) Uterus descends in pelvis

(ii) falls forwards due to its weight.

ii. Uterus

Hegar's sign [after German physician **Alfred Hegar** (1830–1914)]:

(1) Most valuable sign in early pregnancy.

(2) +ve at 6th wk [**Table 4**].

(3) How to elicit:

(i) One hand is placed on abdomen

(ii) Two fingers of the other in vagina.

(iii) Firm hard cervix is felt, and above it the elastic body of the uterus. Between the two, the isthmus is felt as a soft compressible area.

iii. Cervix

(1) **Goodell's sign** - Softening of the vaginal portion of the cervix from -ed vascularization. Softening proceeds from below upwards. Starts at 2nd month [8 weeks] and is well marked by 4th month. After American gynecologist **William Goodell** [1829 – 1894].

(2) **Shortening of cervix** – towards last months of pregnancy

(3) Orifice – becomes circular instead of being transverse. Admits tip of finger to greater depth.

iv. Braxton Hick's sign

Intermittent painless contractions. Felt after 4th month [16 weeks]. Contractions last about 1 minute followed by a relaxation period of 2-3 minutes. Present even when the fetus is dead. After English obstetrician **John Braxton Hicks** [1823 – 1897].

v. Ballottement

Ballottement [Fr ballotter, to toss] is a palpatory technique for confirmation of pregnancy, as the fetus can be tossed up and down like a ball within the uterus.

Salient features:

(1) Sign is +ve by 16-20 wks [**Table 4**].

(2) **Fetus can be tossed up and down at this period** - because (i) fetus is small in relation to the amniotic fluid present, and (ii) the umbilical cord has lengthened enough to allow free movement of fetus

(3) **Demonstration** - (i) External ballottement – (a) Sudden motion is given to the abdominal wall covering the uterus (b) Rebound of the fetus can be felt in a few seconds (ii) Internal [vaginal] ballottement - (a) two fingers inserted into

the anterior fornix (b) sudden upward motion given (c) Fetus “tosses up” in the liquor amnii, and after a moment drops back on the fingers, like a ball bouncing back

(4) **False –ve** – When amniotic fluid is scanty.

Table 4: Important Medicolegal dates (from LMP)

S.No	Weeks after LMP	Sign
1.	**4th week**	Jacquemier's sign
2.	**5th week**	(1) Fetal cardiac action detected with real time sonography with a vaginal probe (2) Gestational sac demonstrated by abdominal sonography (3) Von Braun-Fernwald's sign
3.	**6th week**	(1) Hegar's sign (2) Ladin's sign
4.	**8th week**	(1) Goodell's sign (2) Osiander's sign (3) Palmer's sign (4) Piskaçek's sign
5.	**12th week**	(1) Abdomen gradually enlarges in size (2) Fetal parts detected by X-ray (3) Pregnancy becomes abdominal (4) Uterus becomes an abdominal organ
6.	**16th week**	(1) Ballottement [16-20 wks] (2) Braxton Hick's sign (3) Quickening (in multi) (4) Uterine soufflé heard
7.	**18th week**	(1) Fetal heartbeat detected by stethoscope
8.	**20th week**	(1) Doctor can detect fetal movements (2) Quickening (in primi)
9.	**24th week**	Fetal movements
10.	**36th week**	Fetal parts

vi. Ladin's sign

Softening in the midline of the uterus anteriorly at the junction of the uterus and

cervix [6th wk].

vii. Osiander's sign

Feeling of pulsations through the lateral fornices [**8th wk**]. After German obstetrician **Johann Friedrich Osiander** [1787-1855].

viii. Palmer's sign

Regular and rhythmic contractions of the uterus in early pregnancy [8 wks]. Two fingers of right hand are put into the vagina and left hand is put on the lower abdomen. Wait for 2-3 minutes"uterus becomes firm and then ill defined according to its contractions and relaxation. The sign was important in pre ultrasonography days; nowadays it is only of theoretical importance.

ix. Piskaçek's sign

Asymmetrical enlargement of uterus, if there is lateral implantation. One half of uterus is firmer than the other [**8th wk**]. After Austrian obstetrician **Ludwig Piskaçek** [1854-1932].

x. Uterine soufflé

Uterine soufflé is a soft blowing murmur due to passage of blood through the uterine vessels.

Salient features:

(1) Synchronous with mother's pulse [please also see placental and funicular souffle below, which are synchronous with fetal ht sounds]

(2) Heard by auscultation on either side of the uterus just above ingutinal ligament

(3) +ve at the end of 16th wk

xi. Von Braun-Fernwald's sign

Irregular softening and enlargement of the uterine fundus during early pregnancy [**5–8 wks**]. After Austrian obstetrician **Karl von Braun-Fernwald** [1822–1891].

xii. Biological tests

Biological tests are based on the reaction of test animals to Human Chorionic Gonadotropin (hCG) contained in the pregnant woman's urine.

Salient features:

Prevalent before 1960s, but **now completely taken over by immunological tests**.

xiii. Immunological tests

Based on 'specific' fetoplacental products, eg hCG.

(1) **Advantages** -
(i) Convenient and sensitive [accuracy 98%]
(ii) No animal is required
(iii) quicker [2 min]
(2) **Sample taken** – First voided urine in the morning. Contains highest levels of hCG. Following immunological tests are available.

Table 5: Timings of immunological tests

S.No	Days after LMP	Feature
1.	14	Ovulation and fertilization
2.	21	(i) Implantation occurs (ii) hCG appears in maternal blood
3.	25	hCG appears in maternal urine
4.	28	(i) Time of next expected menstrual period. (ii) Immunological tests are +ve in 98% cases
5.	35	-ve result rules out pregnancy

c. Positive signs of pregnancy

Probability of pregnancy is **100%**.

i. Fetal movements

Felt by 24th wk by placing hands on abdomen.

ii. Fetal parts

Identified by 36th wk by abdominal palpation.

iii. Fetal heart sounds

(1) **Time** - First heard between 18-20 wks [**Table 4**].
(2) **Nature** -
(i) Sounds are similar to ticking of a watch placed under a pillow.

(ii) Not synchronous with mother's pulse

(3) **Rate** -

(i) 20th wk – 160/min

(ii) 36th wk – 120/min

(4) **Fetal ht sounds not heard in following cases** -

(i) Dead fetus

(ii) In live fetus when (a) Examination done before 18 wks (b) Fat abdominal wall (c) Excessive liquor amnii.

iv. Placental soufflé

(1) A soft murmur heard over the placental site.

(2) Synchronous with the fetal ht sounds [unlike uterine soufflé – please see above].

v. Funic or umbilical soufflé

(1) A soft blowing murmur due to passage of blood in umbilical cord.

(2) Synchronous with the fetal ht sounds.

vi. Ultrasound

(1) Done by employing 3-dimensional scanning with Ultrasound

(2) **Time** -

(i) 6th wk – Gestational sac is seen as a white ring

(ii) 7th wk – Distinct echoes from the embryo within the gestational ring

(iii) 10th wk – Fetal heart beat becomes detectable

(iv) 11th wk – Earliest period when sex of baby may be determined. But accuracy rate is very low

(v) 13th wk – Accuracy of determination of sex is between 99% to 100% if there is no malformed external genitalia. Sex of unborn child cannot be disclosed to any person [please see PCPNDT Act below].

(vi) 14th wk – Fetal head and thorax become detectable.

vii. X-ray

(1) **Earliest detection** – 12 wks. At 16 wks, can be detected with absolute certainty [skull and spine are seen at this time]

(2) **Shadows to be seen are** -

(i) Crescentic or annular shadows of skull

(ii) A series of small dots in a linear arrangement along the line of vertebral column [vertebrae]

(iii) A series of fine curved parallel lines [ribs]
(iv) Linear shadows of limbs
(3) X-ray examinations also useful in
(i) Fetal abnormality or death [**ch 27**]
(ii) Hydatidiform mole
(iii) Twin pregnancy.

2. Signs of pregnancy in the dead

(1) **Uterus** -
(i) Weight of a virgin uterus is"30-40 g
(ii) Adult uterus measures 3” # 2” # 1”. Divided into two parts – Body or corpus [2”] and cervix uteri [1”].
(iii) In fetal life, cervix is considerably larger than the body
(iv) After parturition, the uterus nearly regains its usual size, weighing on an average the same as the virgin uterus, but its cavity is larger than in the nulliparous state.
(2) **Ovary** -
(i) The size of each adult ovary: 3 cm x 1.5 cm x 1 cm [uterus is about 2.5 times bigger (in every dimension) than each ovary]
(ii) The weight of each ovary in a non pregnant state: 2-3.5 gm. In pregnancy slightly heavier.

B. ML Aspects of Pregnancy

1. Capital sentence

If woman sentenced to death is pregnant, the High Court shall commute the sentence to imprisonment for life [**S.416, CrPC**].

2. Gestation period

(1) **Average period** – Normal gestation period from LMP is 280 days [or 10 times the intermenstrual period, which is 28 days]. Since ovulation and consequent fertilization occurs on the 14th day, the actual physical existence of the fetus within the womb is 266 days. Please compare with ***S.112, IEA*** below.
(2) **Maximum period** – Maximum period recorded in a reliable medical journal

is **359 days** [British Medical Journal].

(3) **Minimum period with viability** – A viable child means a child capable of independent existence from its mother. Generally accepted period of viability is **28 weeks** [**196 days**] from LMP. Babies born earlier may survive, but suffer from severe neurological deficits.

3. Posthumous child

It is a child born after the death of father, the mother having conceived by the said father. Legal issues involved are:

(1) Compensation case against mother for slander

(2) Inheritance of property

(3) Legitimacy.

4. Pregnancy without being aware of it [unconscious pregnancy]

(1) May occur very rarely.

(2) Still more rarely, pregnancy may progress to full term without the woman being aware of it, although she may be aware of the enlarging abdomen, which might be thought to be due to some disease.

5. Pseudocyesis

Pseudocyesis, ***false pregnancy*** or ***hysterical pregnancy*** is the appearance of clinical signs and symptoms associated with pregnancy when the female is not pregnant.

Salient features:

(1) **Etiology** – Seen in younger women who intensely desire children or women nearing menopause. Most women suffer from some form of psychological or hormonal disorder.

(2) **Signs and symptoms** – All or most are presumptive and probable signs.

(i) Amenorrhea and breast changes may be there

(ii) woman may even feel fetal movements

(iii) -Abdominal size – due to (a) abnormal deposition of fat (b) tympanites [excessive gas accumulation in the GIT] or (c) ascites

(iv) Pseudopregnancy may advance to full term and the woman may go into false labor pains. These labor pains may cease suddenly when the patient is told she is not pregnant.

(3) In case of doubt, clinical examination and ultrasound would reveal an empty uterus. False pregnancy is very common in animals [particularly dogs and mice]; in animals, it is called **pseudopregnancy**. Clinical signs seen are wt gain, mammary enlargement, lactation and maternal behaviour.

6. Determination of period of pregnancy

(1) By counting days since LMP

(2) From quickening [for date please see above]

(3) **McDonald's rule** - Applicable from 22-34 weeks age of gestation.

(i) Fetal age (in weeks) = fundal height in cm (measured from symphysis pubis to top of uterus) # $\frac{8}{7}$

(ii) Fetal age (in months) = fundal height in cm (measured from symphysis pubis to top of uterus) # $\frac{2}{7}$

(4) **Naegele's Rule** - Expected date of confinement = 1st day of LMP +9 months + 7 days.

7. Sex Selection

a. The Pre-Conception and Pre-Natal Diagnostic Techniques (Prohibition of Sex Selection) Act [PCPNDT], 1994

Salient features:

(1) It was enacted in **1994**, and subsequently amended in **2002**.

(2) **Aims:** (i) Pre-natal diagnostic techniques for determination of sex of fetus were prohibited. Aim was to prevent female feticide (ii) Advertisement of pre-natal diagnostic techniques for detection or determination of sex were prohibited (iii) Pre-natal diagnostic techniques for detection of genetic abnormalities were allowed.

(3) **Some important definitions** – Dealt with in Ch I. [A] **Diagnostic procedures and tests.** 3 are defined (i) "pre-natal diagnostic procedures" means all gynecological or obstetrical or medical procedures such as ultrasonography, fetoscopy, taking or removing samples of amniotic fluid, chorionic villi, blood or any other tissue or fluid of a man, or of a woman for being sent to a Genetic Laboratory or Genetic Clinic for conducting any type

of analysis or pre-natal diagnostic tests for selection of sex before or after conception [**S.2i**]. Genetic clinic is a place where such procedures are performed [**S.2d**]. It includes a vehicle where ultrasound machines, or other equipment capable of determining sex is kept. (ii) ”pre-natal diagnostic techniques” includes all pre-natal diagnostic procedures + prenatal diagnostic tests [**S.2j**]; (iii) ”pre-natal diagnostic test” means ultrasonography or any test or analysis of amniotic fluid, chorionic villi, blood or any tissue or fluid of a pregnant woman or conceptus conducted to detect genetic or metabolic disorders or chromosomal abnormalities or congenital anomalies or hemoglobinopathies or sex-linked diseases [**S.2k**]. Genetic laboratory is a place where such tests are performed [**S.2e**]. It includes a vehicle as mentioned above; [Taking of samples is “procedure”; analyzing it is “test”; both together are “techniques”]. Ultrasonography is the ONLY diagnostic test, which is included in ***both*** Prenatal diagnostic procedures and Prenatal diagnostic tests. [B] **Places** – 3 are defined. (i) Genetic Counselling Centre is a place which provides genetic counselling to patients [**S.2c**]. (ii) Genetic clinic and (iii) genetic laboratory are mentioned above. All 3 must be registered with the appropriate authority [**s18**]. In case of any violation, the registration will be cancelled [**S.20**]. If the owner of these places want to institute an appeal against the order, it must be done with 30 days to the State govt [if authority was state level] and to the Central govt [if authority was center level] [**S.21**].

(4) **Regulation of Genetic Counselling centers, genetic laboratories and genetic clinics** –Dealt with in Ch II [S.3 – only one section in this chapter]. (i) No person can conduct a sex selection [**S.3A**] (ii) No person can sell any ultrasound machine or imaging machine or scanner or any other equipment capable of determining sex of fetus to any person or clinic etc not registered under the Act [**S.3B**].

(5) **Regulation of pre-natal diagnostic techniques** [**PNDT**]–Dealt with in Ch III [**S.4-6**]. (A) Prenatal Diagnostic Techniques can be *conducted only for* (i) **C**hromosomal abnormalities (ii) **C**ongenital Abnormalities (iii) **G**enetic metabolic diseases (iv) **H**emoglobinopathies (v) **S**ex-linked genetic diseases (vi) Any other abnormalities or diseases as may be specified by the Central Supervisory Board [**S.4(2)**]. [**Memory aid 2- C^{2}GHS**]. [*These are indications of PNDT*]. (B) Any one [or more] of the following c*onditions must be fulfilled* before the techniques can be carried out (i) **A**ge of the pregnant woman >35 y

(ii) pregnant woman has undergone ≥2 **Sp**ontaneous abortions or fetal loss (iii) pregnant woman had been exposed to potentially **T**eratogenic agents such as drugs, radiation, infections or chemicals (iv) pregnant woman has a **F**amily history of mental retardation or physical deformities such as spasticity or any other genetic disease, and (v) any other condition as may be specified by the Central Supervisory Board. The person undertaking the procedure must record the fulfilling condition in writing [**S.4(3)**]. [**Memory aid 3 - A Special ToFfee**]. [*Since these are preconditions, ideally they should have come before the indications.*] (C) **Informed consent** – Prenatal diagnostic procedures can only be conducted after a written informed consent [**ch 2**]. A copy of the written consent is to be given to pregnant woman [**S.5(1)**]. (D) **Prohibition of communicating sex of fetus** - Person conducting prenatal diagnostic procedure cannot communicate sex of fetus to the pregnant woman concerned or any other person by any means [**S.5(2)**]. (E) **Determination of sex prohibited** - No person or clinic etc can determine the sex of a fetus or cause selection of sex before or after conception [**S.6**].

(6) **Penalties:** (i) on Advertisement – of any of sex selection or sex determination techniques; 3 y + `10,000 [**S.22**] (ii) on Sex selection or sex determination - (a) To doctor – 1st conviction"3 y + `10,000. Subsequent convictions"5 y + `50,000 [**S.23(1)**]. On conviction, name will be removed from medical register for 5 y for 1st offence and permanently [professional death sentence] on 2nd offence [**S.23(2)**] (b) To patient - 3 y + `50,000 for 1st offence and 5 y + `100,000 for 2nd offence [**S.23(3)**]. If the woman was compelled she will not be punished [**S.23(4)**], but the person compelling her would be.

8. Superfecundation

Superfecundation is fertilization of two or more ova from the same menstrual cycle in separate copulatory events.

Salient features:

(1) **Monopaternal superfecundation** [**MS**] – Multiple copulatory events are performed by the same partner. When multiple pregnancies result from a **single copulatory event**, it is known as twins, triplets etc. There is no scientific way to differentiate between monopaternal superfecundation and twinning.

(2) **Multipaternal [Heteropaternal] superfecundation** [**HS**]– Multiple copulatory events are performed by different partners.

(i) If there are two partners [most common], it is known as **dipaternal superfecundation**. Depending on number of partners, there can be tripaternal, tetrapaternal, pentapaternal superfecundation etc. This is common in animals but uncommon in man due to social taboos. The frequency of HS among dizygotic twins whose parents were involved in paternity suits is **2.4%**. Before the era of DNA profiling, they could be detected only by blood examination, and before blood examination era only by external appearance, eg one black and one white baby born at birth clearly indicated a case of **superfecundation**.

(3) **Age of fetuses** – is same. Development is parallel but not equal. Depends upon relative blood supplies from separately formed placentae.

(4) **Fate** – one fetus may end up as fetus papyraceous or may end up in vanishing twin syndrome [please see below].

(5) **Incidence of multiple pregnancies:**

(i) Determined by **Hellin's law** [syn, Hellin-Zeleny law] – 1 in 89 pregnancies ends in the birth of twins; one in 89^2 [7921], of triplets; one in 89^3 [704,969], of quadruplets.

(ii) Of all twin pregnancies, 70% are binovular twins.

9. Superfetation

Superfetation is fertilization of an ovum in the presence of an existing pregnancy.

Salient features:

(1) **Preconditions** – Septate or double uterus facilitates superfetation, but is not an absolute necessity.

(2) **Age of fetuses** – is different. Difference varies from 1-3 months

(3) **Development of fetuses** – different. Elder fetus can be determined by rule of Haase [**ch 3**].

(4) **Parturition** – May be single or multiple. In a single parturition, two or more fetuses in different stages of development may be born at the same time. In multiple parturitions [very rare], full term fetuses are born at different periods. An alternative explanation to multiple parturitions is that both fetuses started life together as twins, but one of them did not develop normally either due to

lack of proper blood supply, or because of some other cause. Once the "dominating" twin is born, the other twin starts receiving proper blood supply, matures in a few months and is born at a subsequent parturition.

(5) **Fetus papyraceus** [syn, Fetus compressus] -

(i) Def - A flattened, partly mummified twin fetus that dies early in pregnancy and remains in the uterus until the term is completed.

(ii) Frequently discovered - during an ultrasound examination.

(iii) Fate - The fetus is compressed between the uterine wall and the amniotic sac of the living twin. It is flattened like a paper or parchment and is mostly mummified. May be incorporated into the placenta of the surviving twin and, if large enough, visible at delivery.

(iv) Age at death – can be determined by its **CR length**.

(v) Occurrence - Common in superfetation, but may be seen in superfecundation also.

(6) **Vanishing twin syndrome** -

(i) Def - Multifetal gestation with subsequent disappearance of one or more fetuses. First described by **Stoeckel** in **1945**.

(ii) Features - There may be (a) complete reabsorption of a fetus, (b) formation of a fetus papyraceus, or (c) development of a subtle abnormality on the placenta such as a cyst, subchorionic fibrin, or amorphous material.

(iii) Causes - (a) chromosomal abnormalities in the vanishing twin, (b) improper cord implantation.

Table 6: Differences between superfecundation, superfetation and twins

S.No.	Criteria	Superfecundation	Superfetation	Twins
1.	Fathers	May or may not be same	May or may not be same	Always same
2.	Copulatory events	Multiple	Multiple	Single
3.	Age of fetuses	Same	Different	Same

III. LEGITIMACY AND PATERNITY

A child is said to be ***legitimate***, if it is born (i) during the continuation of a legal marriage, or (ii) within 280 days of death of husband or (iii) within 280 days after the dissolution of marriage by divorce [***S.112, IEA***].

Salient features:

(1) A legitimate child is entitled to inherit the property of his parents.

(2) The child is **illegitimate** (**bastard**) if the alleged father (i) is under the age of puberty (ii) is physically incapable to beget children [eg due to illness, congenital or acquired deformities, azoospermia, vasectomy etc] (iii) did not have access to his wife when the child was begotten (iv) displays a blood group or DNA profile, which is not compatible with that of child.

(3) **Posthumous AIH** - Child will be considered legitimate even if born **>280 days** after death of husband [**ch 23**].

A. When Does the Question of Legitimacy Arise?

The question of legitimacy arises in following situations:

1. Inheritance

Only a legitimate child can inherit the property of his father.

2. Affiliation cases

An ***affiliation*** (L filius, son) ***case*** refers to a legal dispute in which the paternity of an illegitimate child is sought to be determined, and the obligation of contributing to its support enforced.

Salient features:

(1) In India, affiliation cases are decided by **S.125, CrPC**. If paternity is proved, a First Class Magistrate may order the father to pay a monthly allowance of any sum for the maintenance of such child.

(2) **Medical examination in affiliation cases** – Same as that in suppositious children [please see below].

3. Suppositious children

A ***suppositious*** (***fictitious***) ***child*** is one produced by a woman who claims it as her own whereas in actual fact it is not her child.

Salient features:

(1) Difference from an affiliation case [please see **Table 7**]

(2) **Common situations where suppositious children are produced**

(i) A woman may feign pregnancy and delivery and later produce a living child as her own

(ii) A woman may substitute a male child for a female child

(iii) A woman may substitute a living child for an aborted child

(3) **Reasons for producing a suppositious child** -

(i) Blackmail

(ii) Bringing a charge of breech of promise of marriage against a man

(iii) Claiming property

(iv) Obtaining money

(4) **Medical examination in suppositious children** -

(i) Examine for signs and pregnancy and delivery. (a) If absent, the child is suppositious (b) if present, date of delivery should be calculated from signs. It must be compatible with the age of child produced.

(ii) Blood group examination of mother, child and alleged father

(iii) If blood examination proved inconclusive, DNA profiling. Please see also **ch 29**.

Table 7: Differences between an affiliation case and a case involving suppositious child

S.No	Criteria	Affiliation case	Suppositious child
1.	Child	Belongs to the mother	Does not belong to the mother. Fictitious child [stolen or taken from a colluding friend]
2.	Alleged father	Denies being the father	Denies being the father
3.	Relief sought by mother	Money for maintenance of child	Property, money or marriage
4.	Signs of pregnancy and delivery	Shall be present and their age shall be compatible with the age of child produced	Either not present, or if present, their age will not be compatible with the age of child produced
5.	Legal outcome	Maintenance awarded by a First Class Magistrate u/s 125 CrPC	Child returned to original parents. Mother punished for cheating and fraud

4. Disputed paternity

Paternity is said to be ***disputed***, when the alleged father denies being the father of a child. Paternity is disputed in suppositious children and affiliation cases [also see **ch 23**]. Please see ch 29 for solving disputed paternity.

5. Posthumous births

A birth is said to be posthumous when the alleged father is dead. If the family of the dead man refuses to accept the child, the case becomes similar to that of disputed paternity.

6. Wrongful life

It is a variation of wrongful life suit [**ch 23**]. Here a normal, healthy child sues his parents for his illegitimacy. In Zepeda v. Zepeda, 41 Ill. App. 2d 240, 190 N.E.2d 849, 851 (1963), a healthy illegitimate child brought an action against his father, claiming injury by virtue of his illegitimate status. The court rejected the claim, one of its reasonings being fear of a flood of litigation.

IV. DELIVERY

Delivery is natural or assisted expulsion of the child at full term.

A. Signs of Recent Delivery in the Living

Signs given below are more typical of a full term delivery than of a premature delivery.

1. General indisposition

(1) Woman is pale, exhausted and ill looking with

(2) -pulse and slight fever for first 2-3 days.

2. Breast changes

(1) **Breasts:**

(i) Full, enlarged, tender with a knotty feeling

(ii) Superficial veins prominent

(iii) Colostrum or milk may be expressed by compression

(2) **Areola:** dark, Montgomery tubercles [areolar glands] present

(3) **Nipples** - enlarged

3. Abdominal changes

Abdominal walls are (i) pendulous, (ii) wrinkled and show (iii) linea albicantes [for details, please see above] and (iv) linea nigra (v) **In case of c/s -** Abdomen shall show incision or scar.

4. Perineal changes

(1) Sometimes lacerated

(2) From age of laceration, date of delivery may be calculated.

5. Uterus

a. Morphology

(1) **Immediately after delivery** –

(i) Feel - Contracted and retracted body of uterus feels like hard muscular tumor

(ii) Upper border – lies 3 cm below umbilicus

(iii) Diminishes in size – at a rate of 1.5 cm/day

(2) **1wk -** Upper border – Midway between umbilicus and pubis

(3) **2wks -** Descends into pelvis [becomes a pelvic organ]

(4) **9wks** – Returns to normal condition.

Table 8: Uterus and placental site dimensions after delivery

S.No	Time after delivery	Dimensions [cm]	Weight [g]	Placental site diameter [cm]
1.	Immediate	20 # 15 # 5	1000g	15
2.	1st week	14 # 8 # 4	500	4
3.	2nd week	12 # 7 # 3	350	2.5
4.	6 weeks [returns to normal]	10 # 6 # 2.5	80-120 g	1-2

b. Contractions

Intermittent uterine contractions present for first 4-5 days.

6. Cervix

(1) **Immediately after delivery** -

(i) Cervix soft and dilated

(ii) Edges torn and lacerated transversely

(2) **24 hours** -

(i) Internal os begins to close

(ii) External os is soft and patent and admits 2 fingers

(3) **End of 1 week** – one finger is admitted with difficulty

(4) **End of 2 weeks** – external os is closed.

7. Labia

(1) Bruised (2) Swollen and tender (3) lacerated [sometimes].

8. Vagina

(1) Smooth walled. Rugae begin to appear by 3rd wk.

(2) Capacious and relaxed

(3) Shows recent tears, which heal by 7th day.

9. Lochia

Lochia [Gk. Lokhos, childbirth] is a discharge from uterus which lasts for 2-3 weeks.

Salient features:

(1) **Stages** – 3 stages are seen. All have peculiar, sour odor.

(i) Lochia rubra – (a) Duration - Lasts first 4-5 days. (b) Nature - bloody (c) Color - bright red (d) Quantity discharged – 250 mL [total] (e) Contents - Blood clots, decidual cells, fetal membranes [shreds], lanugo, meconium, vernix caseosa

(ii) Lochia serosa – (a) Duration - lasts 4 days [from 6^{th} -9^{th} day of delivery]. (b) Nature – serous, watery; (c) color – pale (d) Contents – Blood cells [less RBCs, but more WBCs], Cervical mucus, Microorganisms [anaerobic Streptococci, Staphylococci], Wound exudates

(iii) Lochia alba - (a) Duration - from 10^{th} -21^{st} day. (b) Nature – scanty, thick, turbid; (c) color – initially yellowish gray; later whitish. (d) contents – cholesterol crystals, decidual cells, fatty and granular epithelial cells, leucocytes, microorganisms, mucus,

10. Lab tests

Blood and urine continue giving +ve pregnancy tests for 7-10 days

B. Signs of Recent Delivery in the Dead

(1) All local signs mentioned above [except essentially "live" signs eg intermittent contractions of uterus]

(2) **Uterus** [Table 8]- Additional changes are as follows

(i) Immediately after delivery – (a) flabby. (b) Cavity – almost obliterated by apposition of anterior and posterior walls

(ii) 2-3 days after delivery – (a) Length – 17.5 cm (b) Breadth – 10 cm

(3) **Placental site** - Immediately after delivery – Placental site appears as an irregular, nodular, elevated area. Covered with clotted blood, lymph and portions of decidua. Size

(4) **Peritoneum covering of lower uterus** – Immediate till few days – arranged in folds. These folds disappear after a few days.

(5) **Ovaries, fallopian tubes** –

(i) Congested immediately after delivery. Become normal if a few days.

(ii) Corpus luteum – quite large. Found in one of the ovaries

(6) **Urinary bladder** – Immediately after delivery – edema and hyperemia. Sometimes submucous extravasation of blood.

C. Signs of Remote Delivery in the Living

Generally a single full term delivery leaves permanent signs on the body of mother. A premature delivery may show little or rarely no marks.

(1) **Abdomen** – abdominal wall is lax. Shows multiple white scars on lateral aspects. Linear nigra present

(2) **Breasts** –

(i) Lax, pendulous, soft, wrinkled [if baby was nursed]. Show linea albicantes.

(ii) Nipples – enlarged

(iii) Areolae – dark

(iv) Montgomery tubercles - present

(3) **Vulva** – partially gaping, labia do not oppose orifice completely

(4) **Vagina** – partially open, vaginal rugae are absent, walls are relaxed

(5) **Perineum** – may show a scar of an old tear

(6) **Cervix** - **Table 9**.

D. Signs of Remote Delivery in the Dead

Uterus and cervix – **Table 9**.

Table 9: Differences in the uterus of nulliparous and Parous women

S.No	Feature	Nulliparous uterus	Parous uterus
1.	Size	Smaller, thinner. 7.5 # 5.0 # 2.5 cm [3″ # 2″ # 1″]	Larger, thicker 10 # 6 # 2.5 cm [4″ # 2.5″ # 1″]
2.	Weight	40-60 g	80-120 g
3.	Length	Body is of same length as cervix	Body is twice the length of cervix
4.	Fundus	1. Top of fundus is less convex and 2. on the same level as the line of broad ligament	1. Top of fundus is convex and 2. on a higher level than the line of broad ligament
5.	Cavity	1. Walls are convex from inside 2. cavity is smaller and triangular from inside	1. Walls are concave from inside 2. cavity is wider and rounded from inside
6.	External os	Rounded. Appears as a small hollow in the center of cervix. Orifice closed	Transverse, irregular, fissured. Orifice admits tip of finger
7.	Internal os	Circular, Well defined	Not well defined
8.	Cervix	Regular, no scars. Mucus membrane smooth and intact	Irregular. Edges show scars.

E. ML Aspects of Delivery

(1) **Abortion** – It may be alleged that the woman aborted illegally, while it might have been a premature delivery

(2) **Amniotic fluid embolism** - Please see **ch 26**.

(3) **Blackmail** – A lady may produce a **suppositious child** and claim it to be fathered by a man whom she blackmails [please see above under "disputed paternity"]. Signs of recent delivery must be present on the woman. The DNA of child must match with that of mother and claimed father.

(4) **Chastity** – A person may claim that his wife is not chaste and has had sexual relations before, and has delivered a child fathered by someone else. Signs of remote delivery must be present on the female.

(5) **Concealment of birth** – Woman may be accused to concealment of birth by secret disposal of dead body (u/s 318, IPC), while in fact, she never delivered.

(6) **Divorce** – (a) After marriage - Delivery of a child fathered by someone other than the husband after marriage is a ground for divorce. (b) Before marriage - Delivery before marriage is not a ground for divorce even if fathered by someone else than husband. (c) Pregnant at the time of marriage - If at the time of marriage the woman was pregnant by someone other than the husband, it is a voidable marriage. [please see ch 23 – impotence and sterility].

(7) **Feigned delivery** – Woman may feign delivery in order to get leave from office, or to escape capital sentence.

(8) **Infanticide** – woman may have been accused of infanticide, while in fact she never delivered.

(9) **Legitimacy** – A child is legitimate, if he is delivered within 280 days after the dissolution of marriage (S.112, IEA).

(10) **Nullity of marriage** – Delivery of child before marriage is not a ground for nullity of marriage.

(11) **Precipitate labor** - please see **ch 27**.

(12) **Water births** - Water birth is a method of delivery, which involves partial immersion of mother and delivery of child in warm water. Proponents believe that this method is safe and provides many benefits for both mother and infant, including pain relief and a less traumatic birth experience for the baby. However, critics argue that the procedure introduces unnecessary risks to the

infant such as infection and water inhalation.

25. Sexual Offences

I. INTRODUCTION

Sexual offences are criminal forms of human sexual behavior. A **sex offender** is one who commits a sexual offence.

II. CLASSIFICATION

Sexual offences may be classified as below:

(I) **Natural sexual offences** (Offences involving natural penile-vaginal penetration) (A) Violent – Done without consent e.g. **rape** (B) Non violent – done with consent e.g. **incest**, **adultery** (II) **Unnatural sexual offences** (Offences involving penetrations other than penile-vaginal)

(A) Sodomy (B) Buccal coitus (C) Tribadism

(III) **Sexual perversions** (sexual psychiatric disorders. Classified by DSM-5 as paraphilic disorders) **(A) Pedophilia** (sexual intercourse with children); **(B) Exhibitionism; (C) Voyeurism; (D) Sadism; (E) Masochism; (F) Frotteurism; (G) Necrophilia; (H) Undinism; (I) Miscellaneous [please see below]**

III. RAPE

Rape (Latin rapio; seize, snatch) is an assault by a person involving sexual intercourse with another person without that person's consent. **Historical** - Instances of rape occur in **Greek** and **Roman** mythology. In Greek mythology, **Phoebe** and **Hilaeira**, the daughters of **Leucippus** were forcibly abducted and raped by the twin brothers **Castor** and **Pollux** [**Fig. 25.1**]. In **Roman mythology**, the rape of **Lucretia** was a starting point of events that led to overthrow of Roman Monarchy and establishment of Roman Republic. As a

direct result of rape, **Lucretia committed suicide**.

Fig. 25.1: The Rape of the Daughters of Leucippus" [painted around 1617 AD].

A. Legal Provisions Regarding Rape in India

1. Provisions in IPC

a. S.166A, IPC

If a police officer fails to record FIR **u/s154(1) CrPC** of a victim of **rape** [and also of vitriolage (**S.326A, 326B IPC**), outraging the modesty of woman (**S.354 IPC, S.509, IPC**), disrobing a woman (**S.354B, IPC**), trafficking a person (**S.370, IPC**) and exploitation of a trafficked person (**S.370A, IPC**)], he shall be fined and given rigorous imprisonment for a minimum of **6 months** and **up to 2 years**.

b. S.166B, IPC

If any hospital, whether Govt or private does not provide immediate first-aid or treatment **free of cost** to **any victim of rape** [or **vitriolage**] **u/s357C CrPC** [Pl see below], the in-charge of hospital would get a punishment for **1 y** or **fine** or both [Same punishment is also given if free medical aid is not provided to victims of vitriolage (S326A IPC). Pl see ch **11**].

c. S.228A, IPC

Whoever prints or publishes the name or any matter which may make known the identity of victim of rape shall be punished with imprisonment of up to 2 years and fine. Publication is possible only under following circumstances (i) a police officer publishes name for investigation, or (ii) victim (>18 years) authorizes in writing, or (iii) if victim is dead, minor or of unsound mind, next of kin has authorized in writing.

d. S.327, 329 (causing hurt, Grievous hurt, to constrain to an illegal act)

e. S.328 (Causing hurt by means of poison, with intent to commit an offence – Drug Facilitated Sexual Assault)

Please see **ch 31**.

f. S.375, IPC (Definition of rape)

The definition of rape was changed radically after the infamous **Delhi gang rape case** of **2012**. A committee was made under the chairmanship of former Chief Justice **JS Verma** with the task of reforming and invigorating anti-rape law. The comprehensive **630 page report** was completed in **29 days** and lauded both nationally and internationally. This eventually led to the passing of The Criminal Law (Amendment) Act, 2013 [CLAA 2013]. It came into force on **Feb 3, 2013**. Currently the definition is as follows:

Definition of rape - A man is said to commit “rape” if he:

(a) penetrates his **penis**, to any extent, into the **vagina**, **mouth**, **urethra** or **anus**

of a woman or makes her to do so with him or any other person; or (b) **inserts**, to any extent, any **object** or a **part of the body**, not being the penis, into the vagina, the urethra or anus of a woman or makes her to do so with him or any other person; or (c) **manipulates** any part of the body of a woman **so as to cause penetration** into the **vagina**, **urethra**, **anus** or **any part of body of such woman** or makes her to do so with him or any other person; or (d) **applies** his mouth to the vagina, anus, urethra of a woman or makes her to do so with him or any other person, under the circumstances falling under any of the following **seven** descriptions:

Memory Aid 1: 4 acts required for rape

1. **4 acts** required for rape can be remembered by **PIMA**

(a) **P**enetrates [**P**enis] (b) **I**nserts [any object, part of body] (c) **M**anipulates [woman] (d) **A**pplies [mouth]

2. **4 places** where insertion is mentioned [from top] – **mouth, urethra, vagina, anus. 4 acts and 4 penetration places** give a total of **16 possible combinations** **[Table 1]**

3. **Mouth** specifically mentioned only in (a). Not included in (b) and (d). Implied in (c). Understandable why it is not included in (d), because that act would amount to just kissing – a less serious offence than rape.

4. In (c) assailant is not manipulating the inserting object, but rather the object (woman) where insertion occurs.

Table 1: 4 possible acts and 4 possible penetration places give a total of 16 possible combinations. (1) depicts orifices penetrated; (2) depicts action on the part of assailant. ✓ in a cell means that the act constitutes rape; ☒ in cell means that the act is not rape. 14 out of 16 possible combinations are rape.

(2) \ (1)	mouth	urethra	vagina	anus
Penetrates [penis]	✓	✓	✓	✓
Inserts [any object or part of body except penis]	☒ **ex-** inserting, say a finger in mouth, is not rape	✓	✓	✓
Manipulates [woman] so as to cause penetration [what should penetrate is not mentioned]	✓	✓	✓	✓
Applies [mouth]	☒ - May amount only to kissing	✓	✓	✓

(1) Against her will
(2) Without her consent
(3) With her consent, when her consent has been obtained (a) by putting her or (b) any person in whom she is interested in fear of death or of hurt
(4) With her consent, when the man knows that he is not her husband, and that

her consent is given because she believes that he is another man to whom she is or believes herself to be lawfully married

(5) With her consent, when, at the time of giving such consent, (a) by reason of unsoundness of mind or (b) intoxication or (c) the administration by him personally or through another of any stupefying or unwholesome substance, she is unable to understand the nature and consequences of that to which she gives consent

(6) With or without her consent, when she is under **18 years of age** [**statutory rape**]

(7) When she is unable to communicate consent [**Note** - The age of consent was changed from **16 y** to **18 y** by CLAA 2013. Condition

(8) was also added by the same amendment].

Explanation 1: For the purposes of this section, "**vagina**" shall also include **labia majora** [added by CLAA 2013. This effectively means that even touching of penis with vulva amounts to rape].

Explanation 2: **Consent** means an unequivocal voluntary agreement when the woman by words, gestures or any form of verbal or non-verbal communication, communicates willingness to participate in the specific sexual act, provided that a woman who does not physically resist to the act of penetration shall not by the reason only of that fact, be regarded as consenting to the sexual activity [added by CLAA 2013].

Exception 1: A medical procedure or intervention shall not constitute rape. [added by CLAA 2013].

Exception 2: Sexual intercourse by a man with his own wife (even without her consent) is not rape, if she is > 15 years of age. If she is < 15 years, sexual intercourse with or without consent is rape (**marital rape**).

Memory Aid 2: Explanations and Exceptions to S.375 IPC

Vice **C**hancellor **M**edicates **W**ife

Explanations:

Vagina includes labia majora

Consent means an **unequivocal voluntary agreement**

Exceptions:

Medical **procedure** is not rape

Wife **>15 y** cannot be legally raped by husband

i. Comments

(a) **penetrates** his **penis**, to any extent, into the **vagina**, **mouth**, **urethra** or **anus** of a woman or makes her to do so with him or any other person. **Ex** - (1) **“Z”** draws a loaded gun and threatens woman **“W”** to kill her if she does not allow penetration of penis of man **“M”** into her vagina [or mouth, urethra or anus] " under fear of injury or death, she allows this " “Z” has raped “W”. “M” has not raped “W”, if he was under similar threat. However if “Z” and “M” were working in collusion, then both have raped her. (b) **inserts**, to any extent, any **object** or a **part of the body**, not being the penis, into the vagina, the urethra or anus of a woman or makes her to do so with him or any other person. **Ex** - (1) **“Z” inserts his finger** into the vagina [or urethra or anus] of a woman **“W”** " Z has raped W. (2) **“Z” inserts a pencil** into the vagina [or urethra or anus] of a woman **“W”** " Z has raped W. Earlier to 2013 amendments, both these situations were not rape; Z could at most be prosecuted u/s 354 IPC. (3) **“Z” inserts his finger** or **pencil** into the mouth of a woman **“W”** " Z has not raped W, as mouth has been left out of this clause. (4) **“Z”, a medical doctor, after taking informed consent from** woman **“W”** performs a PV examination on her " Her husband alleges rape " Z has not raped W. **Exception 1** excludes medical procedures from definitions of rape. (c) **manipulates** any part of the body of a woman **so as to cause penetration** into the **vagina**, **urethra**, **anus** or **any part of body of such woman** or makes her to do so with him or any other person. **Ex – “Z”** pushes a woman **“W”** from behind [or causes her to trip over] so that she falls " A stick lying on the ground penetrates her vagina [or urethra, anus or any part of her body] " **“Z”** has raped **“W”. This is a very far reaching clause** and it remains to be seen how courts interpret this clause. Importantly, here “Z” **does not manipulate the inserting object**; but **rather the woman** so that insertion occurs. A seriously doubtful portion of the clause is “**any part of body of such woman**”. If in the above example, the stick penetrates her **eye** [or **ear**, **mouth** etc], yet it would be rape. This obviously is absurd, and quite probably courts would not interpret such situations to be rape. (d) **applies** his mouth to the vagina, anus, urethra of a woman or makes her to do so with him or any other person. **Ex** - (1) **“Z” licks** the vagina [or urethra or anus] of a woman **“W”** " **Z** has raped **W** (2) **“Z”** threatens woman **“W”** to allow man “**M**” to **lick** her vagina [or urethra or anus] " Under fear of injury or death, she does so " **Z** has raped **W. Situation regarding M’s culpability is same as above [“penetrates” clause].** (3) **“Z”** forcibly applies his mouth over the mouth of a woman **“W”** " ***Z***

has not raped ***W***, as mouth is not included in this clause. **Z** can however be prosecuted **u/s 354 IPC**.

ii. Difference between will and consent

(1) **Will and consent** - "***Will***" is a psychological desire; "***consent***" is a legal concept [pl see **S.90, IPC** (**ch 2**)]. Illustrations

(i) Mother tells child to eat spinach"Child says "no" because he does not like the taste [he has no "will" or desire to eat]"Mother asks him to go out of house as punishment"Not wanting to take punishment, child reluctantly agrees"He has consented for eating spinach, but the consent is taken by inducing "fear of injury" so it is invalid. Child eats spinach, but it is against his will and without his consent

(ii) Mother tells child to eat spinach"Child says "no" [as above]"Mother says he will not grow well if he does not eat spinach"child eats spinach because he wants to grow, but he eats unwillingly. Consent is not taken by fear of injury, or under a misconception of fact, so it is valid. Will is absent, consent is present. ["Medicine to an ailing child" may be substituted in place of spinach]

(iii) Mother tells child to eat chocolate"Child loves chocolates. He eats readily. Both "will" and "consent" are present.

(iv) Teacher asks student to attend his class"student does not want to attend, because class is boring"teacher says he will not let him sit in examinations if his attendance is short"student attends class reluctantly "consent not valid, as obtained by fear. Both ***will*** and ***consent*** are absent.

(2) **When both will and consent are absent in rape** – Most common situation. Example: Accused seizes woman in bushes and has forcible intercourse with her.

iii. Explanations of S.375, IPC

(1) **Against her will** - Example: teacher promises to pass weak student, if she has sex with him. She agrees "unwillingly". Consent present [because all requirements of **S.90, IPC** (**ch 2**) are fulfilled]. But will is absent"Rape.

(2) **Without her consent** - Example: doctor says to illiterate patient that sexual intercourse with him is the only treatment available. She agrees willingly [because she wants treatment. Will is additionally indicated because she may even visit his clinic daily for this "treatment" and may even pay doctor for it]. Will present; Consent absent [because taken "under a misconception of

fact”]"Rape.

(3) **With her consent, when her consent has been obtained (a) by putting her or (b) any person in whom she is interested in fear of death or of hurt** - Man tells woman he would maim her if she does not have intercourse with her"She agrees"Rape.

(4) **With her consent, when the man knows that he is not her husband, and that her consent is given because she believes that he is another man to whom she is or believes herself to be lawfully married** - (a) Man (M) enters friend (F’s) house to meet him" House is in dark" F’s wife thinks F has come" offers intercourse"M agrees"both have intercourse (woman even assisting him in the act) "Rape [**NB**. Here man does not deceive woman. She is mistaken on her own. A similar situation may occur in case of a blind woman. If man deceives woman, he is liable **u/s 493 IPC** for which punishment is greater (10y)].

(5) **With her consent, when, at the time of giving such consent, (a) by reason of unsoundness of mind or (b) intoxication or (c) the administration by him personally or through another of any stupefying or unwholesome substance, she is unable to understand the nature and consequences of that to which she gives consent** - Man asks insane girl to have sex with him. She agrees"Rape.

(6) **With or without her consent, when she is under 18 years of age** - Girl suggests to boy friend that they should have sex"He agrees"Girl is **<18** years"Rape.

(7) **When she is unable to communicate consent -** Boy has had voluntary sex act with his girl friend several times " Thus consent is taken for granted by him One day he finds her sleeping [or unconscious] has intercourse with her presuming consent rape, because she was unable to communicated her consent at this particular time.

Explanation 1: For the purposes of this section, “**vagina**” shall also include **labia majora** [meaning of “vagina”] - Man seizes woman in bushes and tries to have intercourse with her forcibly. As soon as penis touches vulva, he loses erection because of fear"Rape. [slightest penetration – even touching of penis with vulva is rape. Ejaculation not necessary. Pl also see below under Medicolegal questions relating to rape" Penetration level sufficient to constitute rape].

Explanation 2: Meaning of consent - Consent should be explicit and not implicit.

Ex - Man “M” asks a woman “W” in a lonely place if he can have sex with her"She keeps quiet" M thinks her silence is an indication of consent"he has sex with her during which period also she is quiet"This is rape

Exception 1: A medical procedure or intervention shall not constitute rape - Woman comes to a doctor for antenatal check-up. After taking informed consent, the doctor conducts a PV examination. Since it is a medical procedure, it is not rape, although insertion of finger in vagina is rape.

Exception 2: **Sexual intercourse by a man with his own wife (even without her consent) is not rape, if she is > 15 years of age. If she is < 15 years, sexual intercourse with or without consent is rape** - (a) man asks legally married wife to have sex" she says no"He ties her to bed and has forcible sexual intercourse with her"**NOT rape** [In India, woman is deemed to have given **unconditional consent** for sexual intercourse to her husband at the time of marriage. This is not so in several western countries, where both husband and wife must take consent from the other before having sex] (b) Wife <15 years" Requests husband to have sex with her" they have sex" Rape.

g. S.376(1) and S.376(2), IPC (Punishment for rape)

S.376 (1) : Punishment for rape: Whoever commits rape shall be punished with imprisonment [simple or rigorous (with hard labor)] for **more than 7 years** (up to life) +fine.

S.376 (2) : Added by The Criminal Law (Amendment) Act 1983, and modified further by The Criminal Law (Amendment) Act 2013 [CLAA 2013]. Defines and punishes **14 serious forms** of rape. Punishment in all **14 cases** is **rigorous imprisonment for more than 10 years and up to imprisonment for life** [which shall mean imprisonment for the remainder of that person’s natural life]**+Fine**. The forms are [**with memory aids**]: (a) a **police officer** commits rape (i) within the limits of his jurisdiction, or (ii) in the premises of any police station, or (iii) on a woman in his custody. (b) a **public servant [Memory aid 3]**, takes advantage of his official position and commits rape on a woman working under him. (c) a member of the **armed forces [Memory aid 4]** deployed in an area by the Central or a State Government commits rape in such area. (d) a person on the management or on the staff of a **jail**, **remand home** or **other place of custody**, or of **a women’s or children’s institution [Memory aid 5]**, commits rape on any inmate of such jail, remand home, place or institution

[**custodial rape**]. (e) a person on the management or on the staff of a **hospital** commits rape on a woman in that hospital. (f) a **relative, guardian** or **teacher** of, or a person in a **position of trust** or **authority** towards the woman, commits rape on such woman. **F**amily or **f**ather figure **[Memory aid 6]**. (g) a person commits rape during **communal** or **sectarian violence**. (h) a person commits rape on a woman knowing her to be **pregnant**. (i) a person commits rape on a woman **<16 y** [before CLAA 2013, this age was **12 y**]. (j) a person commits rape, on a woman **incapable of giving consent**. (k) a person **in a position of control or dominance over a woman**, commits rape on such woman. - **K**ontrol **[Memory aid 7]**. (l) a person commits rape on a woman suffering from **mental** or **physical disability. L**unatic **[Memory aid 8**. (m) a person while committing rape **causes grievous bodily harm** or **maims** or **disfigures** or **endangers the life of a woman [Memory aid 9]**. (n) a person commits rape **repeatedly** on the **same woman**. **N**umber of times **[Memory aid 10]**.

S.376 A, B, C, D, E

CLAA 2013 has modified **S.376 A,B,C** and **D** and has added **S.376E**, which did not exist earlier.

(1) **S.376A, IPC - Punishment for causing death or resulting in persistent vegetative state [PVS] of victim** - Whoever, commits an offence u/s 376(1) and 376(2) and in the course of such commission inflicts an injury which causes the death of the woman or causes the woman to be in PVS, shall be punished with **minimum rigorous imprisonment** of **20 y, which** may extend to imprisonment for life [which shall mean imprisonment for the remainder of that person's natural life] or with **death**. **Memory aid 11 - A**runa Shanbaug case.

(2) **S.376B, IPC - Sexual intercourse by husband upon his wife during separation** - Wife living separately from husband (i) under a **decree of separation** [**u/s 10** of **Hindu Marriage Act, 1955**, wife may request court to allow her to live separately from husband] or (ii) otherwise. In either case, husband goes to her premises and has intercourse **without her consent**"punishment is imprisonment (simple or rigorous) of >2 yrs; may extend to 7 y+ fine. **Memory aid 12 - B**ride.

(3) **S.376C, IPC - Sexual intercourse by a person in authority** - Whoever, being (a) in a position of authority or in a **fiduciary relationship** [Pl see **ch 2**"Duties of a medical practitioner"Fiduciary duty]; or (b) a public servant; or

(c) superintendent or manager of a jail, remand home or other place of custody established by or under any law for the time being in force, or a women's or children's institution; or (d) on the management of a hospital or being on the staff of a hospital, abuses such position or fiduciary relationship to induce or seduce any woman either in his custody or under his charge or present in the premises to have sexual intercourse with him, such sexual intercourse not amounting to the offence of rape, shall be punished with **>5 y** imprisonment of either description; may extend to **10 y**, and shall also be liable to fine. **Reasoning**"It is easy to obtain consent from woman, because these are positions of authority. **Memory aid 13** - **C**ustodial; **C**ontrol.

(4) **376D, IPC - Gang rape** - Where a woman is raped by one or more persons constituting a group or acting in furtherance of a common intention, each of those persons shall be deemed to have committed the offence of rape. **Punishment** is **>20 y** rigorous imprisonment. It may extend to life [which shall mean imprisonment for the remainder of that person's natural life]. There will also be fine, which shall be just and reasonable to meet the medical expenses and rehabilitation of the victim, and shall be paid to the victim.

Ex: If four persons each restrain one limb of a woman and actual intercourse is done by the fifth person, all 5 are deemed to have gang raped her. If one of the persons in the gang is a woman, she is not liable according to Supreme court. **Before** CLAA 2013, **gang rape** was mentioned u/**s 376(2) (g) .**

(5) **376E, IPC - Punishment for repeat offenders [Memory aid 14]** -Whoever has been previously convicted of an offence punishable **u/s 376**, **376A** or **376D** and is subsequently convicted of an offence punishable under any of the said sections shall be punished with **imprisonment for life** which shall mean imprisonment for the remainder of that person's natural life, or with **death**.

(Memory Aid 15) D **E A** th penalty is only given in **A** and **E**.

Tables 2, 3 and **4** summarize the special situations relating to rape.

Table 2: Situations where ***sexual intercourse with consent*** is an offence

1. Sexual Intercourse with girl <18 years (statutory rape) – **S.375, IPC;**
2. Sexual Intercourse with wife <15 years (marital rape) – **S.375, IPC**
3. Sexual intercourse **u/s 376C IPC**.

Table 3: Situations where ***sexual intercourse without consent*** is not an offence

1. Intercourse by husband with wife >15 years

Table 4: Situations where ***sexual intercourse with wife*** is an offence (marital rape)

1. wife <15 years (**S.375, IPC**)
2. wife living separately from husband under a decree of judicial separation or otherwise (**S.376B, IPC**)

h. S.493, IPC

Cohabitation caused by a man deceitfully inducing a belief of lawful marriage is **punishment 10 y+fine**. This section appears similar to **S.375**(4) above, but has subtle differences. IN S.493 man uses deceit to make woman believe he is her husband. In ***S.375(4)***. she is deceived on her own. **Ex** (a) Boy 'B' is legally married to girl 'G' in childhood [Under Hindu Marriage Act, 1955, boy should be > 21 y and girl >18 y. But if marriage is solemnized against these conditions, the marriage is valid – **ch 23**]. Soon thereafter B goes to a different country to study. He lives with his friend 'F' and tells about his marriage. Several years later, 'F' goes to that village and tells girl 'G' that he is her husband. She can not

recognize him as many years have passed, and has sex with him"Punishment u/s 493.

i. S.498, IPC

Enticing or taking away or detaining with criminal intent a married woman"**punishment 2 y+ fine**. He may have taken away woman with intention to rape, but even if he has not actually raped the woman, this section comes into play.

2. Provisions in CrPC

a. S.26(a), CrPC

All rape cases should be tried as far as practicable by a Court presided over by a woman.

b. S.53A, CrPC

Medical examination of accused – Please see below under "examination of accused".

c. S.154, CrPC

(1) **S.154(1), CrPC** - If information of offence is given by a woman against whom an offence of **rape** [also offences of vitriolage (**S.326A** and **326B, IPC**), outraging modesty of woman (**s354 IPC),** sexual harassment (**354A IPC**), attempt to disrobe(**S.354B, IPC**), voyeurism (**S.354C, IPC**), stalking (**S.354D, IPC**), insulting modesty of woman (**S.509, IPC**)] has been **attempted** or **committed**, then such information shall be recorded by a woman police officer or any woman officer [Pl see **ch 1** for more on **S.154, CrPC**].

(2) **S.154(1)(a), CrPC** - In case the affected person is temporarily or permanently mentally or physically **disabled**, then police officer will record complaint at the residence of the affected person or at some other convenient place of affected person's choice in the presence of an **interpreter** or a **special educator**

(3) **S.154(1)(b), CrPC** - Such recording of statement shall be **videographed** [All 3 provisions mentioned above added in **2013** by CLAA**.** Pl also see **ch 1**].

d. S.157(1) (b), CrPC

Investigation in relation to an offence of rape - Recording of statement of the victim shall be conducted (i) at the residence of the victim or in the place of her choice and (ii) as far as practicable by a woman police officer (iii) in the

presence of her parents or guardian or near relatives or social worker of the locality.

e. S.164 (5-A), CrPC

(1) In case of offences of **rape** [also offences of sexual harassment (**S.354** and **354A, IPC**), attempt to disrobe (**S.354B, IPC**), voyeurism (**S.354C, IPC**), stalking (**S.354D, IPC**), insulting modesty of woman (**S.509, IPC**)], a **judicial magistrate** shall record the statement of the person against whom such offence has been committed.

(2) If affected person is temporarily or permanently mentally or physically **disabled**, the magistrate shall take the help of an **interpreter** or a **special educator** in recording the statement, and such recording shall be **videographed**. Such statement by the disabled person shall also be considered a statement in-lieu of **examination-in-chief** [**ch 1**], when the case finally goes to court.

f. S.164A, CrPC

Medical examination of victim – Please see below under "examination of victim".

g. S.173(1A), CrPC

Investigation in relation to rape of a child may be completed within three months from the date on which the information was recorded by the officer-in-charge of the police station.

h. S.176(1A)(b), CrPC

Please see **ch 1**.

i. S.198(6), CrPC

No court can take cognizance of marital rape, if the wife is <18 y, if >1 y has elapsed from the date of commission of offence [**limitation period**].

j. S.273, CrPC

If evidence of a **suspected victim of rape** or any other sexual offence is being recorded and if her age is **<18 y**, the court may take appropriate measures to ensure that **such victim** is **not confronted with the accused**, while at the same time ensuring the right of cross-examination of the accused [added by CLAA 2013].

k. S.309(1), CrPC

When the inquiry or trial relates to an offence under S.376 to 376-D, IPC, it shall, as far as possible, be completed within a period of **two months** from the

date of filing of the charge sheet.

l. S.327(2), CrPC and 327(3), CrPC

(1) ***in camera* trial** – The enquiry into and trial of rape of an offence u/s 376 and 376A to 376 E of IPC shall be conducted in camera [from L. camera "vaulted room" - thus in a judge's room; in secret; in empty court] [**S.327(2)**, **CrPC**]. Another in camera procedure is in relation to a mentally ill person [**ch 28**].

(2) **When another person can attend** – If either party wants any particular person to be present, it may request the judge and he may allow

(3) **Proceedings not to be published without permission of court** [**S.327(3), CrPC**] (this is different from **S.228A, IPC**, where name of victim revealing her identity cannot be published).

m. S.357B, CrPC

Additional compensation - In case a crime u/s**376D IPC** has been committed [i.e. **gang rape**], the State would pay **compensation** to the victim for rehabilitation, ***over and above the fine*** which is to be paid by the convicted person. [added by CLAA 2013].

n. S.357C, CrPC

All hospitals, whether **Govt** or **private** must provide immediate **first-aid** or **medical treatment free of cost** to any victim of **rape** [The law of **free medical aid** also applies to victims of **vitriolage** (S.326A, IPC). Pl see ch **11**]. [added by CLAA 2013].

3. Provisions in IEA

a. S.54, IEA

(1) **Previous bad character not relevant, except in reply** — In criminal proceedings, the fact that the accused person has a bad character is irrelevant, unless evidence has been given that he has a good character, in which case it becomes relevant. **Explanation 1**—This section does not apply to cases in which the bad character of any person is itself a fact in issue. **Explanation 2**—A previous conviction is relevant as evidence of bad character.

(2) **Illustration** – If a person [A] is accused of rape, it does not help the prosecution to show that A was a person of bad character. However, if A has already produced evidence that he has a good character, then prosecution can produce evidence to show he has a bad character.

1. **S.45** " Definition of expert witness [**ch 1**]

Transpose the digits of above

2. **S.54** " Previous bad character not relevant, except in reply

Add 101 to both the above sections:

45+101 " 146

54+101 " 155

3. **S.146** Questions regarding **general immoral character** of victim cannot be asked

4. S.**155(4)** – In cases of rape, it may be shown that the prosecutrix (victim) was of generally immoral character [Now removed]

b. S.114A, IEA (Burden of proving consent is on man)

(1) Man "**M**" is accused of raping woman "**W**" **u/s 376(2)(a)** till **376(2)(n)** " Sexual intercourse between "**M**" and "**W**" has been established (e.g. vaginal swab of "**W**" shows DNA of "**M**") " question is of consent only ""M" asserts that "W" had consented, but "W" says she had not consented"court will presume her statement " If "M" wants to defend himself, he must prove that woman had consented. How he does it is his problem. [**S.114A, IEA** is thus also known as "**presumption of lack of consent**"].

Memory Aid 17: 4 most important sections for rape

Remember **114 [114A IEA]**

Now **114×2 " 228 [228A IPC]**

Add 99 " 327 [327(2) and **(3) CrPC]**

Add 30 " 357B and **357C CrPC**

(2) **S.114A, IEA** is the embodiment of a fundamental principle in law known as the "**doctrine of adverse inference**". Simply stated, the **adverse inference doctrine or rule** states that when a party has relevant evidence within his control which he fails to produce, that failure gives rise to an inference that the evidence is unfavorable to him. It is also known as **empty chair doctrine** or **adverse interest rule**.

c. S.146, IEA

In cases of rape or attempt of rape, where the question of consent is an issue, it is **not permissible** to adduce evidence or to put questions in the cross-examination of the victim as to her general immoral character, or her previous sexual experience with any person for proving such consent or the quality of consent.

d. S.155(4), IEA

(1) This has been removed in ***2002***. Earlier it stated, “When a man is prosecuted for rape or an attempt to ravish, it may be shown that the prosecutrix (victim) was of generally immoral character.”

(2) This was highly criticized by women’s groups, leading to its removal. Reasoning same as in **S.146, IEA** above.

B. Types of Rape

Statutory rape

Sexual intercourse with a girl **<18 y**, with or without consent. Before **Feb 3, 2013**, it was **16 y**. CLAA 2013 changed it to **18 y**.

C. Consent

1. Age of consent

(1) Age of consent for allowing physical examination is **12 years**.

(2) Age at which girl can give valid consent for sexual intercourse is **18 years**.

(3) Age at which girl can authorize press to publish her name and picture is **18 years** (S.228A, IPC).

2. When consent is not valid

According to **S.90, IPC**, consent (as is intended by any section of IPC) is not valid when it is given (1) under fear of injury or (2) under a misconception of fact (3) mentally unsound (4) intoxicated person and (5) child <12 y. The last condition stands modified to **18y**, since **S.375** specifically raises it for rape.

3. Consent from intoxicated woman

Girl was administered some intoxicating substance and then raped. When brought to the medical officer she was still drunk. Consent unobtainable; relatives not traceable. How to obtain consent? Doctor must immediately examine **u/s 92, IPC**"Examination for girl’s benefit. She may have got STD or unwanted pregnancy.

4. Rape and arousal

It has been suggested that if arousal occurs during rape, it indicates consent. This is not true. **Arousal** has been known to occur even in **non-consensual** and **forced sex**, as the main function of arousal is **lubrication**, which in turn **protects female genitals from injury**.

D. Important Terms

(1) **Carnal knowledge** - (L. carnis, flesh) Sexual intercourse

(2) **Sexual battery** - Non-consensual touching of the intimate parts of another.

E. Examination of the Victim

1. Objectives

(1) **Record** - all injuries

(2) **Corroboration of victim's story:**

(i) Whether there is evidence of recent sexual intercourse as alleged

(ii) Whether there is evidence of previous sexual intercourse

(iii) Do the physical signs confirm the use of physical force or of stupefying drugs as alleged

(iv) Whether the physical findings are consistent with history

(3) **Collection and preservation of trace evidence** from victim's body [may assist in identification of assailant, eg saliva, hair, DNA profile from semen etc]. All trace evidence must be properly packed, sealed, labeled and then sent to the laboratory to maintain chain of custody [**ch 1**]

(4) **Treatment** of the victim [of physical and psychological damage]

(5) **Examination of scene of offence** – if desirable. Leaves, grass, mud etc entangled in victim's hair, clothes body etc may have to be matched with that present at the scene.

2. Precautions

(1) **For the police** – Should advise victim not to change clothes, bathe, or douche prior to physical examination

(2) **For the doctor** – Preferably visit the scene of crime to collect additional trace evidence (blood and urine stains, seminal stains)

(3) **For both** – Examination should be carried out without delay. Signs of sexual assault may fade rapidly with passage of time (a) Minor injuries may fade rapidly (b) swelling and tenderness of vulva may disappear (c) spermatozoa may not be detectable.

3. Preliminaries and general procedure

(1) **Requisition** from:

(i) Any police officer [SI requirement, (as in case of accused examination) not here] can ask a govt doctor to examine the victim, and if such govt doctor is not available any doctor [16 km clause, (as in case of accused examination)

not here]. Victim's consent is necessary. Victim must be produced before the doctor within 24 hours of receiving information of rape [**S.164A(1)**, **CrPC**]. This requirement is not there for accused of rape.

(ii) Court

(2) **Consent:**

(i) Written and witnessed consent of the woman must be taken. If requisition from police is there, but no consent from woman, examination can not be done [**S.164A(4), CrPC**]

(ii) Court order"examination can be done without consent [if victim of rape does not consent for examination, police official must be advised to get a court order – **ch 2**]

(iii) Consent for additional procedures – For (a) collection of specimens (b) taking photographs (c) treatment (d) release of information to the press, specific consent must be taken.

Memory Aid 18: Important components of medical examination of rape victim [S.164A, CrPC]

T I **GER C**AUGH**T IMMEDIATE**LY

1. **T** – **T**ime within **T**wenty four hours **[S.164A(1), CrPC]**
2. **G** – **G**ovt Hospital. In **E**mergency, i.e. when no Govt doctor available, any other doctor **[S.164A(1), CrPC]**
3. **R** – **R**eport must have **6 components** [**S.164A(2), CrPC**]
4. **R** – **R** for each conclusion to be given **[S.164A(3), CrPC]**
5. **C** – **C**onsent to be recorded **[S.164A(4), CrPC, S.164A(7), CrPC]**
6. **T** – **T**ime of commencement and completion of examination to be recorded **[S.164A(5), CrPC]**
7. **Immediate** – Immediate handing over of the report **[S.164A(6), CrPC]**

(iv) Consent for information to police - No consent is required for release of information to police. That is why an **informed consent** is necessary before examination, whereby the doctor informs the patient specifically that **he would be informing the police** about the event and handing over all evidences to them. If the victim gives **consent only for physical examination**, but not for information to police, the doctor should record it in writing, get signatures from the victim, and **refrain from proceeding** with the examination. Agreeing to her request may make him liable to be prosecuted **u/s 201 IPC** [**causing disappearance of evidence of offence**]. An **analogy** may make is clearer. It is possible that the victim gives consent only for general physical examination and not for anything which she

considers “**invasion of privacy**”, eg. **PV examination** and taking of **vaginal swabs** [**qualified consent**, **restricted consent**]. In such cases, if doctor proceeds with **restricted examination**, he may be sued **u/s 201 IPC** for causing disappearance of evidence of offence.

(v) Examination without consent"Doctor can be charged u/s 354, IPC (outraging the modesty of a woman). **Ex**. 14 year old girl has voluntary intercourse with her boy friend"Statutory rape"Police wants to prosecute boy"produces girl before doctor for medical examination"girl refuses consent [person >12 years can refuse consent]"examination can not proceed. Only court order can be used to force her to submit to examination.

(vi) Consent of parents or guardians is taken"(a) If girl is <12 years (b) mental illness, unsound mind, cognitive disorders (c) acute drug or alcohol intoxication (d) shock due to acute trauma (e) Acute medical conditions leading to unconsciousness

(vii) Conflict of consent – please see **ch 2**.

(viii) Report to be declared null and void in absence of consent – If woman’s consent is not there, examination would be unlawful [**S.164A(7)**, **CrPC**]

(3) **Time** - The exact time of commencement and completion of examination must be noted in the report [**S.164A(5), CrPC**]. This would indicate the interval between the alleged time of incidence and examination.

(4) **Identification:**

(i) Police officer accompanying the victim should identify her. His name and number must be recorded

(ii) Two identification marks recorded

(5) **Presence of a female chaperone** – is a must during examination, otherwise there may be allegations on the doctor himself [u/s 354, IPC – Outraging the modesty of a woman]. Ideally there should be a nurse [from doctor’s side] and a female attendant [from victim’s side] or a female police officer

(6) **Information about victim** [Legally necessary]:

(i) The name and address of the woman and of the person by whom she was brought [**S.164A(2) (i), CrPC**]

(ii) The age of the woman [**S.164A(2) (ii), CrPC**]

(iii) General mental condition of the woman (This is an additional requirement – as compared with S.53A) [**S.164A(2)(v), CrPC**]

(iv) Other relevant particulars in reasonable detail [**S.164A(2)(vi), CrPC**].

Detailed mention of these are not made in law, but the following information is implied.

Memory Aid 19: 6 components of report of rape victim [S.164A(2), CrPC]

First 2 are preliminaries and easy to remember

1. **Name, address [S.164A(2)(i), CrPC]**
2. **Age [S.164A(2)(ii), CrPC]**

For **rest 4** – **DIMM**

3. **D**escription of material taken for **D**NA **[S.164A(2)(iii), CrPC]**
4. **I**njury marks **[S.164A(2)(iv), CrPC]**
5. **M**ental condition of victim **[S.164A(2)(v), CrPC]**
6. **M**aterial particulars (any other) **[S.164A(2)(vi), CrPC]**

(7) **Information about victim** [Other] – Partially implied by [**S.164A(2)(vi), CrPC**]. Thus highly desirable to record (i) Parents' name (ii) Marital status (iii) Occupation (iv) Height and weight (v) Physical development [indicates if she was capable of struggle]. In case of children note additionally the development of breasts and genitalia (vi) Place of examination (vii) Previous history - (a) Menstrual history (b) Operations [Gynecologic, Pelvic] (c) Pregnancies (d) Sexual experience (e) Vaginal discharge (f) Venereal disease etc (viii) Requisitioning authority [police, court etc]

(8) **Statements of the victim** [**history**]– All relevant statements must be recorded. Degree of agreement between various statements goes in her favor; disagreement or contradiction goes against her. Note especially

(i) Whether she knew the accused

(ii) If anything was given to her to eat, drink or smoke before the act. Could she make out if it was alcohol or some intoxicating drug [possibility of drug facilitated sexual assault]

(iii) Whether she [or any of her relatives and friends] was put under any threat [physical or psychological]. If physical threat, what restraints or weapons were used?

(iv) Did she call for help?

(v) Date and time of alleged offence

(vi) Place of offence – whether indoor or outdoors; was the weather wet or dry

(vii) Did she have recent consensual intercourse? If yes, when? [more pertinent in married victims]

(viii) Number of alleged assailants (gang rape) [In case of too many assailants,

she might suffer fractures, or complain of severe abdominal pain due to weight of too many men atop her]

(ix) Did she struggle or was she too afraid to put up resistance

(x) Which injuries is she aware of [their location etc. This may not be too obvious to the doctor otherwise]

(xi) Did she feel any pain during or after the incident? [pain on walking, micturition or defecation is often present]

(xii) Did ejaculation take place? If yes, was it within her body [vagina, anal canal etc] or outside. If occurred outside, did she notice the appearance of discharge

(xiii) Was there any bleeding from the part penetrated [vagina, anal canal, oral cavity etc]

(xiv) Did she scratch or bite the assailant, or use some other weapon on them [would help correlate injuries on the accused]

(xv) What was the relative positions of the victim and accused during the act

(xvi) Type of sexual acts [eg oral, vaginal, anal, between the thighs etc]

(xvii) Number of sexual acts performed by each assailant

(xviii) Did they use condoms or lubricants [if condoms were used, absence of spermatozoa may be accounted for. If lubricants were used, they may specifically be tested for in vaginal swabs]

(xix) Was she using sanitary pads or tampons? If yes, did she dispose them of after the incident?

(xx) Did she lose consciousness during or after the act

(xxi) Events after the sexual assault - (a) Changed or washed private parts or clothing (b) Took bath or douche (c) Defecated or urinated after assault; or washed mouth [brushed] after oral intercourse (d) Did she comb hair (e) Did she take any sedatives or alcohol after the incident (f) Did she take any medication on her own after the incident

(xxii) Time period between sexual assault and police complaint. If there is any delay, what were the reasons?

(xxiii) General demeanor – as she narrates her story [whether aggressive, agitated, angry, calm, co-operative, dazed, disheveled, distressed, excited, intoxicated, shocked, tearful or withdrawn].

4. Physical examination

(1) **Time** – should be done as early as possible after assault, as some signs eg

swelling and edema of clitoris, labia, vaginal introitus etc may fade within 1-2 hours.

(2) **Undressing the victim** – Doctor should make no attempt to undress the victim. She should be asked to undress herself

(3) **Second examination** - If the woman is in menstrual period, a second examination should be conducted after stoppage of menstruation.

a. Clothes

(1) Find out if clothes worn at the time of examination are the same as those worn at the time of assault [If they are different, examination of clothes may not be of much use]

(2) **Removal of clothes** – In layers

(i) Outer clothing (a) Patient stands on a large clean sheet of paper and takes off her outer clothing. Anything that falls eg buttons, earth, fibers, grass, gravel, hair, leaves, mud, sand, seeds etc can thus be collected on the paper. The paper is folded and sent for examination. (b) **Examination** - Clothing is examined [for tears, soiling, loss of buttons] and then sent for laboratory examination separately [To detect stains eg blood, earth, feces, grass, grease, mud, saliva, semen, tears, urine etc. Physical, chemical and biological examination of these trace materials is done in the laboratory. DNA profile of the accused can also be made from blood, semen or saliva stains. Hair, fibers, buttons etc can be compared with those of the accused]. (c) **Drying** - Clothes should always be dried before sending to laboratory [Sending clothes with wet blood and seminal stains etc would encourage growth of fungus, destroying vital evidence] (d) **Packing** – Clothes should never be packed in polythene bags [they would tend to preserve moisture and encourage grown of fungus]. They should always be sent in clean paper bags.

(ii) Inner clothing [panties, bra etc] – Same procedure is adopted. A different paper is used

(iii) Other clothing – Other minor pieces of clothing and related material [eg handkerchief, vulval pads, vaginal tampons etc] must not be overlooked. Sometimes victim uses handkerchief to clean her genitals. Pads and tampons may show seminal stains.

(3) **Examination of clothes:**

(i) Stains – are examined both by naked eye as well as lab examination.

Significance of various stains: Seminal stains – strongly point towards sexual assault. Besides clothing, they may also be found at the scene of crime [eg bedding, carpets, car seats etc]. Seminal stains on undergarments and outer garments do not necessarily indicate ejaculation outside the vagina. They may drip out of private parts due to movements of the victim. Movements of victim may also cause semen to drip from the private parts to virtually any other place, which the victim visited.

(ii) Trace evidence – finding of grass, leaves, mud, sand etc on clothings may support the victim's assertion about the place where the assault occurred.

b. General examination

(1) **Prerequisites:**

(i) Lighting - Examination must be done in (a) well illuminated room and then additionally (b) under UV light also [invisible or faint seminal stains will show up as fluorescent bluish white stains under UV light [because of choline]. Please see below under seminal fluid-physical examination also]

(ii) Photographic record – (a) External – All injuries howsoever minor; broken fingernails, torn clothes, seminal stains etc (b) Internal – Pictures of vaginal vault and cervix with a **photocolposcope** or **video colposcope** [Colposcopes with an arrangement for photography]. Useful for production in court later.

(iii) Labeled diagrams – of all injuries should be made. Sizes of all injuries and their relative positions from one another should be recorded.

(2) **Eyes:**

(i) Redness and swelling of eyelids, conjunctival congestion – indicate constant crying

(ii) Pupils - constricted or dilated [indicate intoxication, head injury];

(iii) Petechial hemorrhages - On conjunctiva [also eyelids, and elsewhere] – indicate attempt by accused to smother in order to stifle cries

(3) **Pulse and respiratory rate** – may be - due to emotional stress or major trauma

(4) **Gait** – Guarded gait due to pain. Victim walks with legs apart and slow paces

(5) **Examination for drunkenness** – If victim appears drunk, examine for drunkenness [please see ch 40].

(6) **Marks of violence** – May or may not be present. [A] When present – (I) Determine age of injuries – to correspond with allege time of offence (II) Observe nature and site - Areas of tenderness, bite marks, bruises, lacerations,

scratches etc must be searched for on the entire body. Describe their size, shape, extent, special characteristics [eg bruises showing restraints on arms and legs], area on which present and possible age. Try to correspond with victim's description of assault. Injuries around special locations may indicate specific activities by accused. From above downwards these signs may be (i) around mouth and throat – Attempts to prevent victim from calling for help (ii) Lips – due to blows, rough handling, attempts to kiss and bite (iii) Shoulders and Back – due to pressure to pin down victim on ground (iv) Wrists and arms – due to attempts to seize victim (v) Nails – Damaged, broken or avulsed nails [due to victim scratching assailant]. Collect debris from under the nails. Examine for epidermal cells, blood, hair, fibers etc. DNA profile may be prepared. This may be matched to the suspect and his clothes etc. [same procedure is performed in throttling"**ch 19**]. (vi) Breasts – due to manual squeezing, manipulation, rough handling. Love bites may be present [**ch 3**] (vii) Nipples – may be completely bitten off [class IV bites, **ch 3**] (viii) Thighs and buttocks – due to attempts to separate thighs or buttocks for penetration. Both inner and outer side of thighs may be scratched, bruised or lacerated.

Rarely even incised wounds may be seen on thighs due to application of knife in order to forcibly separate thighs, while the victim attempts to adduct thighs together [this is a rare example of **defense wounds** on thighs. Generally they are found on hands [**ch 12**]. [B] When absent – Accused often takes the plea of absent marks of violence as indicative of consent. Marks of violence absent even in the absence of consent due to (i) fear of injury, death"submission by the victim (ii) Incapacitation caused to victim by (a) throat grips (b) strong neck holds (c) severe blow on the head (iii) force used is insufficient to cause injury (iv) Too early examination. Bruises may not be noticed for up to 48 h following assault (v) Too delayed examination – due to late reporting. Marks of minor injuries would fade or heal. (vi) Description of marks of injury – is a legal necessity [**S.164A(2) (iv), CrPC**]

(7) **Bite marks** – They may comprise of true bites or just **love bites** [**ch 3, 12**]. [A] True bites – Class II, III and IV bite marks [**ch 3**]. [B] Love bites - (i) Class I bites (ii) Etiology - strong continuous suction kisses during passionate sexual intercourse (iii) Pathogenesis - Negative pressure produced by suction"rupture of small vessels from .ed air pressure"Intradermal petechial

hemorrhages (iv) Appearance - (a) discoid or elliptical patterned bruises [1-2 cm] or (b) a shower of petechial hemorrhages which may be confluent, or (c) both (d) Sometimes the shape of upper and lower lips is reproduced (v) Site – wherever the victim is kissed with strong suction. Commonly on face, neck, shoulders, breasts, nipples, chest wall. Less commonly at lower abdomen, upper part of thighs.

(8) **Petechiae on face** or **conjunctiva** – May indicate

(i) intentional attempts to make victim unconscious to silence her or

(ii) unintentional partial asphyxia caused during forcible restraint.

Fig. 25.2: Abrasions and bruises on inner thighs and genitals in a case of rape.

c. Local examination

Local examination of genitalia is most crucial

(1) **Position:**

(i) Victim placed in lithotomy position [legs drawn up and widely open] in good light.

(ii) Application of local anesthetics [eg cocaine] may become necessary if too

much pain is felt on separation of thighs.

Memory Aid 20: Ano-genital injuries in rape

TEARS (**T**ears, **E**cchymoses, **A**brasions, **R**edness, **S**welling)

(2) **Methods of examination:**

(i) Direct visual inspection

(ii) Colposcopy – Better [**ch 26**]

(iii) Toluidine-blue [Tb] contrast application – (a) Best. (b) Reveals microinjuries, not revealed even by colposcopy (c) Procedure - (I) Take vulval swabs for forensic analysis before application of stain (II) Toluidine blue (1%) is then painted on the posterior fourchette, using a swab, before any instrumentation. (III) After a few seconds, the residual stain is removed with lubricating jelly and gauze. (IV) The patient may experience some stinging at the application site. [toluidine blue is also used to detect microfractures of the hyoid and thyroid – **ch 19**]. Please co-relate all injuries [Normal female genital anatomy] in ***ch 24***.

(3) **Clitoris** – red, swollen, edematous. **D/d** - May be due to uncleanliness and consequent scratching, masturbation.

(4) **Labia:**

(i) Finger nail abrasions, bruises, reddening, lacerations, soiling etc should be noted on major and minor labia. These are due to attempts of the assailant to draw apart labia.

(ii) Thin tears in the folds of skin between the labia majora and minora are seen sometimes. These are due to labia minora being inverted towards the vaginal orifice, causing stretching of their junction with the labia majora.

(5) **Hymen:**

(i) In case of virgins tears usually occur posteriorly at 6 O' clock position, or posterolaterally [5 O' clock, 7 O' clock], or less commonly still more laterally [4 O' clock, 8 O' clock] [**Fig 25.3**]. Abrasion collars in firearm wounds are also described by referring to clock positions – **ch 13**].

(ii) **Reasons why tears occur more commonly in posterior structures** [fourchette, posterior commissure, perineum etc], rather than anterior structures [vestibule, clitoris, anterior commissure, mons etc] - (a) the anatomic site of fixation to the perineal body lies posteriorly; consequently the tissues are at greatest stress in this location when forceful stretching

occurs. (b) Anteriorly periurethral tissues buttress the hymen. There is no such support posteriorly.

(iii) In case of already ruptured hymen [as in married women] there may only be abrasions and bruising of hymen.

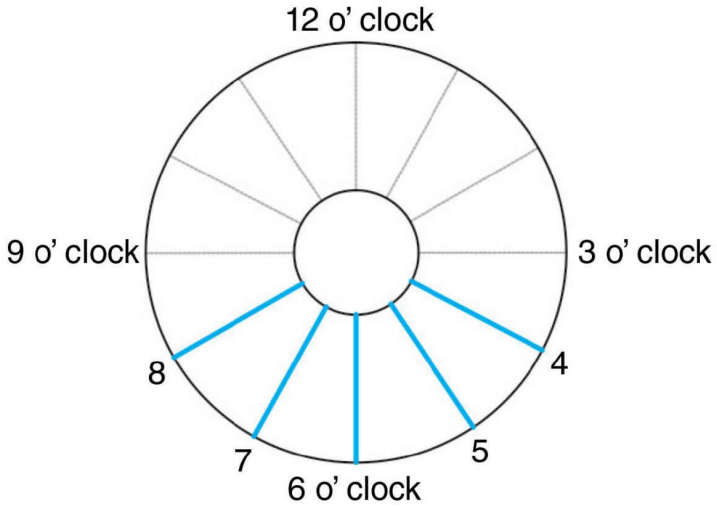


Fig. 25.3: Blue lines show the most common hymenal tears in a virgin. For ease of referencing of tears, vaginal opening is likened to a clock, as the woman is examined in a lithotomy position.

(6) **Method of hymenal examination** -

(i) Conventional method using Glaister-Keene rods - These are glass or plastic rods bent at about 150° one end. They have a small spherical head at one end. So called because first made by **John Glaister** in Pyrex glass. They can be fitted with a light, which travels even through the bend by total internal reflection. Before insertion, the rod should be at body temperature. The spherical glass head is gently passed through the hymenal aperture and then eased forwards, when the hymenal edges become slightly everted. By slowly rotating the sphere, around which the edges are deployed hymenal tears can easily be seen. This method does not cause any pain or injury.

(ii) Foley's catheter or an inflatable balloon – Same as above, except that a Foley's catheter can be used. Easily available in all hospitals. Glaister-Keene rods are not easy to get, and may have to be order-made.

(iii) Conical measures [**Fig. 25.4**] - These are wooden cones similar to the ones used for measuring circumference of heart valves [**ch 5**].

(iv) Hymenoscope - This is a refinement of **Glaister-Keene rods** mentioned above. While GK rods have a source of light at the rear, **hymenoscope** has

a source of light **within the ball** (2) **[Fig. 25.5]**. A urethroscope light bulb (1) is screwed into the cavity of the rod (3) and projects into the ball. Wires (4) are led from the light bulb, through the rod to an ordinary pocket battery. Secondly clear plastic material is used instead of glass. Thirdly, the ball and the rod are hollow, and the ball can be unscrewed from the rod. The ball is constructed in two parts which are then joined. A series of interchangeable balls, graded in size can easily be made (5). One with diameter of **3/4 inch (18 mm)** is best for general use, but is too large for virgins and children, and too small for women who have had frequent sexual intercourse. A ball with **1/2 inch (12.5 mm)** diameter is good for virgins and with **1 inch (25 mm)** diameter for married women. The rod has an external diameter of **4/10 inches (10 mm)** and an internal diameter of **2/10 inches (5 mm)**. The instrument can be easily made by an electrician. It is simple, light in weight, not fragile, and easily cleaned with soap and water followed by spirit. The long flex is useful in allowing the battery to be out of the examination field (eg it can be in the pocket).

(v) Through rectum - Pass a finger into the rectum"push posterior vaginal wall forwards and downwards"This pushes the hymen forwards which can now easily be seen lying against the posterior vaginal wall. Procedure may be painful in simultaneous rectal assault.

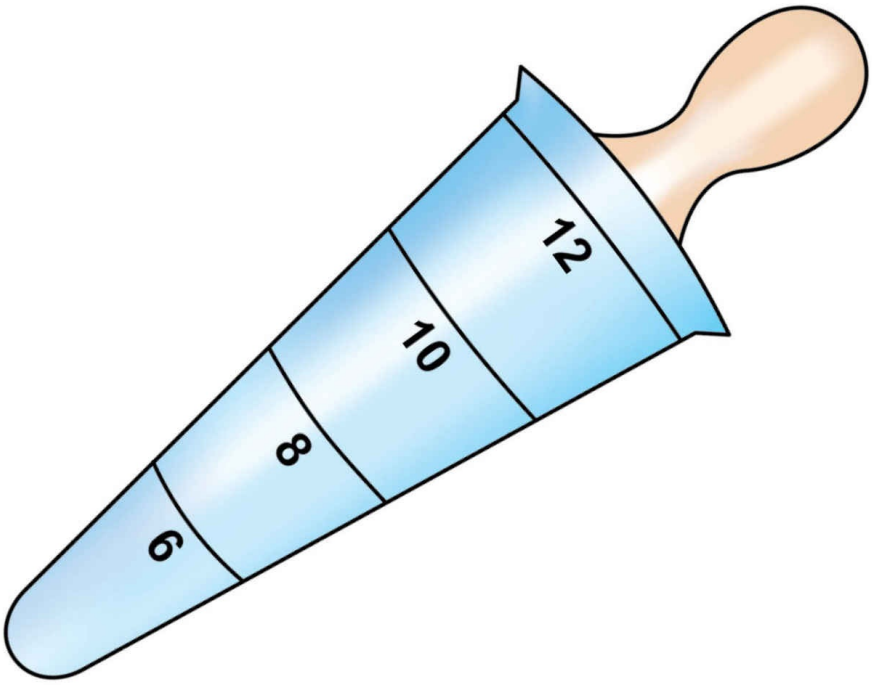


Fig. 25.4: Measuring cone used for measuring the circumference of the hymenal opening during gynecological examination of victims of sexual offences. Pl compare with similar cones used to measure heart valves [ch 5].

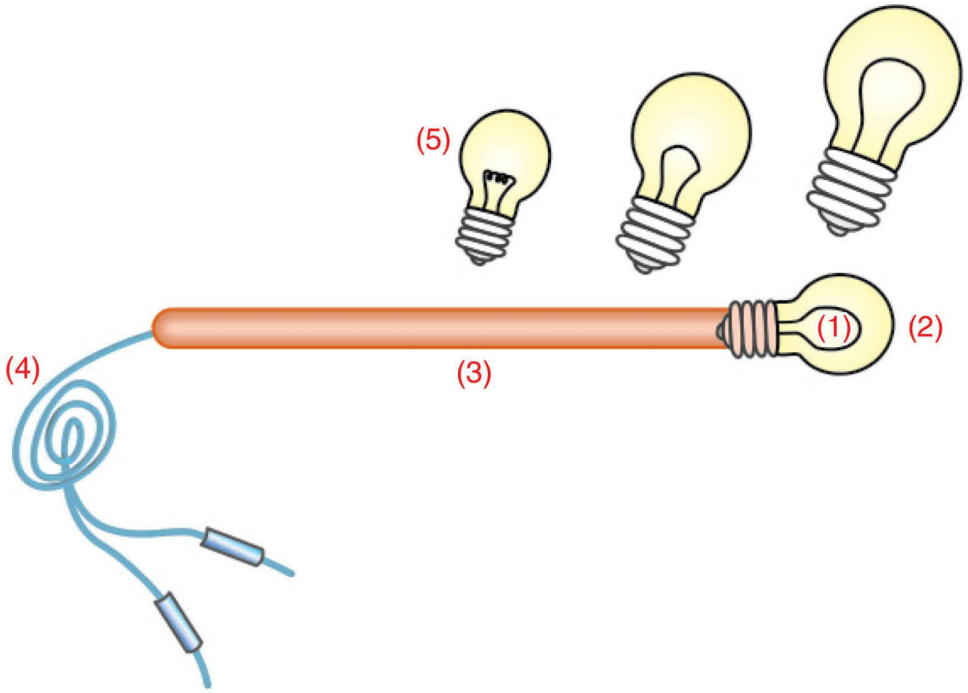


Fig. 25.5: Hymenoscope. Please correlate nos (given in red). with text.

(7) **Character and extent of hymenal injuries would depend upon:**

(i) Amount of force used

(ii) Disproportion between male organ and vaginal introitus

(iii) Extent of penetration

(iv) Nature of hymen [taut, elastic, tough, deep seated, fimbriate etc]

(8) **Age of Hymenal tears:**

(i) Immediately after the act – margins of torn hymen are red, sharp and bleed on touch. The tissues round them are tender

(ii) 3-4 days – edges of tears are congested and swollen

(iii) 7 days – complete healing takes place, but the torn parts do not unite. Appear as V-shaped defects

(iv) Several months – V-shaped

(9) **Fossa navicularis** – May show small tears 1-2 mm in size. Caused by excessive stretching. It disappears after repeated sex acts; thus may not be seen in married women

(10) **Fourchette** – Fragile. May show similar tears as fossa navicularis. Generally torn at the first intercourse; thus an imp sign in virgins but not in

married women.

(11) **Vaginal introitus** – slightly edematous. Shows abrasions, bruises.

(12) **Vaginal canal:**

(i) Use a vaginal speculum for examination

(ii) Look for abrasions, bruises, lacerations. Tears may extend up rectum [rectovaginal fistula] or bladder [vesicovaginal fistula].

(iii) Bruising - (a) site – most common is anterior vaginal wall [lower 1/3rd] and posterior vaginal wall [upper 1/3rd]. Bruising of this nature is more consistent with penetration of penis than with digital penetration. (b) color – (I) Immediate – dark red (II) 24 h – deep red or purple.

(iv) In consensual intercourse, since there is preliminary stimulation and consequent lubrication, injuries are not seen.

(v) Lacerations – May rarely be present even in consensual intercourse if (a) there is marked disproportion between penis and vagina (b) If the copulation is very active, enthusiastic and forceful (c) when there has been a complete abstinence from intercourse for a considerable length of time

(vi) If there are no fresh injuries, assess the following: (a) Number of fingers that can be introduced through the hymenal orifice. (I) This allows the examiner to assess elasticity of hymen and to determine the degree of penetration which would be possible without its rupture. (II) In most young women, a finger may be passed into vagina through an intact annular hymen, which is felt as a constricting ring round the tip of finger. (III) If two fingers may be passed easily, sexual intercourse is possible without hymenal rupture. (b) The areas and degree of tenderness complained of by the patient (c) Laxity of vaginal orifice - Indicates previous penetration (d) Length of vagina into the posterior fornix - Elongation indicates previous repeated sexual intercourse

(13) **Vaginal vault:**

(i) May vary from minimal mucosal trauma to extensive lacerations. The latter may occur when there is marked disproportion between penis and vagina [eg rape on young girls] or if a sexual pervert penetrated some instruments.

(ii) Lacerations are usually seen in posterior fornix of the right side of the vault and less frequently on the left side.

(iii) Not seen in the anterior fornix.

(14) **Cervix** – Examination of cervix is best done with a **colposcope**. It is a

stereoscopic binocular field microscope with a long focal length and powerful light source and permits a magnification between **2** to **40 times**. **Video colposcopes** allow digital image capture. Abrasions of cervix are mostly seen in digital rather than penile penetration. Abrasions must be differentiated from erosions [**Table 5**]. Cervical erosions do not occur due to penetration.

Table 5: Differences between cervical abrasions and cervical erosions

S.No	Criteria	Cervical abrasions	Cervical erosions
1.	Location	Away from external os	Around external os
2.	Margins	Not clearly defined	Well defined margins
3.	Color	Dull color	Bright red

(15) **Posterior commissure** – rupture may be seen, but is more common in young virgins because of disproportion between male organ and female vagina.
(16) **Perineum** – torn, especially in young children.
(17) **Genital injuries *may be absent*** due to
(i) Genitals only touched or rubbed with penis
(ii) Elasticity of genitalia and hymen in a post-pubertal female
(iii) Victim sexually experienced; roomy vagina
(iv) Use of lubricants.
(18) **Presence of genital injuries** – eg abrasion, bruises or lacerations are consistent with intercourse with a consenting woman and do not necessarily indicate rape. Local genital injuries and genital bleeding in a newly married bride are well-accepted phenomena.
(19) **Back and buttocks** – Grass, leaves, mud etc on back or buttocks would confirm sexual assault taking place outside in specific areas
(20) **Anal canal** – Tears if anal penetration occurred.
(21) **2nd examination** – of the victim must be done a day after the first, as some

bruises, especially those in the lower vagina may take some time to come up to the surface [**delayed bruising** (**ch 12**)].

Fig. 25.6: Grass against back indicates the victim was forced on grass. DNA from grass and other vegetable matter from back can be matched with that at the scene to confirm where the incident occurred.

d. Hair

(1) **Scalp hair:**

(i) Note general condition – Disheveled, tangled, torn

(ii) Look for entangled grass, leaves, sand, soil etc. Can reveal place of assault.

(iii) Blood indicates local lacerations etc.

(iv) Comb and send any trace evidence that falls out

(v) Collect hair from front, top, back, left and right side

(2) **Pubic hair:**

(i) Should be combed out

(ii) May reveal non matching male pubic hair; foreign material

(iii) If matted, clip them with scissors. Send for DNA; matting may be due to dried semen

(iv) In case of deceased – pull 15-20 hair with forceps [makes available root characteristics for comparison]

(v) In case of living victim – cut; don't pull

(vi) Areas to collect from – pubic region, vulva

(3) **Preservation and maintenance of chain of custody** – Vital for legal validity [**ch 1**]. Pack, seal, label and sent to lab.

e. Finger nails

(1) **Fingernail scrapings** – Take from beneath each nail. Send all ten fingernail scrapings in separate envelop marking each finger and side of hand. Skin, blood, cloth fibers, hair etc of assailant may be found on microscopic examination. DNA profiling from these materials can reveal identity

(2) **Fingernail clippings** – Take [of all ten nails] with a nail cutter. Send each in a separate envelop with nail cutter separately. Nail cutter should be new and unused before.

f. Seminal stains

Steps should be followed in strict sequence

(1) **If pubic hair are matted** – Nip away with scissors entire matted hair as close to skin as possible. Moisten them with normal saline for half an hour and examine under microscope. If examination not possible, place in a small paper envelop, seal, label and hand over to police to be sent over to forensic biology section for examination.

(2) **Swabs from outside areas:**

(i) Before starting local examination take swabs from (a) vaginal area [outside] (b) Perineum (c) Pubic area (d) Inner side of thighs (e) Any other suspected area [buttocks, abdomen, hands, breasts, face], which can be discovered by taking the patient in dark and examining entire body area by UV light. Seminal stains would fluoresce bluish white.

(ii) Use a normal throat swab moistened with normal saline.

(iii) Take four swabs [from all areas contaminated with semen] with cotton moistened with saline. (a) Prepare smears with the first– on a glass slide immediately, and dry (I) fix - by heat or chemically [Fixatives that can be used are (1) commercial spray fixative (2) equal quantities of absolute alcohol and ether (3) methanol]. **Fixation** is a process by which biological tissues [eg spermatozoa] are preserved from decay by preventing autolysis, putrefaction and fungal growth etc. It also .es the mechanical strength and stability of tissues. (II) Label slides – using a diamond pencil. Write

patient’s name and MLC number. (III) Examine for spermatozoa, or hand over to police to be sent over to suitable department [forensic biology]. (IV) If very few sperms are seen, record location on X and Y axis, by a specialized microscope. Defense may want a re-examination of slide by its own expert (V) After examination, preserve slide for possible production in court (b) Dip the second swab in a small quantity of saline"examine for presence and motility of sperms (c) Dry the other two. Send one for chemical [eg acid phosphatase], immunological, TLC or other methods of analysis [please see below] and other for DNA profiling.

(iv) If seminal stains have dried out - scrape with a blunt knife, seal, document and send entire flake for chemical and biological examination.

(3) **Tampons** – if present, remove and send for examination

(4) **Vaginal aspirates** [VA]:

(i) Instill 5 ml of normal saline in posterior fornix.

(ii) Aspirate by means of a blunt end pipette or a flexible plastic pipette. Preferably concentrate by centrifugation at 2,000 g for 15 min. Collect cellular pellet.

(iii) Alternatively take some mucus on a glass rod or wooden spatula from upper vagina [less preferable].

(iv) Immediately place onto a clean, prewarmed microscope slide, and examine for motile spermatozoa under a phase contrast microscope.

(v) Store at -80°C, if examination cannot be done immediately.

(vi) If VA cannot be taken for some reason, take a vaginal swab as below, insert in a small amount of normal saline and examine.

(5) **Vaginal swabs from inside:**

(i) Swabs to be taken after taking vaginal aspirates, but before any digital examination

(ii) Low vaginal swab – Take after examination of hymen

(iii) High vaginal swab – (a) insert a small vaginal speculum, and take under direct vision through speculum (b) In case of small children or gross injuries – use local anesthetic before inserting vaginal speculum; rarely GA may be required

(iv) Take 4 swabs and deal as with “swabs from outside areas”.

(v) **If there has been penetration, but offender had vasectomy, or did not ejaculate** - Non-sperm male cells [from penile shaft] may be present [if

condom was not used]. These are identified by Y-chromosome specific DNA probes [Fluorescence in situ hybridization or FISH; please see below]. The cells can be amplified by PCR and a DNA profile made [**ch 29**].

(6) **Cervical swabs:**

(i) From within the cervical canal.

(ii) Take in all cases, especially if offence was committed >48 h previously.

(iii) Gives better results than vaginal swabs.

(iv) Take 4 swabs and deal as with "swabs from outside areas".

(7) **From other objects** – eg condoms etc found at scene [please see below under "seminal fluid"collection of material].

(8) **Interpretation:**

(i) Detection of at least one motile spermatozoa denotes sexual assault within **last 24 h**.

(ii) Detection of at least one intact non-motile spermatozoa confirms sexual assault, but timing cannot be ascertained

(iii) Finding of broken spermatozoa [eg separate heads and tails] - Not conclusive, as these appearances can be mimicked by several artifacts.

(9) **Description of material taken for *DNA profiling*** – is a legal necessity [**S.164A(2) (iii), CrPC**].

g. Blood stains

(1) Note presence or absence of blood stains around genitals and on legs

(2) Determine if these could be due to menstruation

(3) Profuse bleeding from vagina may wash out seminal fluid and spermatozoa may not be found. Despite this fact, a vaginal swab should be taken and slide made.

h. Venereal disease

(1) Common VDs and microorganisms associated with them, and which may be seen after rape are:

(i) **Bacterial** - (a) Chancroid [Haemophilus ducreyi] (b) Chlamydia [Chlamydia trachomatis] (c) Granuloma inguinale [Klebsiella granulomatis] (d) Gonorrhea [N gonorrhoeae] (e) Syphilis [T pallidum]

(ii) **Fungal** - Candidiasis [yeast infection]

(iii) **Viral** - (a) Herpes simplex (b) HIV (c) HPV [Human papillomavirus] (d) Molluscum contagiosum [molluscum contagiosum virus MCV] (e) Viral hepatitis [Hepatitis B virus]

(iv) **Parasites** - (a) Crab louse or "pubic lice" [Pthirus pubis] (b) Scabies [Sarcoptes scabiei]

(v) **Protozoal** - Trichomoniasis [Trichomonas vaginalis].

(2) **General cleanliness** – of the victim should be noted. In unclean women, irritation and resultant abrasions may be there due to self-scratching.

(3) **Inflammation** – Due to most of above VDs. Erythema may be due to general uncleanliness. Must be differentiated

(4) **Discharge** – may be due to gonorrhea, syphilis or several other VDs. Collect swab for microscopy and culture.

i. Lab Examination

(1) **Blood and urine:**

(i) for date rape drugs and therapeutic medications.

(ii) for pregnancy tests – in late arrivals

(2) **Vaginal swab** – Should be collected by introducing sterile dry cotton swab into the posterior vault of the vagina or preferably into the cervical canal.

(i) Look for spermatozoa [please see below – seminal fluid – microscopic examination].

(ii) If condom use has been alleged by victim, test for lubricants and other materials found on condom. (a) **Lubricants** - (I) A polydimethylsiloxane (PDMS) liquid as lubricant is used by many condom manufacturers. Detected by Fourier transform infrared spectroscopy [FTIR]. (II) Corn starch, Lycopodium, silica, and talc can also be observed microscopically. (b) **Spermicides** – (I) Nonoxynol-9 as spermicidal jelly – detected by FTIR.

j. Fingerprints of assailant

On body of victim may be recovered with proper techniques [**ch 3**].

5. Psychiatric examination

(1) Whether well-oriented in time, place and person [for relevant questions to determine this, please see **ch 1**]

(2) whether amnesic, sleepy [drug facilitated sexual assault]

(3) Unduly afraid, depressed
(4) harboring any suicidal tendencies
(5) Any signs of **post traumatic stress disorder** [PTSD]. 24 hour vigil should be ordered if victim is likely to harm herself out of depression or guilt.

6. Opinion

The opinion is given in one to the following ways

(1) **There is evidence of recent sexual intercourse** – When recent injuries to genitals and other body parts are found and motile spermatozoa are recovered from vagina
(2) **There is evidence of remote sexual intercourse** – When partially healed injuries to genitals and other body parts are found and non-motile spermatozoa are recovered from vagina [typically occurs when victim reports late to the police]
(3) **There is evidence of recent penetration** – When there are local recent injuries to genitals, but no spermatozoa are recovered. [typically occurs when penetration is by vasectomized person, by a person wearing a condom, with a finger, dildo, pencil, stick or some other similar object].
(4) **There is evidence of remote penetration** – When there are local partially healed injuries to genitals, and no spermatozoa are recovered.
(5) **There is evidence of recent sexual assault** – When there are recent injuries/bite marks etc on face, cheeks, neck, breasts, nipples, thighs etc, but injuries to genitals are absent [typically occurs when help arrives before actual penetration occurs]
(6) **There is evidence of remote sexual assault** – When there are partially healed injuries on face, cheeks, neck, breasts, thighs etc; injuries to genitals are absent.
(7) **There is no evidence of vaginal penetration, but the possibility cannot be ruled out** – When there are no injuries, and no spermatozoa are recovered from vaginal canal.
(8) **There is evidence of recent oral/anal intercourse** – When recent injuries are noted and motile spermatozoa are recovered from these areas.
(9) **There is evidence of remote oral/anal intercourse** – When partially healed injuries are noted, and non-motile spermatozoa are recovered from these areas.
(10) **There is evidence of recent/remote oral/anal penetration** – When

recent/partially healed injuries are found and spermatozoa are not recovered.

(11) **There is no evidence of oral/anal penetration, but the possibility can not be ruled out** – When there are no injuries, and no spermatozoa are recovered from oral cavity/anal canal.

(12) **Reasons to be stated** - The report must state precisely the reasons for each conclusion arrived at [**S.164A(3), CrPC**].

7. Handing over of report

Doctor must immediately give the report to the investigating officer, who must then forward it to the magistrate immediately [**S.164A(6), CrPC**].

F. Examination of Victim in Special Cases

1. Rape on children and virgins

Findings are same as above. Following additional points must be kept in mind because of gross disproportion between size of male organ and vaginal introitus. In general younger the child, greater the disproportion between genitals and greater the local injuries.

(1) **Gait** – Child walks with great difficulty due to pain

(2) **Signs of general violence** – are typically absent as a child neither has any idea of what is happening, nor is capable of any resistance.

(3) **Thighs** – Attempt to separate thighs may cause child to cry because of great pain. Local anesthetic should be applied in such cases.

(4) **Labia** - must be separated with gentle traction as they may be extremely tender.

(5) **Hymenal tears:**

(i) Hymen is deeply situated in children

(ii) Rupture of hymen is the main evidence.

(iii) **Salient features** (a) injuries are more severe than those in the adults (b) Multiple hymenal lacerations (c) hymen may be entirely destroyed

(iv) Tear caused by acts other than rape – eg by digital penetration or by tampons would be old; would not bleed on touch.

(6) **Vaginal canal:**

(i) Abrasions, bruises and lacerations would be more severe because of disproportion.

(ii) Lacerations of anterior and posterior vaginal walls. Anterior tears may involve the bladder and posterior tears, the anorectal canal.

(iii) Clots of blood – may be found within the vagina. Blood may be oozing from the vaginal canal.

(iv) Mucopurulent discharge from vagina – if parents have brought child late and she had acquired infection.

(v) If vagina is very small, accused would place the penis either within the vulva or between the thighs. In such cases injuries to vagina would be absent.

(7) **Vaginal vault** – shows extensive lacerations. There may be vaginal herniation of abdominal viscera.

(8) **Fourchette** – fragile, tears during first intercourse. Very important sign in virgins.

(9) **Posterior commissure** – rupture is more common, because of disparity between male organ and vaginal canal

(10) **Perineal body** – may be torn.

2. Rape on deflorate women

(1) **Changes seen in deflorate women**

(i) Hymen - completely destroyed

(ii) vaginal orifice – dilated

(iii) mucus membrane – wrinkled and thickened. These changes are seen even without childbirth.

(2) Complete penetration in such women may occur without any evidence except presence of seminal fluid. Absence of injuries in such women does not rule out penetration.

(3) **Local injuries may be seen:**

(i) If excessive violence was used

(ii) Victim offered resistance

(4) **Injuries on other parts of the body** – eg breasts, lips, inside of thighs, buttocks etc are indicative of rape. Such injuries are unlikely to occur in a consenting woman.

3. Rape on pregnant women

(1) Injuries to abdomen are more severe because of its prominence

(2) Breasts are tender and may show greater injury

(3) Injuries to fetus may be there. May result in abortion.

4. Rape on postmenopausal and elderly women

Findings are same as above. Following additional points must be kept in mind

(1) **Reporting** - Alterations in mental status, dependence on others, and physical restrictions can prevent reporting.

(2) **Vaginal canal** –

(i) Abrasions, bruises and lacerations would be more severe because of atrophic post-menopausal vagina.

(3) **Perineal trauma** – would be more for the same reasons as above.

G. Examination of the Accused

(1) Procedure similar as in case of victim.

(2) **Better done:**

(i) after examination of victim – (a) evidence evaluation in victim is more vital as it may be lost quickly [eg motility of spermatozoa may be lost soon] (b) confirmation of injuries, which she states to have inflicted on the accused (c) Victim needs psychological support. Earlier examination allows earlier psychological referral

(ii) by the same doctor who examined victim – better co-relation of injuries in both [scratch marks on accused co-related with broken nails in victims; bite marks on accused co-related with teeth of victim etc; more severe genital injuries in victim co-related with disproportionately large size of male organ]

1. Preliminaries

(1) **If brought by general public** - Inform police. Label case as MLC [**ch 11**]

(2) **If brought by police** - should be identified by the escorting police officer. Two identification marks must be recorded on the MLC.

(3) **Consent:**

(i) If not arrested [eg when brought by mob]- Should be taken. If not given, wait till police arrives and arrests him formally

(ii) If arrested – (a) can be done without consent **u/S.53ACrPC** (b) **Request to be made by *SI* and above** - In case of suspected rape, a police officer of the rank of sub-inspector (SI) can ask a govt doctor to examine the accused,

and if such govt doctor is not available any doctor within a radius of 16 km from the place of offence. If accused does not give consent, doctor can use reasonable force to examine him (no consent necessary). (c) **What doctor must note** - The doctor must include following particulars in his report (I) The name and address of the accused and of the person by whom he was brought [**S.53A(2) (i), CrPC**] (II) the age of the accused [**S.53A(2) (ii) CrPC**] (III) marks of injury [**S.53A(2) (iii), CrPC**] (IV) Description of material taken for DNA profiling [**S.53A(2) (iv), CrPC**] (V) Other relevant particulars in reasonable detail [**S.53A(2) (v), CrPC**] (VI) Reasons - The report must state precisely the reasons for each conclusion arrived at [**S.53A(3), CrPC**] (VII) Time - The exact time of commencement and completion of examination must be noted in the report [**S.53A(4), CrPC**]

(4) **Note** - Date, month and place of examination

(5) The examination must be begun **without delay**, as the signs proving act may disappear rapidly.

(6) **General medical history:**

(i) past illnesses, serious accidents and surgical operations.

(ii) H/o medication, consumption of alcoholic and other intoxicating agents.

(7) **Last few activities** -

(i) history of any sexual act indulged in last 24 h.

(ii) when was last bath taken

(iii) when did he change his clothes last

(iv) if there are injuries [fresh or old] on the body, what explanation does he have for them.

2. General examination

(1) **General behavior** and **mental state** - whether angry, excited, fearful, guilty looking, inhibited.

(2) **Clothes** - note

(i) blood and semen stains

(ii) cosmetic traces [eg face powder, lipstick stains]

(iii) physiological stains - mucus, saliva, tears, milk etc

(iv) presence of hair, fibers and foreign matter [eg dust, mud, grass, soil, sand] and whether the same matches with that found on victim’s clothes.

(v) tears and loss of buttons.

(vi) Trousers - (a) Soiling - Do the trousers show any soiling with mud and grass at the level of knees. The soiling may be at a level above knees as trousers may have been lowered to expose genitals. (b) Zip fly - preserve any hair, fiber etc caught in between, as it may be from victim. There may be blood and seminal stains on the inside of trouser fly

(vii) Underwear - does it show any tears; underwear cord fastened, unfastened or loosely fastened. Presence of blood and seminal stains

(viii) Preserve all clothes, seal and send for examination.

(3) **Female hair:**

(i) Besides noting their presence on clothes [as above], note their presence also on face, body, penis, prepuce and pubic hair of the accused.

(ii) Pubic hair may have to be combed to separate female hair lying loosely entangled.

(iii) Preserve for cross matching with victim

(4) **Shoes** - note particles of earth or any other foreign matter trapped between the uppers and soles at the welt of shoes. Foreign material on clothes and shoes can help establish the place of occurrence. May help to corroborate or refute victim's story.

(5) Whether appears to be under **influence of alcohol or other intoxicating drugs**. If yes conduct examination for drunkenness simultaneously [**ch 40**]

(6) **Comparison of characteristics with that of victim** - Note age, development of genital organs and physical powers. Compare each with that of victim and determine if the incompatibility can explain injuries, possibilities of overpowering etc.

(7) **Hair** - if disheveled, disorderly, contaminated with soil etc. If bunches of hair are missing

(8) **Injuries:**

(i) Note injuries eg abrasions, bruises, lacerations, teeth bite marks, over face, neck, hands, chest, abdomen, genitals and thighs.

(ii) Establish if they could be as a result of resistance offered by victim.

(iii) Determine their age and note if consistent with time of occurrence of incident.

(9) **Nails** - should be scraped, and scrapings examined for blood, epithelial cells, fibres etc.

(10) **Pubic hair:**

(i) Present or shaved.
(ii) If present note if matted due to emission of semen.
(iii) Cut away about 10-15 at the base for matching with those found on victim
(11) **Venereal diseases [VD]**- Note all signs of any VD. Prophylactic treatment of patient may be started. If victim has reported late and shows same VD, it is a corroborating symptom. Significantly VD may be transmitted both from accused and victim to each other.

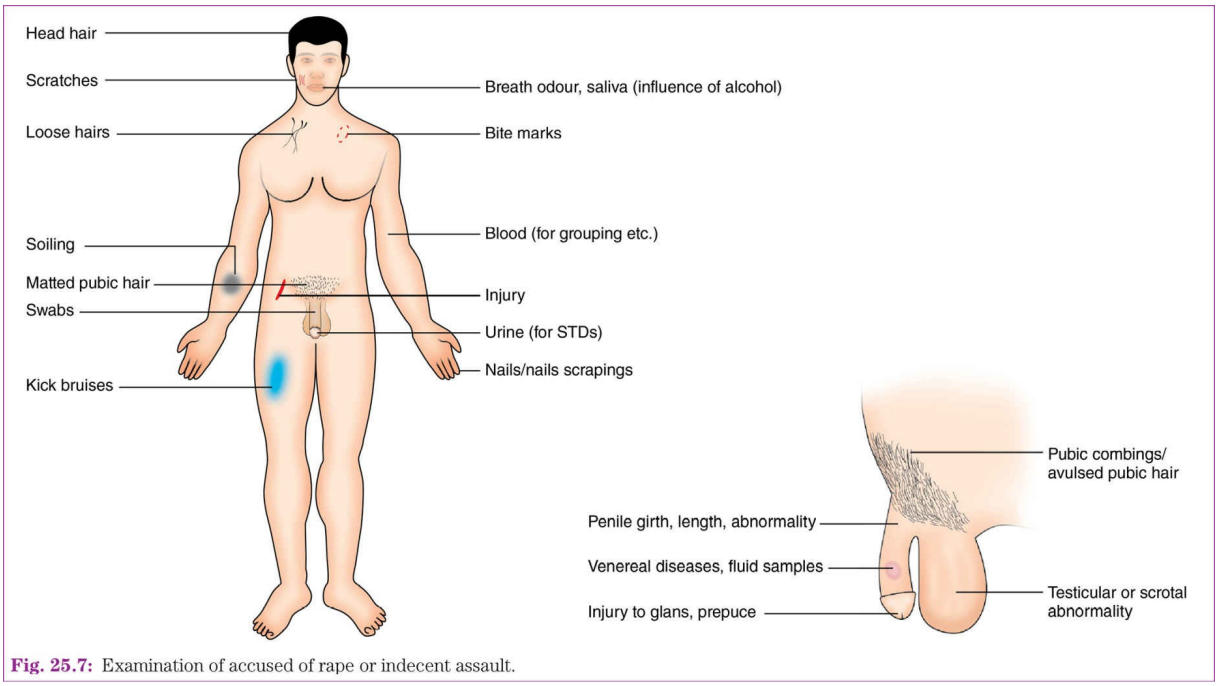


Fig. 25.7: Examination of accused of rape or indecent assault.

3. Examination of genitals

(1) **Development of genitals** - to be noted. Conduct an examination for erectile dysfunction [impotency] also [**ch 23**]
(2) **Injuries on the body** - abrasions, bruises, lacerations all over the body especially to local parts
(3) **Local injuries:**
(i) Abrasions, bruises, lacerations of the shaft of penis, prepuce, scrotum, inside of thighs.
(ii) Forceful penetration, produces injuries to frenulum of prepuce in the uncircumcised.
(iii) Abrasion of glans is seen in both circumcised and uncircumcised.
(iv) Such injuries are common in young children and virgins with narrow

introitus, because of disparity in size.

(v) When disparity in size is not there, patchy redness of glans, especially around the rim is more common.

(vi) Dried blood and seminal stains may be present on the shaft of penis, scrotum, inside of thighs and adjoining skin

(4) **Smegma** - Smegma [Gk sm*e*gma, soap] is a cheesy secretion which collects under the foreskin of the glans it requires about 24 hours to collect and is usually rubbed off during complete penetration, as the foreskin is pressed backward during intercourse.

(i) Retract the foreskin and record the presence of absence of smegma.

(5) **Examination of penis and glans for vaginal cells:**

(i) Suspect's penis is washed with saline"material stained with **Papanicolaou's stain**"vaginal and cervical cells are stained"indicate that penis was in contact with female genital tract. Barr bodies in the cells indicate cells of female origin. A DNA profile can be made from female cells using PCR [**ch 29**], which can establish the identity of the victim.

(ii) Suspect's penis is cleaned with a filter paper"Expose filter paper to vapors of **Lugol's iodine**"If vaginal epithelial cells are present, paper turns brown, because of the presence of glycogen in vaginal cells. This test is +ve up to 4th day.

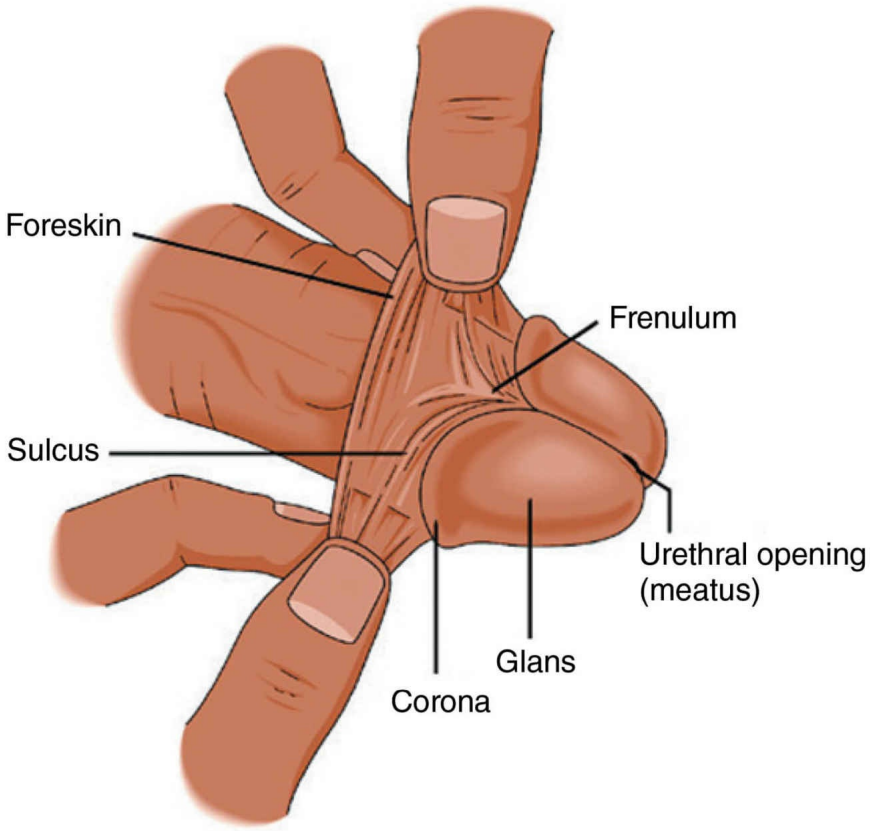


Fig. 25.8: Penis showing frenulum

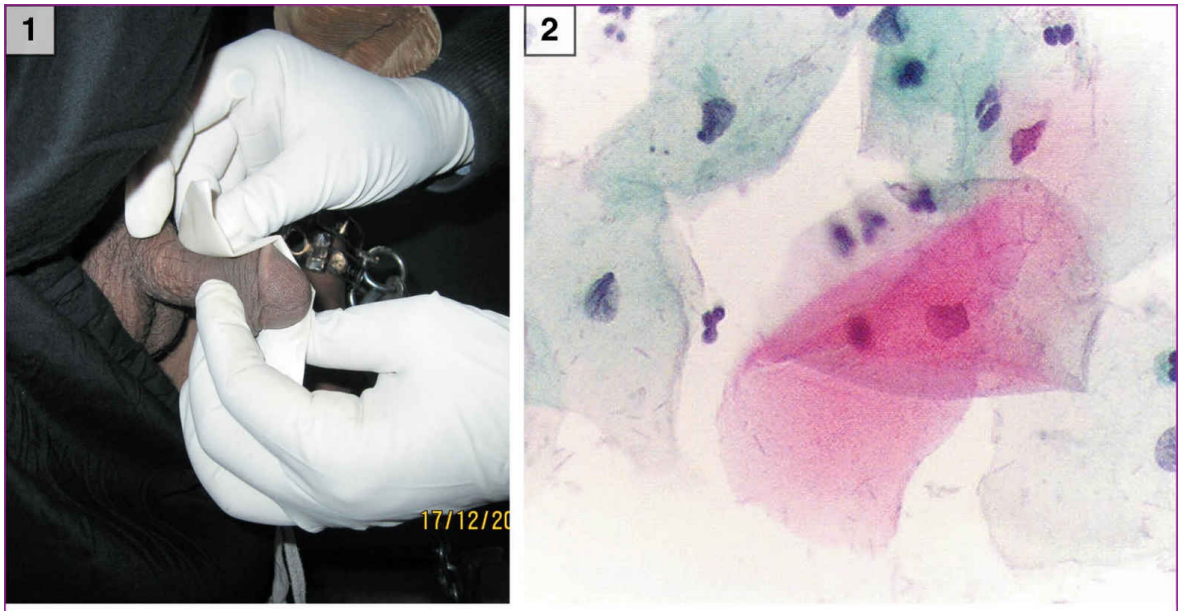


Fig. 25.9: **(1)** Swabbing the penis of accused for detection of vaginal cells **(2)** Vaginal cells on penis seen with Pap stain [400x].

4. Specimens to be collected

(1) **Blood** - for grouping

(2) **Head hair** - May have to be matched with those found on victim's hand, clothes and at the scene.

(3) **Pubic hair:**

(i) Combings [for sample of victim's hair].

(ii) Cut pubic hair [for sample of accused's hair]. Of great value if they are matted, because they indicate emission. Avulsed pubic hair must not be taken. It is a painful procedure and serve no additional purpose.

(4) **Loose hair** - anywhere on the body

(5) **Swabs** - from urethral orifice, coronal sulcus, prepuce, penile shaft, scrotum, pubic area and inner aspects of thighs. They may reveal infecting organisms, blood, leucorrheal stains, vaginal cells etc which may be cross-matched with that found on victim

(6) **Nail scrapings** - victim's skin underneath show struggle with the victim.

5. Opinion

The opinion is given in one to the following ways:

a. On the question of having performed the act

(1) Findings are suggestive of subject having been involved in a recent non-consensual sexual act [if there are gross injuries on body and genitals + smegma is not present under the glans + there is presence of vaginal cells on the penile shaft]

(2) Findings are suggestive of subject having been involved in a recent sexual act [if there are no injuries on body + smegma is not present under the glans + there is presence of vaginal cells on the penile shaft]

(3) Findings are suggestive of subject having attempted a non-consensual sexual act. There is nothing to suggest penetration of the penis in the vaginal tract. However a superficial touching of penis with female genital organs cannot be ruled out [if there are gross injuries on body and genitals + smegma is present under the glans + there are no vaginal cells on the penile shaft]

(4) There is nothing to suggest that the subject performed a recent sexual act, consensual or non-consensual [if there are no injuries on body and genitals + smegma is present under the glans + there are no vaginal cells on the penile

shaft].

b. On the question of ED

Please see **ch 23**.

6. Handing over of report

Doctor must immediately give the report to the investigating officer, who must then forward it to the magistrate immediately [**S.53A(5), CrPC**].

H. Medicolegal Questions Relating to Rape

1. Can a female rape a male?

Legally this is not possible in India? In western countries (eg Canada, France) it is possible. In India in similar circumstances, at most female can be charged under S.350, IPC (criminal force). Punishment 3 months and/or `500 fine (S.352, IPC).

2. Is emission necessary to constitute an offence of rape?

No.

3. How much penetration is sufficient to constitute rape?

Even **touching of penis with vulva** would legally constitute rape.

4. Age of the accused

(1) **India** - Charge of rape cannot be brought against a child <7 years of age (**S.82, IPC**). Please also see **ch 3** – Criminal responsibility.

(2) **Under common law [ch 1] of England:**

(i) A boy <14 y cannot commit rape [R v. Phillips (1839) 8 C&P 736; R v Waite [1892] 2 Q B 600].

(ii) This rule has now been abolished by **s1** of the Sexual Offences Act 1993.

5. Age of the victim

Females of all ages have been raped (from newborns to over 90 years). Children are more commonly raped because

(1) in India, there is a false belief that having sexual intercourse with a virgin girl can cure venereal disease. So many persons attempt intercourse with very

young girls, who are bound to be virgins
(2) children offer less resistance.

6. Rape during sleep, under narcotics or hypnosis

(1) **Administration of narcotics** – Rape is possible (please see DFSA above)
(2) **During anesthesia:**
(i) Rape is certainly possible. Doctors have been charged. On emerging from light anesthesia, patients [especially neurotic] may have hallucinations or dreams of an erotic or fearful nature. Such patients may genuinely believe that the doctor had sexual intercourse with them while they were unconscious.
(ii) Putting a woman under the influence of chloroform is very difficult [**ch 40**].
(iii) there is no drug which can produce immediate unconsciousness when placed over the face of the person [**ch 40**].
(3) **Sleep** – impossible to have sexual intercourse with a woman in her sleep
(4) **Hypnosis** – No one can be hypnotized against his/her consent. Thus woman must understand fully the consequences before giving consent for hypnosis. However consent for hypnosis is not consent for sexual intercourse, and thus sexual intercourse without consent under hypnosis is rape.

7. Rape and resistance

If the victim does not have injuries, it may be believed that she did not apply resistance to the maximum and thus she had consented. This is not necessarily true. Lack of resistance can be due to

(1) terror
(2) feeling of helplessness
(3) when a loved one has been put under threat
(4) she was tied
(5) her own clothes obstructed movement
(6) multiple assailants
(7) weak woman. However cases have occurred when a woman consented for intercourse to obtain some reward, which was not given afterwards. Consequently she denied having given consent. Please see **S.114A, IEA** above for details.

8. Impersonation

Rape by impersonation of husband is possible

(1) **Dark** - If woman is lying in the dark, and accused secretly enters. Woman may think he is the husband

(2) **Identical twin** – may impersonate his brother

(3) **Long absence** - Marriage at young age after which husband went abroad. Somebody else returns after some years claiming to be her husband.

9. Rape by fraud or misrepresentation

Sexual intercourse by fraud or misrepresentation is rape

(1) Doctor tells patient sexual intercourse with him is necessary for treatment

(2) Music teacher tells student, it is necessary for improving her voice.

10. Intercourse other than penile-vaginal intercourse

S.375. IPC now includes all kinds of **illegal penetration** [by penis, any part of the body, by any object, eg pencil] under rape. The part penetrated may be vagina, anus, urethra, tightly closed thighs, axilla, or any part of woman's body [pl see **S.375, IPC** above]. However anal or oral intercourse even with a legally married wife is illegal u/s377 IPC.

I. MLI of rape

Rape is a cognizable offence

G. Child Sexual abuse

Child sexual abuse [syn child molestation, sexual exploitation] is using the child as an object for sexual satisfaction.

IV. INDECENT ASSAULT

Indecent assault is any unwanted sexual behaviour or touching which is forced upon people against their will [ex - touching arms, shoulders, breasts, buttocks etc of a female colleague against her will].

A. Legal Aspects of Indecent Assault

294, 354, 509, [IPC Sections].

B. Sexual Harassment

Sexual harassment, is intimidation of a sexual nature, or inappropriate promise of rewards in exchange for sexual favors.

1. Legal aspects

a. Vishaka judgment (1997)

In **India**, the first major case of sexual harassment occurred in the state of Rajasthan in **Sep 1992**. A female social worker tried to stop the wedding of an infant girl, less than a year old in her village, Bhateri, about 45 km from Jaipur. Outraged by this, five men from the upper-caste family of the infant decided to "teach her a lesson" and gang-raped her repeatedly in the presence of her husband. In its landmark judgment, Vishaka v. State of Rajasthan, A.I.R. 1997 S.C. 3011, the Supreme Court of India, for the first time laid down guidelines defining what exactly is sexual harassment at the work place, and what should be done to deal with it.

b. S.354A, IPC

S.354A, IPC (added in **2013**) **defines sexual harassment** and its **punishment**.

(1) **Definition -** A man doing any of the following **4 acts** commits the offence of **sexual harassment**.

(i) physical contact and advances involving unwelcome and explicit sexual overtures; or

(ii) a demand or request for sexual favors; or

(iii) showing pornography against the will of a woman; or

(iv) making sexually colored remarks [**S.354A(1),** IPC]

(2) **Punishment -**

(i) For **first 3 acts** mentioned above"Rigorous imprisonment up to **3y** or **fine** or **both** [**S.354A(2),** IPC]

(ii) For **4th act** mentioned above" Imprisonment [simple or rigorous] up to **1 y** or **fine** or **both** [**S.354A(3),** IPC].

V. SEXUAL PERVERSIONS

Sexual perversions or ***paraphilias*** are sexual behaviors that involve unusual objects (eg animals, dead people) and activities (eg exhibitionism, voyeurism). If not fulfilled they can cause clinically significant distress [DSM-5]. Most common paraphilias include exhibitionism, fetishism, frotteurism, pedophilia, sexual masochism, sexual sadism, transvestic fetishism and voyeurism.

A. Buccal Coitus

please see **Fellatio**.

B. Eonism

Please see transvestism below.

C. Exhibitionism

Exhibitionism is obtaining sexual arousal by exposing genitals to an unsuspecting stranger.

Salient features:

(1) May or may not be accompanied by masturbatory acts

(2) **Prevalence** – 2nd most common paraphilia after sadomasochism

(3) **Perpetrator** – mostly males, although female exhibitionists are also known

(4) **Victims** [to whom genitals are displayed] – children, persons of opposite sex

(5) **Etiology:**

(i) **Majority suffer from psychotic disorders** - (a) Alcoholism (b) Epilepsy (c) GPI (d) OCD (e) Psychopathic personalities (f) Senile dementia

(ii) **Monoamine excess** – especially dopamine. Patients being treated for Parkinsonism with carbidopa/levodopa [metabolic precursor of dopamine] and pergolide [direct dopamine agonist] show -ed tendency of exhibitionism. On discontinuing medications, these tendencies subside.

(6) **Legal aspects** – punishable **u/s S.268 IPC** - Public nuisance; **S.290, IPC** - Punishment for public nuisance in cases not otherwise provided for [fine of `200]; **S.294, IPC** - Obscene acts and songs [3 m or fine or both]; **S.509, IPC** - Word, gesture or act intended to insult the modesty of a woman [1 y or fine, or both].

(7) **DSM-5** [**ch 28**] assigns code **302.4** to exhibitionistic disorder.

D. Fetishism

Fetishism is obtaining sexual arousal using or thinking about an inanimate object or part of the body.

Salient features:

(1) **The inanimate object** [**fetish**] - is generally a piece of cloth [eg bra, handkerchief, panties, petticoat, stocking or undergarments] or even shoe or sandal of the opposite sex. Sometimes it may be a flower or garland worn by the female. A picture of fetish may also suffice.

(2) **Acts that produce orgasm:**

(i) Fetish is usually stolen and genitals are rubbed with it till ejaculation occurs.

(ii) Shoe may serve as an artificial vagina for such fetishists.

(iii) Looking at the fetish may have the same effect.

(iv) Sometimes just the act of stealing a fetish produces an orgasm.

(3) **Sex** – almost exclusively seen in males.

(4) **MLI:**

(i) Fetishist may commit theft, robbery etc to obtain fetish and may get caught for theft.

(ii) Women may be attacked to get the fetish. This may rarely be accompanied by rape or sexual assault.

E. Frotteurism

Frotteurism [French frotter, to rub] is obtaining sexual arousal and gratification by rubbing one's genitals against others in crowds and public places.

Salient features:

(1) Offenders generally are unable to perform normal sexual acts, and would have a premature ejaculation if they attempted it.

(2) **Co-morbidity** - Often seen with other paraphilias such as exhibitionism, voyeurism etc

(3) **Legal aspects:**

(i) **S.354, IPC** [assault or criminal force to woman with intent to outrage her modesty]

(ii) All **IPC sections** discussed under exhibitionism are applicable also.

F. Lesbianism

Lesbianism [syn tribadism] is sex between two women. The term comes from the name of an island, the Isle of **Lesbos**, mentioned in Greek mythology, where women used to practice homosexuality.

Salient features:

(1) Active partner [often a transsexual or transvestite] is called a **butch** or **dyke**; passive partner known as **femme**.

(2) The active partner uses a dildo either strapped around waste, or manipulated by hand. It is punishable **u/s377, IPC**.

G. Lust Murder

Lust murder [syn erotophonophilia] is homicide in which the offender gets erotic satisfaction by killing.

Salient features:

(1) Considered an extreme form of sadism.

(2) **Sex** - Most lust murderers are males, but female lust murderers are known.

(3) **Manifests by:**

(i) Murder - during sexual intercourse.

(ii) Mutilation – of victim's sexual organs, cutting or stabbing of breasts, lower abdomen evisceration. Usually takes place postmortem, but may occur before death also. May be accompanied by sucking, licking or mouthing of wounds, drinking of blood [vampirism]

(iii) May include other activities - (a) removing clothing from the body, (b) posing and propping of the body in different positions, generally sexual ones, (c) insertion of objects into bodily orifices, (d) Masturbating over dead body (e) necrophilia and necrophagia

(iv) Murder after rape, plainly to prevent victim from going to police, or due to anger, jealousy or revenge is not lust murder.

(4) **Fantasies** - are a key component in lust murders. These may be fueled by pornography and facilitated by alcohol.

(5) **Most common among** - serial killers. They chose a victim with a specific trait [offender's Ideal Victim Type (IVT)]. Once the offender has found in IVT, he might engage in stalking [following victim everywhere] or other predatory behaviors before acting out his fantasy on his victim.

(6) **Periodic outbursts** - are often seen. Due to offender's recurring compulsion or sudden outbursts of sexual desire. Behavior is normal during two outbreaks.

H. Masochism

Masochism [syn, passive algolagnia] is sexual arousal through pain inflicted on onself through another person, usually a partner of the opposite sex. Named after an Austrian novelist **Leopold Sacher Masoch** (1836-1895), who suffered from this perversion. Stimulant for his work was being whipped by his wife.

Salient features:

(1) This condition is opposite of sadism. The term **algolagnia** [Gk, algos, pain; lagneia, lust] includes both sadism or masochism.

(2) **Epidemiology:**

(i) Sex – Mostly found in males. About 1 in 20 of all masochists are female. Masochist females willingly expose themselves to the risks of severe bodily injury at the hands of their sexual partners.

(ii) Found in all age groups and in all socioeconomic strata.

(3) **Characteristics:**

(i) Masochists get pleasure from being abused, beaten, degraded, dominated, enslaved, humiliated or tortured by their sexual partner.

(ii) Tend to place themselves repeatedly in self-defeating situations.

(iii) Painful stimuli may entirely replace the ordinary sex stimuli.

(4) **Combinations** – Both sadism and masochism are rarely found in a pure state. Usually found together in combination, with one trait dominant over other [**sadomasochism**].

(5) **Bondage** - is the tying, binding, or restraining of a person for the sexual pleasure of both parties involved. Rope, cuffs, bondage tape, self-adhesive bandages, or other restraints are used for this purpose. Giving and receiving pain occupies their sexual life of such partners so much that sexual intercourse may never be indulged into.

I. Masturbation

Masturbation [syn ipsation, onanism] is deliberate self-stimulation resulting in sexual climax.

Salient features:

(1) mild masturbatory exercise in both men and women are part of normal physiological response and are considered completely normal and even healthy activities

(2) **Technique:**

(i) Males - manual, rubbing genital parts against bed or other objects.

(ii) Females - (a) Manual – (I) Moving a finger gently and rhythmically over clitoris or labia minora (II) Application of steady pressure over these parts with several fingers or whole hand (b) Other – Bananas, dildos [artificial masculine genital parts, made of plastic or rubber], electrical vibrators, fingers, glass tubes, hollow articles [eg test tubes, bottles], metallic bars and wooden rods. Also rubbing genitalia against pillows, bed or some other similar object

(3) **Anal and urethral insertions** – also seen, but rare

(4) **Legal aspects:**

(i) Privately done - Not an offence

(ii) Publicly performed – Punishable **u/s S.268**, **S.290**, **S.294** and **S.509, IPC** [for details, please see exhibitionism above].

J. Mixoscopia

Observing the sex performance of other couples.

K. Necrophilia

(1) **Necrophilia** [Gk nekros, corpse; philia, love] Intercourse with dead people.

(2) **Necrophagia** [Gk phagos, to eat][syn, anthropophagy] is eating their flesh for sexual gratification.

(3) **Etiology:** (i) genetic (ii) temporal lobe anomalies (iii) head trauma during infancy (iv) sexual inadequacy with living (v) drugs and alcoholism (vi) sexual abuse during childhood (vii) unconscious suppressed hostility towards females (viii) helplessness and resistlessness of the victim [exercising sexual power over corpses] (ix) Castration anxiety

(4) **Characteristics:**

(i) May have sadistic tendencies [necrosadism]. May mutilate corpse after intercourse.

(ii) Decomposition, foul smell and coolness act as stimulants

(5) **MLI** – punishable **u/s297, IPC** - Offering any indignity to any human corpse. Punishment [1 y or fine or both] [please also see **ch 5**].

L. Oedipus complex

Intended sex between mother and son.

M. Pyromania

Getting sexual stimulation by seeing flames and destruction of a building. It is a psychosexual disorder.

N. Sadism

Sexual arousal through inflicting pain on another person. From French author **Marquis de Sade** (1740-1814), who wrote books in which the characters suffered from this condition. Also known as **algolagnia**.

Salient features:

(1) Seen more commonly in men

(2) Often seen in association with masochism [sadomasochism, which is the commonest paraphilia].

(3) **Etiology:**

(i) Psychotic disorders – sociopathic, schizoid or other personality abnormalities

(ii) Organic brain abnormalities and damage – gliomas, temporal horn dilatation

(iii) Endocrine abnormalities

(iv) Early experiences - of brutality in relation to sex

(v) family background

(vi) substance abuse.

(4) **Methods employed:**

(i) Beating [may cause fractures, rupture of internal organs]

(ii) Biting [eg cheeks, breasts, nipples]

(iii) Bondage –tying up or otherwise restraining [syn sexual bondage]

(iv) Burning skin by cigarettes, lighters, hot wax

(v) Inserting bottles, candles or sticks in anus or vagina

(vi) Producing cuts

(vii) Whipping etc

(5) **MLI** – Generally done in association with a consenting masochist. If done

forcibly without consent, person may be charged with **S.323, 324, 325 and 326 IPC** [**ch 11**]. Even consensual acts have been successfully prosecuted in foreign countries, because sadomasochism per se is illegal.

O. Sexual oralism

Sexual oralism is obtaining sexual pleasure from application of mouth to the sexual organs.

Salient features:

Seen in both homosexuals and heterosexuals

Types

a. Anilingus

Oral stimulation of the anus. Active and passive agents may be of either sex.

b. Cunnilingus

Cunnilingus [syn, mouth job] is oral stimulation of female sexual organs [clitoris, vagina etc].

Salient features:

(1) It can be done by a person of either sex, a child or even an animal.

(2) **Risks** - If female is pregnant, may accidentally cause air embolism [suction of air through uterine veins].

c. Fellatio

Fellatio [syn, blow job, buccal coitus, Coitus per os, fellation, intercourse through mouth, irrumation, Sin of Gomorrah (for origin, please see sodomy below)] is oral stimulation of male sexual organs [penis and occasionally scrotum].

Salient features:

(1) Like cunnilingus it can be done by a person of either sex, a child or even a trained animal.

(2) **Risks** - May cause accidental gagging of the fellator [Please see **ch 19**].

(3) **Lesions caused by fellatio:**

(i) Oral submucosal hemorrhages - secondary to repetitive negative pressure and blunt trauma associated with fellatio. May be an incidental finding in

sexually active adults and in children suffering from sexual abuse. Appear as erythematous, petechial, or ecchymotic macules or patches at the junction of the hard and soft palate. May appear bilaterally or as single lesions extending across the midline. The lesions do not blanch with pressure, are asymptomatic, and non-ulcerated. The uvula and structures of the oropharynx are typically spared.

(ii) STDs including HIV.

P. Sodomy

Please see below.

Q. Transvestism

Transvestism [Latin trans, opposite; vestere, clothing] is sexual arousal from dressing in the clothes of the opposite sex.

Salient features:

(1) There is often a desire to be identified with the opposite sex. Also known as **cross dressing**, **eonism** [After French diplomat **Chevalier d'Éon** [1728-1810] who had this disorder], **fetishistic transvestism** or **transvestic fetishism**. The sufferer is known as a transvestite.

(2) **Epidemiology:**

(i) Prevalence – 3rd most common paraphilia after sadomasochism and exhibitionism.

(ii) Age – Most develop the disorder by late adolescence

(iii) Sex – mostly seen in males. Very few cases of female transvestites are known.

(iv) Associations – Generally associated with (a) autoerotic asphyxia [**ch 19**], (b) bondage and (c) sadomasochism. A small percentage are homosexuals.

(3) **MLI:**

(i) suicide by transvestites may appear like cases of autoerotic asphyxia.

(ii) May be punishable **u/s290, IPC** - Punishment for public nuisance in cases not otherwise provided for [fine of `200], eg a man loitering around wearing a bra etc may be considered to cause public nuisance.

(4) **DSM-5** [**ch 28**] assigns code **302.3** to transvestism.

R. Triolism

Intercourse between three people, either two females and a male or two males and a female. Another variation is when a person shares his wife/girl friend with another, and observes the act from a hidden place. Also known by various other names such as Triolism and Ménage a trois.

S. Undinism

Same as Urolagnia.

T. Uranism

Uranism is a general term given to the perversion of sexual instincts. Sexual gratification by fingering, fondling, licking also goes under the name Uranism. Not to be confused with Urolagnia.

U. Urolagnia

Sexual excitation at the sight of urine, or people urinating. Also known as undinism and urophilia.

V. Voyeurism

Same as Peeping Tom; sexual arousal by observing nude individuals without their knowledge.

Salient features:

(1) **Legal definition** - Watching or capturing the image of a woman engaging in a private act in circumstances where she would usually have the expectation of not being observed either by the perpetrator or by any other person at the behest of the perpetrator or disseminates such image **[S.354C,** IPC].

(2) **Punishment** -

(i) **On first conviction** - Imprisonment [simple or rigorous] of **minimum 1 y**. May extend to **3 y**. Also fine [any amount]

(ii) **On second or subsequent conviction** - Imprisonment [simple or rigorous] of **minimum 3 y**. May extend to **7 y**. Also fine [any amount]. There are 2 explanations to this section. **Explanation 1** - For the purpose of this section, “private act” includes an act of watching carried out in a place which, in the circumstances, would reasonably be expected to provide

privacy and where the victim's genitals, posterior or breasts are exposed or covered only in underwear; or the victim is using a lavatory; or the victim is doing a sexual act that is not of a kind ordinarily done in public. **Explanation 2** - Where the victim consents to the capture of the images or any act, but not to their dissemination to third persons and where such image or act is disseminated, such dissemination shall be considered an offence under this section **[S.354C,** IPC].

(3) **DSM-5** [**ch 28**] assigns code **302.82** to voyeurism.

VI. SODOMY

Sodomy [syn, buggery, Greek love] is anal intercourse between two men [homosexual] or a man and a woman [heterosexual].

Salient features:

(1) The practice was prevalent in ancient Greece [Greek love]

(2) Gerontophilia [sex with old] and pederasty [sex with young] includes acts of sodomy [please see below].

(3) **Partners:**

(i) Active agent is one who penetrates; passive, the one who is penetrated.

(ii) When passive agent is a child [usually a boy], he is known as a **catamite**. **Sodomite** is one who practices sodomy. There may be both **active** and **passive sodomites**.

(iii) Both active and passive agents may interchange, even if one of the partners is a female. She uses a dildo to penetrate when acting as an active partner.

(4) **Constituents of offence:**

(i) Any degree of penetration, or even an attempt at penetration into the anal margin

(ii) Ejaculation is not necessary

(5) **Prevalence:**

(i) Frequent among people living in hostels, military barracks, prisoners and sailors etc because they are isolated from women from long periods

(ii) Socioeconomic status - seen among all levels

(iii) Often resorted to, when the attacker discovers that the woman is menstruating [**Aruna Shaunbaug case - ch 2**].

A. Male Prostitutes

In India some **eunuchs** act as male prostitutes to earn livelihood. They act as passive agents in sodomy. They grow long hair, wear lipsticks and female ornaments, dress like women and develop mannerisms similar to women. They cannot offer vaginal intercourse. Mostly they would satisfy customers by axillary, intercrural or oral intercourse or masturbation. Two types exist:

1. Hijrahs

(1) These are males who have been castrated [removal of testis] and emasculated before puberty.
(2) They keep adding to their tribes by abducting young boys and castrating them. When the wound heals, the scar invaginates and comes to resemble those of females, although the depth is not that of normal vagina.
(3) Being castrated before puberty, female characteristics such as female voice, feminine distribution of fat and hair, and development of breasts are common.
(4) They intentionally wear feminine attire and behave in a feminine way, in order to act as a perfect female substitute for prostitution.
(5) But they are biological males; their buccal smears show male sex chromatins.

2. Zenanas

(1) These are males whose genitals are intact.
(2) They simply wear female attire and behave like females.
(3) They live separately from hijrahs
(4) Both hijrahs and zenanas maintain a line of separation between themselves and the female prostitutes.

B. Examination of the Passive Agent

Examination is conducted on same general lines as that of victim of rape. Findings may disappear within 1-2 days so immediate examination is a must. Only hematoma and lacerations are visible for several days - hematoma for 7-10 days and lacerations for 2-3 weeks.

1. Preliminaries

(1) **Consent** - informed consent must be taken. If <12 y, consent of parents or guardian

(2) **Profession** - is it his profession to act as a habitual passive agent

(3) **General history** - hemorrhoids, anal fissures etc. Victim would complain of pain during walking, defecation and anal examination.

(4) **History relating to bowel movements:** (i) any surgical operation (ii) bowel habits (iii) instrumentation of the bowel (iv) previous constipation (v) use of enema, laxatives and suppositories (vi) previous acts of anal intercourse, their frequency, i.e. whether the victim was a habitual agent (vii) last act of anal intercourse

(5) **History relating to current act:**

(i) Date, time and place of alleged act

(ii) Bleeding or any other discharge - at the time of act; afterwards.

(iii) Defecation after the alleged act

(iv) Degree of penetration

(v) If any struggle or resistance was offered, or was the act consensual

(vi) Pain experienced - at the time of act; subsequently

(vii) Position in which act was performed

(viii) Use of lubricant

(ix) Whether bathed or washed anal area after the act

(x) Whether changed clothes or not

(xi) Whether ejaculation occurred or not

2. Examination of clothes

Please see above “examination of rape victim" examination of clothes”. **Fecal stains** should especially be searched for as incontinence may be a symptom.

3. General examination

(1) **Mental state** - Note if child is tense, fearful, crying or relaxed. **Mild sedation** is advisable if child tense.

(2) **Genital examination** - for injuries [eg abrasions, bruises, lacerations, teethbite marks] is done as in case of rape victim. Look specifically for scratches around buttock and anal region.

4. Anal examination

(1) **Position:**

(i) Older children are examined under a powerful light in the knee-elbow

position by pulling the glutei to both sides. The anus is readily visualized in this way. Lesions observed are indicated on a diagram according to clock quadrant [as in hymen above, **Fig 25.3**].

(ii) Because knee-elbow position is difficult to get in younger children and may cause some victims unusual embarrassment or recall memories of prior abuse, **left lateral position** relaxing the glutei and knees with the head on a pillow may be employed in a very young child. Knees may be drawn as far towards the chest as possible [lateral decubitus] (iii) In case of female victim there should be a female chaperone.

(2) **Normal anal orifice:**

(i) Normal orifice is slit like, and runs anteroposteriorly.

(ii) Surrounding skin shows marked natural folds due to the action of **Corrugator Cutis Ani** muscle.

(3) **Local swabs** - Take swabs from anal verge, skin of perineum, from inside the anal canal and rectum. May show lubricant matter, seminal fluid or organisms of venereal diseases on culture, all of which are positive proof of anal penetration.

(4) **Proctoscopy** - Pass a small unlubricated proctoscope "inspect lower rectum and anal canal for injuries or mucosal abnormality"take swabs from lower rectum and anal canal

(5) **Lesions:**

(i) marked in children because of great disproportion in size between anal orifice of victim and penis of accused.

(ii) Lesions may be produced if penis or any similar objects [fingers, dildos etc] are introduced

(iii) if a moderate sized penis is introduced with care and if adequate lubricants have been used, there may be no injury. This is partially because of anal dilation and partially due to lubricants.

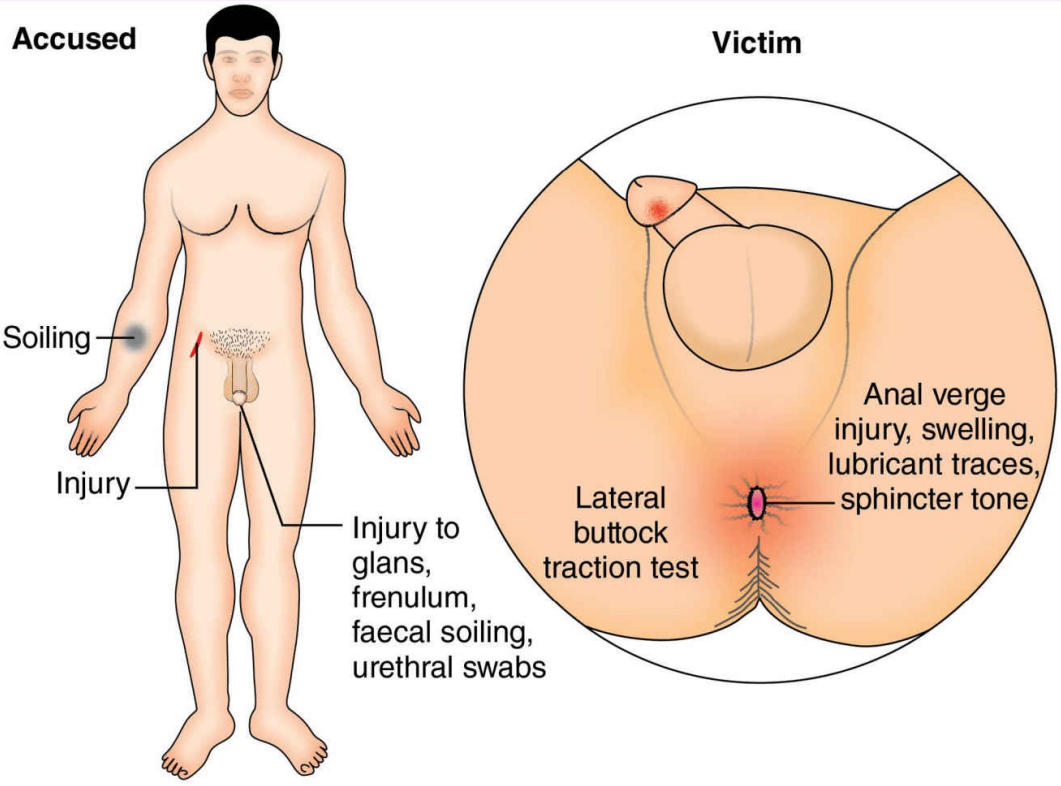


Fig. 25.10: Examination of victim and accused of sodomy. To be made on the same pattern as examination of victim earlier.

a. Findings in a first time or occasional passive agent

(1) **Buttocks** - Note fresh or healed lesions, dried secretions, ecchymoses, rashes, STD lesions, handprints or fingerprints.

(2) **Perianal skin** - Examine for presence of inflammation. Record findings of dried secretions, bruising, tears, lacerations, fissures, tears, or lacerations that are located on the external surface, internal to the sphincter, or extend across the pectinate line, which is the juncture between the anal mucosa and the anal epithelium.

(3) **Anal orifice** [Fig 25.11]: (i) irritable (ii) mucosal rim - prolapsed (iii) tender to touch (iv) see how it admits index finger - (a) only one finger is admitted without discomfort - normal (b) only one finger is admitted with discomfort - full act has not taken place (c) two fingers are admitted with slight discomfort - full act might have taken place.

(4) **Anal laxity:**

(i) Note dilation and loss of elasticity [loss of normal anal tone].

(ii) Estimate or measure the diameter of any anal dilatation.

(iii) **Lateral buttock traction test** [syn, **'O' sign**, **reflex relaxation of the anus**] **- Procedure** - (a) The buttocks are spread by the thumbs of both hands **[Fig 25.11]** causing enough lateral traction to separate the buttocks, and visualize the anus. (b) Anus dilates slightly in a habitual catamite [**positive lateral buttock traction test**]. Dilation of **>15 mm is significant, and** up to **40-50 mm [4-5 cm**] may occur in a habitual catamite. Rectum can be seen through it. (c) Anus contracts slightly in normal persons [**negative lateral buttock traction test**].

(5) **Blood stains** - around anus, on perineum and on clothes. If occult blood is suspected, perform a **guaiacum** [or any other presumptive] test for blood [**ch 29**].

(6) **Fecal matter** - around anus, if there has been incontinence

(7) **Hematoma** - Frequently seen (i) as a localized swelling, or (ii) as a diffuse swelling around anal margins. There may be obliteration of normal anal skin folds

(8) **Abrasions:**

(i) scratch abrasions varying in number.

(ii) Extend from anal margin into the anus itself.

(iii) May be present at any side around the anus.

(iv) If specks of blood are adherent, it suggests recent act

(9) Anal canal - look for any foreign bodies

(10) **Lacerations of anal canal:**

(i) Seen if violence has been used, especially if the victim is young

(ii) rare in adults

(iii) best seen by pulling the skin outwards [**Fig 25.11**].

(iv) Cause - overstretching of skin

(v) Appearance - (a) seen as large fissures. (b) Triangular or wedge shaped. (c) Generally single and seen at the posterior part of the anus. (d) Base is directed externally at anal ring. (e) Apex upwards and inwards. (f) may involve only external skin or may extend within the anal canal to the mucocutaneous junction.

(11) **Laceration of sphincter ani:**

(i) Seen if violence has been used, especially if the victim is young

(ii) Rare in adults

(12) **Anal prolapse**

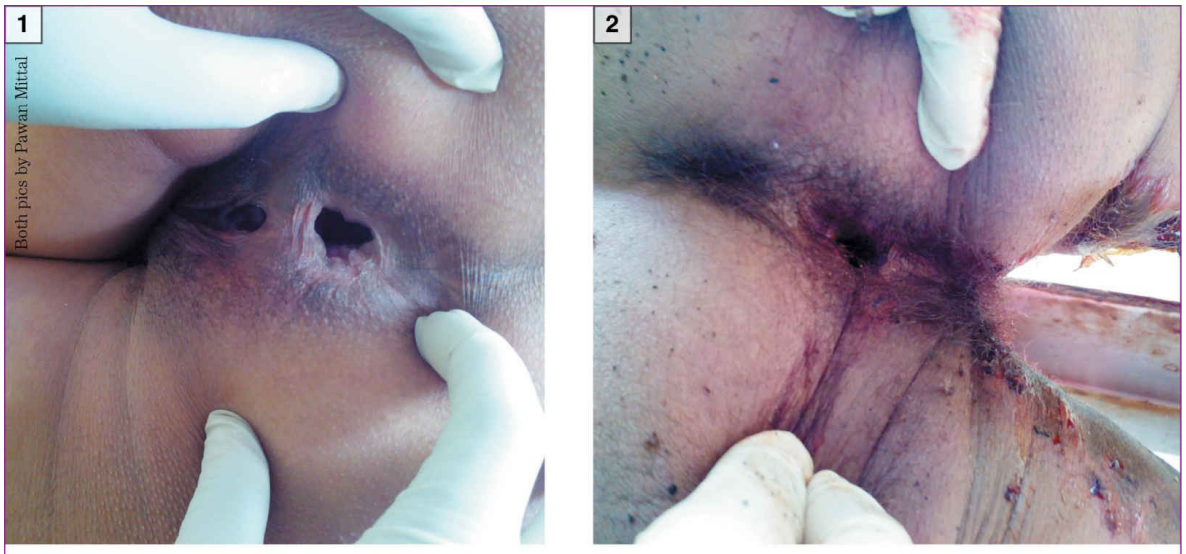


Fig. 25.11: (1) Dilated anus in a habitual passive agent [female]. Please also note anal tags, epithelization and hyperpigmentation around anus. **(2)** Normal anus. Pl note comparative anal orifices. Other characteristic features are also missing.

b. Findings in a habitual agent

(1) **Anal hair** - shaved

(2) **Anal orifice** - is dilated, inelastic, lax and overstretched. Elastic anus (i.e. one capable of dilation) does not indicate habitual agent.

(3) **Anal margin and skin around anus:**

(i) Smooth and thickened extending into the anal canal up to the mucocutaneous junction, and sometimes into the upper anal canal.

(ii) Epithelization of anal mucosa

(iii) There may be anal fissures and scars

(iv) May reveal hemorrhoids

(4) **Anal muscles** - tone is lost. Anal reflex [contraction of the anal sphincter on irritation of the anal skin] is delayed.

(5) **Funnel shaped anus** - is rare, and is usually an anatomical variation. However slight depression of buttocks towards anus may be seen [due to absorption of fat]

(6) **Lateral buttock traction test:** Please see above.

(7) **Anal mucosa** - absence of fine wrinkles

(8) **Venereal disease** - Anal condyloma of venereal disease [condylomata acuminata] and signs of and anal neoplasia may be present.

C. Examination of the Active Agent

Examination is conducted on same general lines as that of accused of rape.

1. Preliminaries

(1) **Consent** - Must be taken. If arrested, may be examined without consent **u/s 53, CrPC** [**ch 11**]

(2) **History** - Active agent is usually the accused. His version of details of the event must be recorded.

2. Examination of clothes

(1) **Disheveled and torn** - if passive agent resisted

(2) May show **seminal** and **fecal** stains.

3. General examination

(1) Injuries consistent with resistance and struggle faced - eg abrasions, bruises, teeth bite marks.

(2) Blood and seminal stains on the body.

4. Local examination

(1) **Penis:**

(i) May be elongated and constricted at some distance from the glans.

(ii) Urethra may be twisted.

(iii) Redness and swelling due to friction and irritation

(iv) Traces of fecal matter and lubricant

(v) Peculiar smell of feces and anal glands

(vi) Abrasions on prepuce, glans penis

(vii) Tearing of frenulum

(viii) Coronal sulcus - shows fecal soiling, blood, foreign hair

(2) **Urethra:**

(i) Urethral swabs - Smears should be taken from the external meatus after applying pressure on the undersurface of the penis along the urethra. May show fecal material

(ii) Culture - May show organisms [eg gonococci] similar to those found on the anal verge swabs from passive agent [corroborates penetration]

D. Specimens to be Collected

In both active and passive agents. The agents may have interchanged roles if it was a consensual act.

(1) **Fluids:**

(i) Blood - for venereal diseases, testing for HIV

(ii) Urine

(2) **Nail** scrapings

(3) **Hair** - (i) head hair (ii) pubic hair (iii) loose hair and fibres anywhere on body

(4) **Swabs:** (i) from any soiled areas of skin (ii) from anal, perianal and lower rectal area [from suspect passive agent] (iii) from shafts of penis, coronal sulcus and glans [from suspect active agent].

VII. SEMINAL FLUID

A. Collection of Material

(1) **Fluid and dried semen from vaginal canal and body surface:** Please see above under "Examination of victim"

(2) **From condoms** – use throat swab

(3) **From cloth** – eg panties, undergarments, rugs, carpets, sari, salwar etc. Cut out the stained portion. Dry (to prevent fungal contamination) and preserve

(4) **From smooth impervious surface** – eg cement and wooden floor etc. Scrape with the tip of knife and preserve in an envelop.

B. Physical Examination

(1) **Appearance** – Dry seminal stains are grayish white in color, and show an irregular map like outline

(2) **Feel** – Cloth is stiff as if starched

(3) **Odor** – Characteristic

(4) **Under UV light** – Shows bluish-white fluorescence [not specific, as other albuminous substances also fluoresce, eg nasal and leucorrheal discharge, detergents]. Fluorescence absent in a semen +ve sample

(i) Presence of blood along with

(ii) Stain soaked in water.

C. Chemical Examination

1. Acid phosphatase test

Method of detection: (i) Reagent is prepared by adding **α**-naphthyl phosphate and Brentamine Fast Blue B (ii) suspected stain is touched with a cotton swab moistened with sterile water (iii) reagent added to swab (iv) development of intense purple color within 2 min is +ve for semen. Any color developing after this is –ve.

2. Ammonium molybdate

Seminal fluid contains **free phosphate**. The presence of free phosphate in acidic solution can be detected by adding **ammonium molybdate** solution. In the presence of nitric acid, **yellow colored ammonium molybdophosphate** is formed after a few minutes. This test is obsolete now.

3. Barberio's test [1911]

Add saturated aqueous or alcoholic sol^{n} of picric acid to spermatic fluid. Yellow needle shaped rhombic crystals of spermine picrate are formed [**Fig 25.12**]. Reaction depends on presence of **spermine**, which comes from **prostate**. See also Puranen's test.

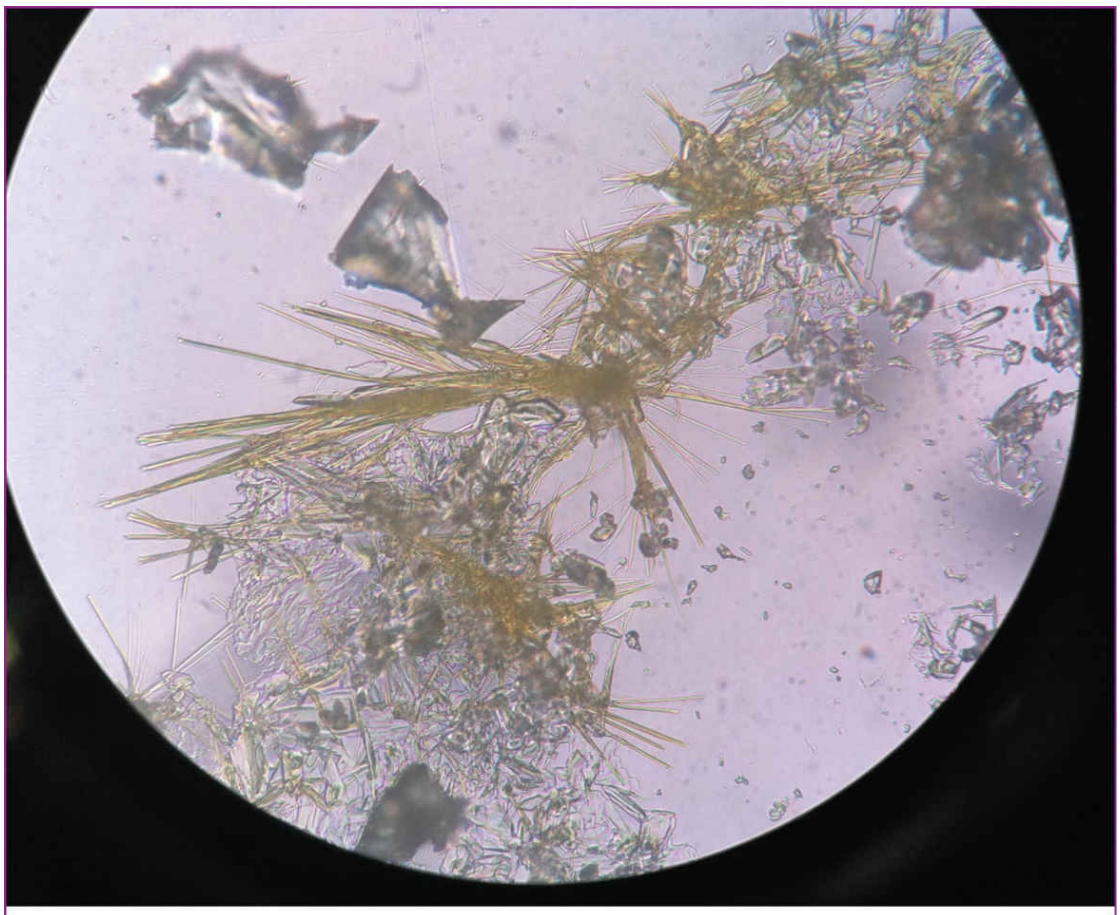


Fig. 25.12: Characteristic crystals formed in Barberio's test

4. Chromatography

(1) Choline is detected by chromatographic methods [paper chromatography, TLC].

(2) Choline and spermine are present in a unique ratio only in semen. This can be determined by TLC. 1 µL of semen can be detected by this method.

5. Creatine phosphokinase

Creatine Phosphokinase (CPK) [syn, Creatine kinase (CK), phospho-creatine kinase] acts as an energy reservoir and is expressed by several cells and tissues including spermatozoa that use large amounts of energy.

Salient features:

(1) CPK is found both in spermatozoa and seminal plasma. The CPK activity of sperm is **2.0 # 10^{-6} IU/10^{6} spermatozoa**. CPK activity of seminal plasma is

660 IU/L.

(2) The enzyme is stable and can be demonstrated even in several month old stains.

(3) Since CPK is present in several other cells [skeletal muscle, smooth muscle, brain, retinal photoreceptor cells, hair cells of the inner ear], it is only a presumptive test.

6. Florence test [1896]

(1) **Florence test** depends on the presence of **choline** in semen, which comes from **seminal vesicles**. Choline is responsible for seminal stains fluorescing bluish white under UV light.

(2) **Procedure** - To the suspect material a drop of **Florence reagent** ($KI+I_2+H_2O$) is added

(3) **Crystals** - If semen is present dark brown rhombic [or lance-like] crystals of **choline iodide** appear. They resemble haemin crystals, but can be differentiated. CI crystals are larger and arranged in clusters, rosettes, crosses etc.

(4) **Specificity** - Florence test is not specific for semen; several substances of animal and vegetable origin give +ve test. Crystals very similar to choline crystals can be made from noncoital postmortem vaginal swabs.

7. Immunological methods

a. Mab 4E6

Some seminal plasma proteins are absorbed to the spermatozoa surface [**sperm-coating antigens**]. These components can be detected both on spermatozoa and in the ejaculated fluid using **Monoclonal antibodies (Mab)**. The antibody **Mab 4E6** recognizes a **sperm coating antigen 4E6** which is secreted by the epithelial cells of the ejaculatory duct and binds to the surface of spermatozoa. Advantages of using ***Mab 4E6*** -

(1) Specific to human seminal plasma only [not found in animals]

(2) found in azoospermic patients as well.

b. SVSA

SVSA (***seminal vesicle-specific antigen***) protein is produced by seminal vesicles

and is present even in aspermic individuals. It is a substrate of p30. It can be detected by using a Monoclonal **M**ouse anti-**H**uman-**S**perm antibody no. **5** (**MHS-5**). Advantages of using ***MHS-5*** -

(1) **Stability** - It is fairly stable, and has been demonstrated even 48 h after intercourse in vaginal swabs.

(2) **High specificity** - Only primate semen shows cross reactions.

8. Prostate Specific Antigen (PSA, P30)

Prostate Specific Antigen (PSA) is a glycoprotein produced by prostatic epithelial cells.

Salient features:

(1) Detection of PSA [**syn**. p30 (so called because its mol wt is 30 kilodaltons)] is strongly indicative of semen.

(2) Also useful if the offender has azoospermia, or has undergone a vasectomy operation.

(3) Initially believed to be prostate specific, PSA is now known to be present, in lower concentrations, in various body fluids, including urine, serum, breast milk and amniotic fluid. Conc in semen is much higher.

9. Puranen's test [1936]

Detects spermine like Barberio's test. Dinitrona-phtholsulfonic acid [syn, Naphtholgelb S, Naphthol Yellow S] is used instead of picric acid. Characteristic orange microcrystals indicate semen.

10. Other methods

(1) **Glyoxalase 1** (GLO 1). It is a genetic marker found in **semen** and in several other body fluids and tissues namely RBCs, hair roots, lymphatic tissues, and dental pulp.

(2) **Oxytocinase**

(3) **Peptidase A**

(4) **Phosphoglucomutase**

(5) **Semenogelin I** and **II [SgI, SgII**], are proteins originating in the seminal vesicles and a substrate for p30, is a useful marker for the identification of semen.

(6) **Single photon fluorometry**

(7) **Sperm specific lactate dehydrogenase** [LDH-C4; LDH-X]

(8) **Trace metals in seminal plasma** – Vaginal swabs may be tested for following trace metals. Normal levels in human seminal plasma are (a) Aluminum 46 µg/dL (b) Cadmium 12 µg/dL (c) Cobalt 31 µg/dL (d) copper 46 µg/dL (e) Lead 31 µg/dL (f) Manganese 7 µg/dL (g) Nickel 106 µg/dL and (h) zinc 190 µg/dL.

Memory Aid 21: Tests for Semen

1. **Barber creat**es **gli**stening **luc**id ha**i**r
2. **Semen p a** sses **f r o m p**r o **s t a t** e

Barber – Barberio's test

Creat es – Creatinine phosphokinase

Glistening - glyoxalase

Lucid - leucine aminopeptidase

Ha **i** r - Immunological

Semen - Semenogelin I and II

P eptidase A, **P**GM

Acid phosphatase

S

S

E

S

Florence, **F**ISH

RNA based assays

Oxytocinase

Ga **M** ma Glutamyl transpeptidase [GGT]

PSA, **P**uranen's test

r

o

Sperm specific LDH

Trace metals

Ammonium molybdate

Chroma **T**ography

E

D. Techniques using Fluorescence

Fluorescent techniques which detect some specific component of sperm [DNA

sequence, sperm antigens] are as specific as detection of a sperm itself, but far easier.

1. Fluorescent in situ hybridization (FISH)

FISH (***fluorescent in situ hybridization***) is a cytogenetic technique used to detect and localize the presence of specific DNA sequences on chromosomes. **Fluorescent probes** are used that bind to **specific DNA sequences** eg those from Y-chromosome only.

2. Immunofluorescence

Several fluorescent dyes, each with their own ideal performance parameters, have been introduced for use in immunofluorescence.

a. SpermPaint

SpermPaint, is an immunofluorescent technique that uses **two mouse monoclonal antibodies**, each coupled to an **Alexa 488 dye**, to fluorescently label the **equatorial segment of the sperm head** and the **sperm tails** within a given sample.

b. SPERM HY-LITER

SPERM HY-LITER^{TM} (by Independent Forensics of Illinois, Lombard, IL) is a novel kit similar to **SpermPaint.** It contains a proprietary **human sperm–specific mouse monoclonal antibody**, combined with a **fluorescent Alexa 488 dye** so that the entire head of the sperm cells fluoresces when viewed under a microscope.

E. Microscopic Examination

Detection of spermatozoa on microscopy is the ***confirmatory test*** for semen.

Salient features:

(1) **Problems associated with microscopy:** (i) it is of no use in azoospermia [found in 10–15% cases dealt with by the Forensic Science Service], vasectomized accused, or if condom has been used (ii) difficult to visualize intact spermatozoa in stain samples, particularly where semen adheres to clothes (iii) sperm can disintegrate if the stain is washed or comes into contact with another object.

(2) **Procedure** – Specimens may be a swab from vaginal canal, or other material (eg fabric stained with semen). Procedure is slightly different for each.

1. Swab from vaginal canal

(1) Both dry and wet specimens are examined.

(2) In a **dry specimen**, the vaginal swab is smeared on a glass slide, stained and then examined for spermatozoa. Sperm motility cannot be seen by this method. At least one intact spermatozoa must be visible to confirm emission. It is because both sperm head and tail may be mimicked by a number of other artifacts. Look for microorganisms for venereal disease [details above]. If present send wet specimen for **culture**. If accused is infected with the same organism, it forms a corroborative evidence.

(3) **Wet specimens** are used for sperm motility

(i) Procedure - (a) Moist swab (or a drop of mucus removed from vagina) may be touched with a glass slide (b) Dilute with a drop of normal saline and look for motile sperms.

(ii) Interpretations - (a) Motile sperms – (I) In vaginal samples are found up to 24 hours [100% are motile up to 3 hours; 50% are motile by 8 hrs; 10% are motile by 24 hrs] (II) in endocervix up to 6 days. (b) Non motile sperm heads are detected in the living (i) up to 7 days in the vaginal cavity, (ii) 2-3 days in the anus and rectum, and (iii) 24 hours in the mouth.

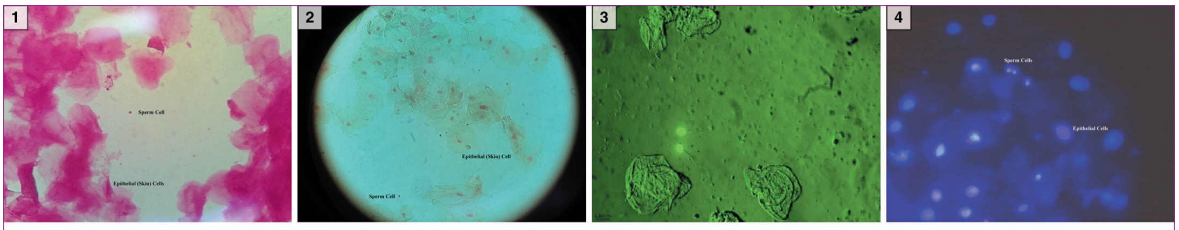


Fig. 25.13: Spermatozoa and vaginal epithelial cells as they appear with **(1)** Baecchi stain **(2)** Christmas tree stain **(3)** Sperm Hy-Liter using FITC filter and **(4)** Sperm Hy-Liter using DAPI filter.

2. Stained fabric or other material with dried stains

(1) The specimen is allowed to moisten with a few drops of 1% HCl or 3% acetic acid in a watch glass.

(2) Keep for 1 hr if stain if fresh; 4 hrs if old.

(3) Maximum concentration of sperms is in the central portion of stain.

(4) Prepare a film by rubbing the moistened portion of fabric on a slide.

(5) Dry in air without heat or fix for 1 minute in methyl or ethyl alcohol.

(6) **Stains used for spermatozoa**

(i) **Baecchi Stain** - A combination of **methylene blue** and **acid fuchsin**. Stains sperm heads pink, and nuclei blue. Developed by **Baecchi** in **1909**. (ii) **Christmas tree stain** [syn, **Kernechtrot-Picroindigocarmine Stain**, **KPIC**]. Most commonly used. Consists of two dyes. **Nuclear Fast Red dye** [Kernechtrot Solution] stains nucleus **red** and **Picroindigocarmine** (PIC) stains most other cellular material (e.g. epithelial cells) various shades of **green** [Appearance is much like red ornaments on a green Christmas tree] (iii) **Methylene blue** for 30 min and counterstain with **eosin** for 2 min or (iv) **Haemalum** [soln of hematoxylin and alum] 5 min and counterstain with **eosin** for 2 min **[Fig. 25.14]**. [Note to editor – Please check the number of figure number manually, because computer file is not being accepted]

(7) For all stains, anterior **2/3rd** of head (occupied by acrosome) is unstained or very faintly stained; only the posterior **1/3rd** occupied by nucleus is stained.

(8) **Older stains** – are less likely to reveal intact spermatozoa

(9) **Note co-ordinates** – If only one or two sperm heads are found, their co-ordinates should be noted and entered in the report. MLC number or PM number must be etched on the glass slide with a glass marking pencil.

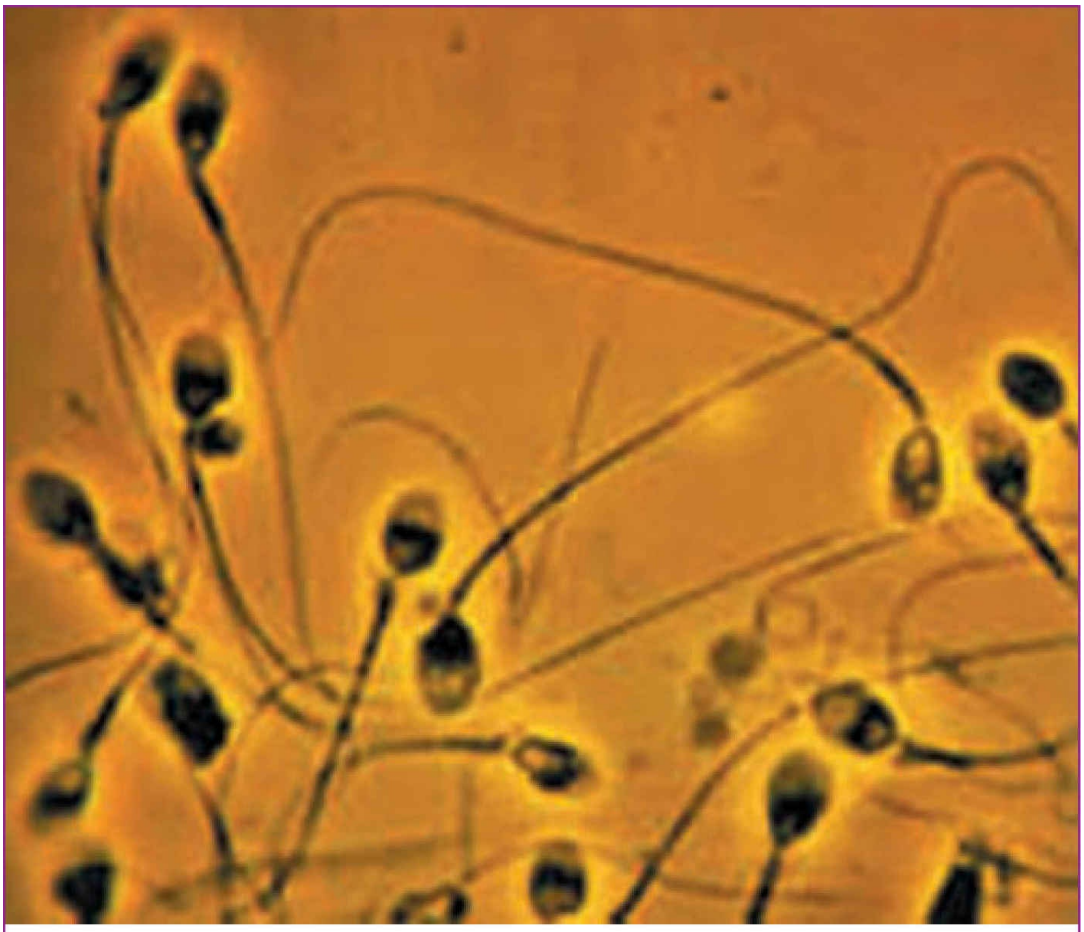


Fig. 25.14: Stained spermatozoa

26. Abortion

I. INTRODUCTION

[A] ***Legal definition***: ***Abortion*** (***miscarriage***) is premature expulsion of fetus from the mother's womb at ***any time of pregnancy***, before full term of pregnancy is completed (***S.312, IPC***). In medicolegal practice, periods during gestation are not indicated in calendar months [as they vary]. They are always indicated in lunar months [each lunar month = 28 d], or weeks [called postmenstrual weeks or PMW], or days. Thus in medicolegal language, normal

pregnancy lasts 10 lunar months, 40 weeks or 280 days. All periods are counted from LMP. Since ovulation occurs 14 days after LMP, actual physical existence of fetus within the womb is only 266 days. [**Fig 26.1**].

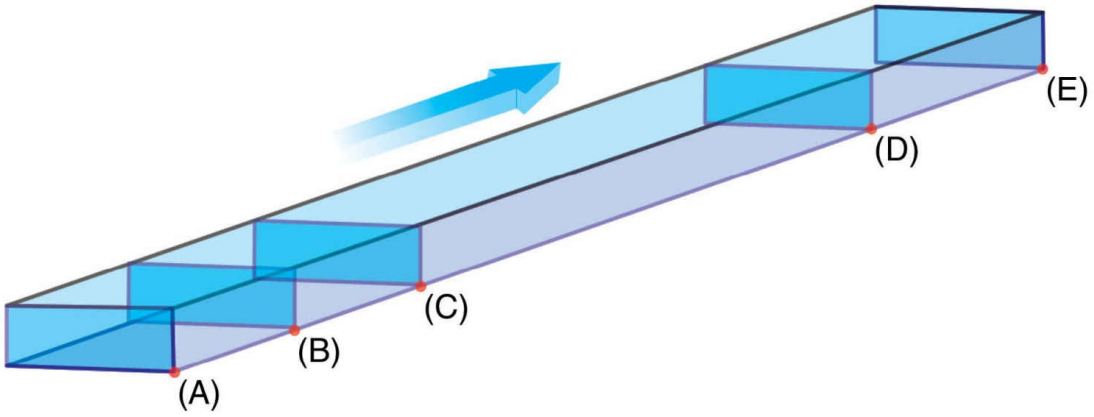


Fig. 26.1: Most important pregnancy related periods. PMW = Post menstrual weeks. (A) LMP (0 PMW) (B) Ovulation and fertilization (2 PMW) (C) 1st missed period (4 PMW) (D) Age of viability (28 PMW) (E) Parturition (40 PMW). AE = Nomal duration of pregnancy from LMP = 280 days = 40 wks; BE = Actual physical existence of fetus = 266 days = 38 wks.

[B] ***Medical definition***: ***Abortion*** is premature expulsion of fetus from the mother's womb ***before the age of viability*** (28 weeks). After this period, it is ***stillbirth***.

II. IMPORTANT TERMS

(1) **Preembryo** - Conception till the end of 5 post-menstrual week [PMW].

(2) **Embryo** – Start of 6^{th} till end of 9^{th} PMW.

Memory Aid 1: Stages in fetal life

• Remember the mnemonic from **ch 3** for **ossification of radius [**2 " 5 " 6 " 9**].**

• Now replace 2 with 0 **[**0 " 5 " 6 " 9**].**

• **0-5 PMW " Preembryo [**First 7 days of this stage is sometimes referred to as a ***"Developing ovum".*** This is time when implantation occurs.

• **6-9 PMW " Embryo**

• **10 PMW - delivery " Fetus**

(3) **Fetus** – Start of 10th PMW until delivery.

III. CLASSIFICATION OF ABORTION

[A] **Clinical classification:** (1) **Complete**- Expulsion of all products of conception (2) **Incomplete** - Expulsion of some products of conception (3) **Induced**- Termination of pregnancy for medical or elective reasons (4) **Inevitable** - Vaginal bleeding or rupture of the membranes accompanied by dilation of the cervix (cf threatened) (5) **Missed**- Undetected death of an embryo or a fetus that is not expelled and that causes no bleeding (also called blighted ovum, anembryonic pregnancy, or intrauterine embryonic demise) (6) **Recurrent** or habitual- Three or more consecutive spontaneous abortions (7) **Septic**- Serious infection of the uterine contents during or shortly before or after an abortion (8) **Spontaneous** - Non induced abortion (9) **Therapeutic** - Termination of pregnancy because the woman's life or health is endangered or because the fetus is dead or has malformations incompatible with life (10) **Threatened** - Vaginal bleeding occurring before 20 wk gestation without cervical dilation, indicating that spontaneous abortion may occur (cf inevitable) [B] **According to method induced** (1) **Natural** - (a) Spontaneous (b) accidental (2) **Artificial** – (a) Therapeutic or Justifiable (safe. Conducted by qualified personnel) (b) criminal (unsafe. Conducted by quacks, by unrecognized methods in unhygienic surroundings) [C] **According to time period** (1) **Early** - before 12 wk gestation (2) **Late** - after 12 wk gestation.

IV. NATURAL ABORTION

A. Epidemiology

(1) **Incidence - 15%** among clinically recognized pregnancies. **2-3** times higher among very early and unrecognized pregnancies

(2) **Past obstetric history** - incidence higher among women with prior abortions

(3) **Maternal age** – Incidence -s with maternal age from 12% in women < 20 years to over 50% in women > 45 years

(4) Fetal age – Most common at 2-3 months.

B. Causes

1. Embryonic factors

(1) **Chromosomal abnormalities** - Cause of majority of early abortions. Autosomal trisomies are most common (51.9%). Due to meiotic nondisjunction during gametogenesis in normal parents. (2) Abnormal germ cells (3) defective implantation (4) defects in the developing placenta or embryo (5) accidental injuries to the fetus (6) **Anembryonic pregnancy** (7) **Empty gestational sacs** - or "blighted ova" represent pregnancy failures with subsequent embryonic resorption. (8) Low implantation of zygote. (9) Disease of decidua or placenta.

2. Parental factors

(1) One parent is the carrier of a balanced translocation - Chromosome translocation is caused by rearrangement of parts between nonhomologous chromosomes. When there is equal exchange of material it is balanced; when unequal, resulting in missing genes it is unbalanced. (2) Infections (3) cigarette smoking (4) diabetes mellitus (5) emotional disturbances (6) hormonal deficiency (7) hypertension (8) nephritis (9) Rh incompatibility (10) sudden shock (11) syphilis (12) toxicity [arsenic, lead, other heavy metals, cantharides, drugs, ethanol, occupational exposure to benzene, gasoline, H_2S] (13) uterine anomalies - malformed uterus, retroverted uterus, submucous uterine fibroid, uterine hypoplasia.

V. THERAPEUTIC VS. CRIMINAL ABORTION

[A] ***Therapeutic abortion*** is that which is done to save the life of woman. It is done by qualified personnel. [B] ***Criminal abortion*** is unlawful expulsion of products of conception from the uterus, i.e. outside of the provisions of MTP Act 1971. It may include (1) abortion by unqualified personnel (2) abortion for indications other than those mentioned in the Act and (3) abortion after 20 weeks of pregnancy (excluding those conducted to save the life of woman). Criminal abortions come to light when the woman dies or some one gives information to the police. (4) **Abortionists** are traditionally divided into three categories (i) **Expert** or **medically qualified** (ii) **Semi-skilled** – nurses, midwives, chemists

etc who have rudimentary knowledge of human anatomy and (iii) **unskilled**.

VI. LEGAL ASPECTS OF ABORTION

A. S.312, IPC

(1) Scope - Abortion can only be done to save the life of a woman. Abortion done for any other purpose would attract this section

(2) Consent of the woman should be there

(3) Woman herself is punishable under this section, if she performs abortion on herself

(4) Punishment – (a) Before quickening - Imprisonment of 3 y or fine or both (b) After quickening - Imprisonment of 7 y + Fine.

B. S.313, IPC

(1) All aspects remain same as above, except that

(2) Consent of the woman is not there

(3) Punishment – Imprisonment of 10 y + Fine [whether quickening has occurred or not]

C. S.314, IPC

(1) All aspects remain same as in S.312, except that

(2) Death of the woman occurs

(3) Punishment – (a) If consent of woman was there - Imprisonment of 10 y + Fine. (b) If consent of woman was not there – Life imprisonment + Fine.

(4) It is no defense to the abortionist that he did not know that his act could cause death. **Table 1** summarizes these three sections.

Table 1: Table illustrating various IPC sections related to criminal abortion. Remember that as one goes from left to right of the table or from top to bottom, the offence become graver and more serious.

		Consent Yes	Consent No
Death No	Not quick	S.312→3 years or fine or both	S.313→ imprisonment for life or for 10 years *plus* fine
	Quick	S.312→7 years *plus* fine	
Death yes	Quick or not quick	S.314→10 years *plus* fine	S.314→ imprisonment for life or for 10 years *plus* fine

D. S.315, IPC

(1) Scope – The act must be done (a) with intent to prevent child being born alive or (b) to cause it to die after birth (c) If act is done to save the life of woman, than this section is not attracted

(2) Punishment – Imprisonment of 10 y or fine or both

(3) Difference from previous sections - Here the length of pregnancy is typically > 28 weeks (age of viability) [**Fig 26.2**].

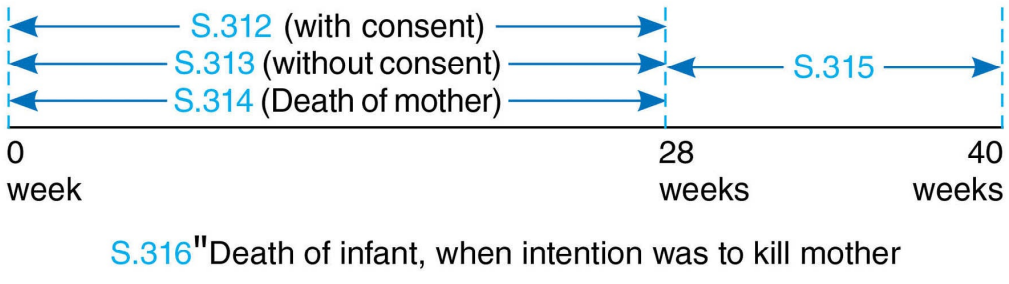


Fig. 26.2: Various sections of IPC related to criminal abortion.

E. S.316, IPC

(1) Scope – Primary aim was to kill mother, but death of her quick unborn child is caused. She remains unharmed

(2) Punishment – Imprisonment of 10 y or fine or both.

F. S.511, IPC

Failed attempt – In all the above sections, if the persons fails in his attempt to cause abortion, he would still be punishable with half of the imprisonment prescribed under that section.

VII. THE MEDICAL TERMINATION OF PREGNANCY ACT, 1971

A. Indications

Listed **u/s 3** of **MTP Act, 1971**. For convenience of study, they are grouped under following headings. The law nowhere mentions these headings (1) **Therapeutic** – When the continuance of the pregnancy would involve a risk to the life of the pregnant woman or of grave injury to her physical or mental health [**S.3(2)(i)**]. (2) **Eugenic** - There is a substantial risk that if the child were born, it would suffer from such physical or mental abnormalities as to be seriously handicapped [**S.3(2)(ii)**]. This may occur **(A)** Diseases - If during the first 3 months the pregnant woman suffers from (i) Chickenpox (ii) German measles (iii) Smallpox (iv) Toxoplasmosis (v) Viral hepatitis (vi) Viral infections **(B)** Drugs - If the pregnant woman is treated with (i) Aminopterin (ii) Antimitotic drugs (iii) Cortisone (iv) Thalidomide [of historical imp only] etc, or if she takes (v) antidepressants or (vi) hallucinogens **(C)** X-rays – If mother is treated with X-rays or radioisotopes **(D)** Heritable mental illness – If both parents are suffering from a heritable mental illness [eg if one parent has schizophrenia"chances of child developing it are 10-12%. Both parents have schizophrenia"chances are 40%]. (3) **Humanitarian** - When pregnancy is caused by rape [**S.3(2), Explanation 1**]. (4) **Social** - (A) When pregnancy has resulted from failure of contraceptive methods in case of a married woman [**S.3(2), Explanation 2**]. (B) When social or economic environment, actual or

reasonably expected may result in injury to mother's health [**S.3(3)**].

Memory Aid 2: Indications of MTP

SETH – **S**ocial, **E**ugenic, **T**herapeutic, **H**umanitarian

B. Other Salient Features

(1) **Under the MTP Act** -

(i) MTP rules were formed in 1975 [as mandated by **S.6** of MTP Act, 1971]. Repealed in 2003, via **rule 10** of MTP rules 2003; the new rules are called MTP rules 2003]

(ii) MTP regulations were formed in 2003 [as mandated by **S.7** of MTP Act, 1971]. (1) **Conflict with IPC** - If a physician performs an abortion under the provisions of this Act, then even if he violates any section of IPC [eg S.312-326], he will not be guilty of any offence [**S.3(1)**]

(2) **Consent** -

(i) Pregnant woman <18 y or mentally ill – written consent of guardian required [**S.3(4)(a)**]

(ii) Pregnant woman ≥18 y - can give consent [**S.3(4)(b)**]

(iii) Consent of husband – not legally required. However if woman repeatedly undergoes MTP without consulting or informing her husband, he can get a divorce under "cruelty clause" [**ch 23**] as it may amount to "cruelty to husband".

(3) **Who can terminate pregnancy** [**rule 4** of MTP Rules, 2003] – Any one who has following experience and qualifications

(i) 6 months house job in G&O

(ii) 1 y experience in the department of G&O

(iii) Holds MD or diploma in G&O

(iv) If registered as a doctor before 1971 [i.e. commencement of the Act], must have experience of **3 y** in practice of G&O [such cases are rare now due to passage of time]

(v) Has assisted in performance of 25 MTPs of which at least 5 must have been performed independently [with this experience, doctor can terminate pregnancy only up to 12 wks].

(4) **Up to what period pregnancy may be terminated** -

(i) Up to 12 wks – Opinion regarding existence of indications may be expressed

by 1 doctor [**S.3(2)(a)**]

(ii) 12-20 wks – Opinion must be 2 doctors or more [**S.3(2)(b)**].

(iii) >20 wks – please see below.

(5) W**here pregnancy may be terminated** –

(i) In a hospital established or maintained by Govt [**S.4(a), MTP Act 1971**]

(ii) In a place approved for MTP by (a) Govt or (b) District Level Committee [DLC] constituted by Govt [**S.4(b), MTP Act 1971**]

(6) **Emergency provisions** – If MTP is required to save the life of a woman

(7) **Pregnancy caused by rape,** woman has not lodged complaint with police, wants MTP -

(i) MTP can be done

(ii) Doctor must report the matter to police, preserve the products of conception and hand it over to police for DNA profiling. Please see **ch 24**.

VIII. METHODS OF PROCURING CRIMINAL ABORTION

Methods differ according to different periods of pregnancy. They become more drastic as pregnancy advances.

(1) **Up to the end of 1st month** – When woman has not missed a period, but believes she may have become pregnant (i) Violent exercise (ii) hot baths (iii) purgatives

(2) **After missing the first period up to the end of 2nd month** – Suspicion becomes a surety now. More drastic measures are adopted (i) Abortifacient drugs

(3) **After 2nd month till parturition** – Methods become more drastic; woman may resort to mechanical interference.

Memory Aid 3: Methods of Criminal Abortion

Criminal abortion violates local mores
Abortifacient drugs
Violence
Local methods
Miscellaneous

A. Abortifacient Drugs

1. Drugs acting directly on the uterus

a. Ecbolics

Ecbolics [Gk ec, away from; ballein, throw] are drugs that increase uterine contractions.

Salient features:

(1) **Actions** - -contractions, but do not relax or dilate the cervical canal and external os, necessary to expel the fetus. Thus dangerous to use.

(2) **Drugs used:** (i) Apiol – Active principle of common parsley [Petroselinum sativum] (ii) Ergot - (a) Most common ecbolic (b) Side effects – arterial spasm and gangrene of extremities (c) Frequently fails during earlier months of pregnancy (iii) Goldenseal [Orange-root, Orangeroot; Hydrastis canadensis]- (a) perennial herb (b) Acts both as oxytocic [due to hydrastine and hydrastinine] and emmenagogue. It is a stronger ecbolic than emmenagogue (iv) Hormone preparations – Synthetic estrogens (v) Lead - Taken in the form of pills made from diachylon [lead oleate] or lead plaster (vi) Pituitary extract - (a) No effect when given by mouth; effective only by injection (b) Effect is significant only near term (vii) Quinine - was in popular demand among young service women during the 2^{nd} WW for abortions. Caused serious toxicity. (viii) Savin [Juniperus sabina] – useless in small doses (ix) Miscellaneous - (a) Decoctions of cotton root bark (b) Nitrobenzol (c) Picrotoxin (d) strychnine.

b. Emmenagogues

Emmenagogues are drugs which increase the menstrual flow.

Salient features:

Two types:

(1) Direct – which directly stimulate the uterus [used more often for criminal abortion]. **Ex** – Aloes, Anemone, Apiol, Asarum arabicum, Borax, Broom, Cantharides, Caulophyllin, Cimicifuga, Grains of paradise, Hellebore [white and black], Juniper, $KmnO_4$, Laburnum, Male fern, Manganese dioxide, Mentha pulegium, Myrrh, Nutmeg [**ch 46**], Pennyroyal, Polygala, Pulsatilla, Racemosa, Rue, Saffron, Sanguinarin, Santonin, Savin, Senecio, Senega, Squills, Tansy, Thuja and Yew leaves.

(2) Indirect – which correct anaemia, or disturbed pelvic circulation, etc. [used more often for treatment of amenorrhoea as folk medicine]. **Ex** – Tonics and

hematinics eg iron.

2. Irritants of the genitourinary tract

(1) **Applied locally** - they produce reflex uterine contractions.

(2) **Taken systemically** - produce inflammation of kidneys.

(3) **Ex** (i) cantharides (ii) Oil of pennyroyal (iii) Oil of tansy (iv) Potassium permanganate crystals, solution or tablets [100-300 mg]. (a) May cause acute local punched out ulcers with raised edges and granular black base. (b) May erode arterial vessels and cause severe hemorrhage.

3. Irritants of the GIT

(1) Any substance which produces irritation of colon, may produce hyperemia and contractions of uterus also

(2) **Emetics** – Tartar emetic

(3) **Saline cathartics** [$MgSO_4$]

(4) **Strong purgatives** - (i) Aloes (ii) Calomel (iii) Castor oil (iv) Colocynth (v) Croton oil (vi) Elaterium [syn, elaterin. Plant extract from the juice of the fruit of Ecballium Elaterium, the squirting cucumber] (vii) Gamboge [resin from various species of trees of the family Guttiferae] (viii) Jalap (ix) Phenolphthalein (x) Podophyllum (xi) Rhubarb (xii) Scammony [Convolvulus scammonia] (xiii) Senna.

4. Local application of irritants

(1) **Corrosives** – eg carbolic acid, oxalic acid etc

(2) **Irritants** – All of the above may be applied locally also

5. Systemic poisons

Rationale is that at sublethal doses, the drug may be lethal to fetus which is much more susceptible

(1) **Inorganic irritants** - eg salts of antimony, copper, iron, lead and mercury.

(2) **Organic irritants** - Barks [eg of plumbago rosea], cantharides, Caryophyllus, juices [eg of calotropis], methi, saffron, seeds [eg of carrot, Moringa (a flowering plant)] and unripe fruits [eg of papaya, pineapple etc].

B. Violence

Violence may cause abortion by (1) producing congestion of pelvic organs (2) producing hemorrhages between uterus and membranes (3) dislocation of placenta.

1. Direct violence

Force is directly applied over lower part of abdomen by following methods (1) Kicks and blows (2) Kneading (produces severe pressure on the abdomen) (3) Massage of uterus through the abdominal wall (4) Tight lacing (wearing a tightly-laced corset).

a. Cupping

(1) This is a special method of direct local violence used in advanced pregnancy.

(2) Method - A wick is placed over hypogastrium and lighted A mug is placed over it, mouth downwards Lighted wick consumes oxygen producing partial vacuum mug is pulled upwards mug sticks to the hypogastrium due to partial pressure forcible pulling separates placenta.

2. Indirect violence

Force is indirectly applied over lower part of abdomen by such activities as (1) Carrying or lifting heavy weights (2) Cycling (3) Driving over rough roads (4) Horse riding (5) Jumping from a height and running upstairs and downstairs

C. Local Methods

A **tenaculum** is often used in following procedures to steady the cervix and uterus. Its marks are often found on the uterus during examination.

1. Abortion pastes

(1) **Iodine** containing pastes:

(i) **Utus paste** - A mixture of soap, myrrh resinoid and potassium iodide. Iodide acts as an irritant causing abortion.

(ii) **Interruptin** - contains elemental iodine. The risks were the same as with "Utus paste"

(iii) **Fetex Paste** (brand name) contains benzoin, iodine, thymol, potassium iodide and saponified vegetable oil paste.

(2) **Lead** containing pastes - **Diachylon** - contains lead salts (e.g. lead oleate) and oil.

2. Abortion stick

Abortion stick is a thin wood or bamboo stick about 12-18 cm long [**Fig 26.3**].

Salient features:

(1) **Construction:** (i) Traditional stick - At one end of the stick, a piece of cloth or cotton wool is wrapped, which is soaked with one or more irritants eg, Abrus precatorius, arsenic sulfate, arsenious oxide, asafetida (Ferula asafetida), marking nut juice, mercuric chloride, red lead etc. (ii) Variation – Instead of stick, a twig of some irritant plant may be used, eg Calotropis, Cerbera thevetia, Nerium odorum, Plumbago rosea or zeylanica

(2) **Method of use** - Introduced into the uterus and retained there till uterine contractions begin.

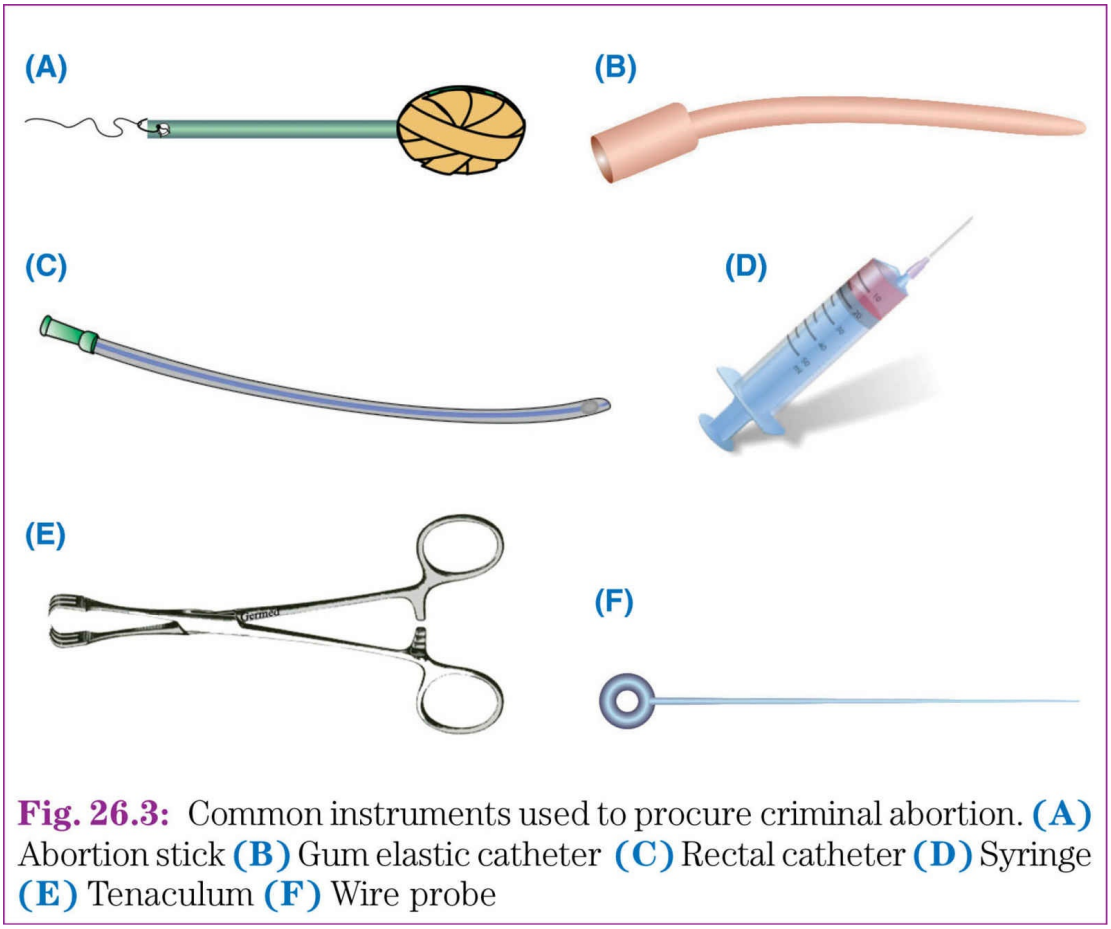


Fig. 26.3: Common instruments used to procure criminal abortion. **(A)** Abortion stick **(B)** Gum elastic catheter **(C)** Rectal catheter **(D)** Syringe **(E)** Tenaculum **(F)** Wire probe

3. Air insufflation

Air is introduced into the uterus through vagina by means of douche tips,

orogenital contact, pumps and syringes in a false belief that air would dislocate placenta causing abortion. This rarely happens. May cause death by air embolism.

4. Curettage

More like a professional D&C. Done only by experienced criminal abortionists.

5. Dilation of the cervix

Dilation of cervix expels the contents of the uterus. Generally used after 8 wks of pregnancy. Methods used are:

(1) **Laminaria tent** [syn, Laminaria stick, sea tangle tent]:

(i) A cone [tent] or thin rod of dried Laminaria digitata [large seaweeds belonging to the brown algae group].

(ii) Availability - in a box containing 6 laminaria sticks in liquid. Each one is 8 cm in length; diameters measure 3, 4 and 8 mm. A white thread is attached to one end for easy removal.

(iii) Laminaria sticks are inserted into the cervix" over the course of several hours, they slowly absorb water and expand.

(iv) Introduction of several thin sticks was considered preferable to one with a larger diameter. The latter became so tight with expansion that its removal could become impossible, even with threads.

(v) Causes dilation of cervix and expulsion of uterine contents.

(vi) Also contains iodine, which is a uterine irritant.

(2) **Slippery elm bark** - It is the inner bark of Ulmus fulva, a tree which grows in Central and North America. Contains mucilage. Pieces of bark are inserted into the cervical canal. Absorbs water and swells to a slippery jelly. Dilates the cervix. Acts as a mechanical irritant and lubricant also.

(3) **Miscellaneous methods** – Introduction in the cervix of (i) Pessaries (ii) sponge and leaving them there.

6. Electricity

(1) **Placement of poles**: Generally **+ve** pole over the lumbosacral region; **-ve** over the cervix [or posterior vaginal cul-de-sac], but can be done other way round also

(2) **Low voltage current** is passed"Uterus contracts and expels contents.

7. Gum elastic bougies

(1) In more advanced pregnancies **gum elastic bougies**, **catheters** or **balloons** are introduced through the cervix into the uterus [**Fig 26.3**].

(2) **Mechanism** - irritation of the uterus.

(3) **Complication** -Sepsis.

8. Rupturing of the membranes

(1) **Method** - Rupture of membranes is done by introduction of an instrument into the cavity of the uterus eg a cannula, catheter, crochet needle, curtain rod, douche, gloss rod, hairpin, knitting needle, nail, pen, pencil, penholder, probe, screwdriver, stick, umbrella rib, uterine sound, wire etc.

(2) **Who can do** - abortionist, woman herself

(3) **Mechanism** - rupture of membranes"escape of liquor amnii"induction of abortion

(4) **Time of abortion** - within a few hours to 3 days. May be delayed for several weeks.

(5) **Complications:**

(i) Perforation of vaginal or uterine walls

(ii) breakage of instruments of parts of instruments within the uterine cavity

(iii) Infection.

9. Syringe aspiration

Large syring is taken"a catheter or plastic tubing is attached to it"introduced within the uterus"suction applied"-ve pressure ruptures chorionic sac" precipitates abortion.

10. Syringing

(1) **Instruments used:**

(i) Ordinary enema syringe [**Fig 26.3**]. May be used for douches of hot or cold water, which are projected with considerable force towards the uterine os.

(ii) Higginson’s syringe [**Fig 26.3**]. One end is dipped into the solution, and the other into the uterus. Repeated compression of bulb pushes fluid into the uterus. If there is imperfect filling of the bulb, a mixture of air and fluid is forced into the uterine cavity at a pressure higher than that in uterine veins.

(2) **Injection material** - Any irritant fluid, eg sol^{n} of irritating chemicals in water, eg acids [inorganic], alum, cresol, formalin, $KMnO_4$, lysol, Metallic irritants [Arsenic compounds, Hg compounds eg corrosive sublimate, lead compounds], soap, turpentine etc.

(3) **Used by** - woman herself or an unskilled or semi-skilled abortionist.

(4) **Mechanism** - The fluid detaches amniotic sac and placenta from the uterine walls"uterine contraction "Abortion.

(5) **Complications:**

(i) Air embolism - **ch 11**.

(ii) Extensive local tissue destruction"fatal hemorrhage, infection.

(iii) systemic absorption of irritant substances "causing toxemia, shock and death.

(iv) Vagal inhibition - May cause sudden death. Causes are (a) Sudden and rough insertion of instruments in the uterus (b) rapid injection of unduly hot or cold liquids.

D. Miscellaneous Methods

(1) **Alternate hip baths** in very hot and cold water

(2) **Partial abortion by untrained abortionist**; completed by a trained doctor because it becomes threatened -

(i) Abortionist ruptures membranes and sends patient to doctor

(ii) Woman applied some corrosive substance to vagina (eg $KmnO_4$) to produce ulceration and bleeding; visits a doctor who completes abortion

(iii) Woman complains that her uterus is displaced; unsuspecting doctor passes a sound resulting in abortion.

IX. THERAPEUTIC METHODS TO PROCURE (SAFE) ABORTION

(1) Manual Vacuum Aspiration

(2) Dilatation and Suction Curettage

(3) Dilatation and Evacuation

(4) Other Surgical Methods

(i) Dilatation and extraction (D & X)

(ii) Induction
(iii) Hysterotomy

X. EVIDENCE OF CRIMINAL ABORTION

A. In the Living

Signs of recent abortion – same as those of recent delivery.

B. In the Dead

Following points must be proved to convict the abortionist

(1) that the dead woman was pregnant
(2) that the accused was responsible for the act which resulted in abortion
(3) that the accused intended illegal abortion
(4) that death was due to abortion.

1. PM findings

(1) Depend upon
(i) mode of abortion practiced
(ii) time elapsed between abortion and death. In sudden deaths woman may be found dead in a posture consistent with recent abortion, eg lower garments pushed up, legs spread wide apart, syringe or other instrument within genital canal, apparatus and paraphernalia strewn around.
(iii) Time elapsed between death and PM examination
(2) In all cases of criminal abortion, female genitalia must be excised out of the body and examined [**ch 5**]
(3) Maintain a full photographic record of all procedures conducted and findings.

a. Abortion by drugs

(1) **Local examination of genitals** – corrosion, erosions, inflammation of vulva, vagina, cervix etc indicate local application of corrosives and irritants. Preserve vaginal and vulval washings. Match with those found at the scene [please see above].
(2) **GIT** –
(i) look for signs of congestion, inflammation, irritant poisoning

(ii) If present – (a) indicate administration of GIT irritants. (b) Examine entire GIT carefully. (c) Preserve entire GIT contents for chemical analysis

(3) **Urinary tract** -

(i) Look for signs of congestion, inflammation, irritant poisoning

(ii) If present – (a) indicate administration of cantharides, turpentine.

b. Instrumental abortion

(1) **Air embolism**

(2) **Infection:**

(i) Seen in delayed deaths. Indicates instrumental abortion.

(ii) Vagina - walls may show pus. There may be frank discharge

(iii) Uterus - (a) Uterine sepsis usually involves endometrium, especially the placental site and pieces of retained products (b) Myometrium, tubes, adjacent pelvic organs [eg ovaries], peritoneum"infected.

(iv) Infection is rare in natural abortion.

(3) **Injuries:**

(i) Site - on vagina, cervix, uterus. Sometimes on abdomen also. There may be abrasions, bruising, foreign bodies, hemorrhage, inflammation, lacerations, perforations, punctures, ruptures etc. Bleeding in peritoneal cavity indicates puncture through and through the genital tract. In the vagina, injuries are present in the walls and especially in the posterior fornix.

(ii) Extent - Damage is more in unskilled or semi-skilled hands and also during attempts at self abortion. All organs constituting genital canal [vagina, cervix, uterus] may show perforations of various sizes and shapes. The size of perforations may vary from a small ragged pinpoint to a much larger tear of oval, round, stellate or irregular shape. Perforation of uterus indicates penetration into the abdominal cavity and abdominal injuries must specifically be searched for. Perforations, tears etc may be found on bladder, intestines [small and large including rectum], mesentery and omentum. If very long instruments [eg cycle spokes] are used, even diaphragm may be pierced causing injuries to lungs and heart.

(iii) Nature - of injuries reveal type of instrument used eg pointed, blunt, penetrating etc.

(4) **Materials to be preserved:**

(i) Fetal blood and tissues - DNA profiling would establish maternity and if

required paternity, especially if suspected father was the once who conducted criminal abortion.

(ii) Fluid from cut surface of lung and pulmonary blood - for fatty acid estimation and for phenolic derivatives depending on the agent used.

(iii) Foreign bodies - if recovered from anywhere especially within the genital canal or uterus, it must be preserved and sent for analysis. Sometimes rags soaked in irritant solutions may be recovered. Sometimes they are inserted there merely to stop post-abortion bleeding.

(iv) Instruments of abortion - (a) for toxicology [eg abortion stick] (b) for microbiology (c) for typing of blood, and whether it matches that of mother, fetus or both.

(v) Maternal Blood - (a) For toxicology and microbiology (b) for typing and matching with that found at the scene and on the instruments

(vi) Scene of crime - All suspect material found at the scene of crime, eg barks, bottles, containers, nozzles, syringes, tubes, twigs of plants etc. Material expelled from uterus.

(vii) Tissue - from vagina, uterus, cervical canal, ovaries and all major internal organs for histopathology.

(viii) Washings - from vagina, cervix, uterine cavity. For toxicology and microbiology. Alternatively entire genital system may be sent frozen for toxicology.

c. Abortion by syringing

Same as in instrumental abortion. Additional findings are

(1) **Clothes** - may be soiled with fluid

(2) **Genital canal** - may show corrosion or tissue damage due to use of antiseptics, irritants or corrosives.

(3) **Fluid** - dark red fluid found between uterine walls and fetal membranes with partial detachment of placenta. If soap has been used, fluid may be foamy.

(4) **Venous system** - Uterine, ovarian and pelvic veins and all veins extending from the sides of the uterus up to the right heart, coronary vessels, superior and inferior vena cavae and pulmonary conus may show bubbles of gas and slimy fluid. Veins may be “ballooned out”. May show segmentation and may have a “beaded” appearance. On touching, have a characteristic elastic feel. Mechanism of entry - As fluid is pushed into the uterus under pressure, it

enters dilated uterine sinusoids along with air.

2. Collection of material

Same as in living and as noted above.

XI. CAUSES OF DEATH IN CRIMINAL ABORTION

A. Immediate Deaths

Occur within minutes.

(1) Embolism [air, amniotic fluid, fat]
(2) Hemorrhage
(3) Poisoning [if poison was used to procure abortion]
(4) Vagal inhibition.

1. Amniotic fluid embolism

Amniotic fluid embolism (AFE) is condition in which amniotic fluid, fetal cells, hair, or other debris enters the mother's blood stream via the placental bed. Presence of fetal debris in the pulmonary blood vessels of a mother who had died suddenly in labor was first described by **Meyer** in 1926. In 1941, **Steiner** and **Luschbaugh** were the first to attribute sudden death during labor to AFE.

Salient features:

(1) **Occurrence** - It occurs not only during (i) criminal abortion, but also during (ii) first and second trimester abortions (iii) abdominal trauma (iv) Amniocentesis (v) normal labor and delivery (vi) immediately postpartum
(2) **Risk factors** – (i) -ed maternal age (ii) augmented labor (iii) cervical laceration (iv) cesarean delivery (v) Diabetes (vi) fetal macrosomia (vii) induction and augmentation of labor (viii) placenta previa or abruption (ix) Strong uterine contractions (x) uterine rupture, and(xi) vacuum, and forceps deliveries.
(3) **Incidence** - 1 in 8000 to 80,000 deliveries. In 50% of cases, death occurs within 1st hour of criminal abortion. In late deaths, DIC occurs
(4) **Etiology** –
(i) Mechanical blockage - pelvic trauma [inc rupture of uterus] due to instrumental interference of criminal abortion opens up venous sinuses in

the placental bed"Amniotic fluid [along with a fetal debris] enters venous sinuses"causes pulmonary microvascular obstruction.

(ii) Anaphylactoid syndrome of pregnancy – amniotic fluid constituents in circulation"release of various primary or secondary endogenous mediators [arachidonic acid metabolites, bradykinin, endothelin, histamine, leukotrienes]"severe transient vasospasm of pulmonary vasculature, pulmonary hypertension, hypoxia, right heart failure, sudden death.

(5) **Clinical features** – (i) altered mental status (ii) DIC (iii) shortness of breath (iv) sudden cardiovascular collapse (v) maternal death

(6) **Lab diagnosis** – (i) -Fibrin split products (ii) .Fibrinogen (iii) Prolonged partial thromboplastic and prothrombin times (iv) thrombocytopenia

(7) **Postmortem diagnosis** – [A] Examination of dead body (i) Examination of uterus – May reveal entrance site of emboli. (ii) Histopathology - (a) Demonstration of (I) Amniotic and Chorionic cells (II) Fat globules (III) Fetal squames (IV) Lanugo hair (V) Meconium (VI) Mucin and (VII) Vernix in pulmonary vasculature and in uterine veins. (b) Stains used are (I) Alcian Blue - detects mucin (II) H&E - routine staining (III) Attwood's stain - stains the keratin red and the mucus turquoise blue (IV) Lendrum stain contains Phloxine-Tartrazine. Detects squames by staining them red (V) Mucicarmine (VI) Sudan Black or Oil Red - Detects vernix caseosa. (c) Squamous debris may persist for some weeks after the embolic event, and it is worth looking for even in late deaths. (d) DIC - (I) deposition of fibrin microthrombi [demonstrated with Martius Scarlet Blue] and extensive bleeding in internal organs.

2. Vagal inhibition

The cervix and lower uterine segment are richly supplied with parasympathetics. Touching by instruments, irritants, or hot and cold fluids, especially in a nervous mother may prove instantaneously fatal. Anesthetization may contribute to rapid death.

B. Delayed Deaths

Occur with **2-3 days** [**Fig 26.4**]. (1) Local infection (2) Peritonitis (3) Pyemia (4) Septicemia (5) tetanus (6) Toxemia.

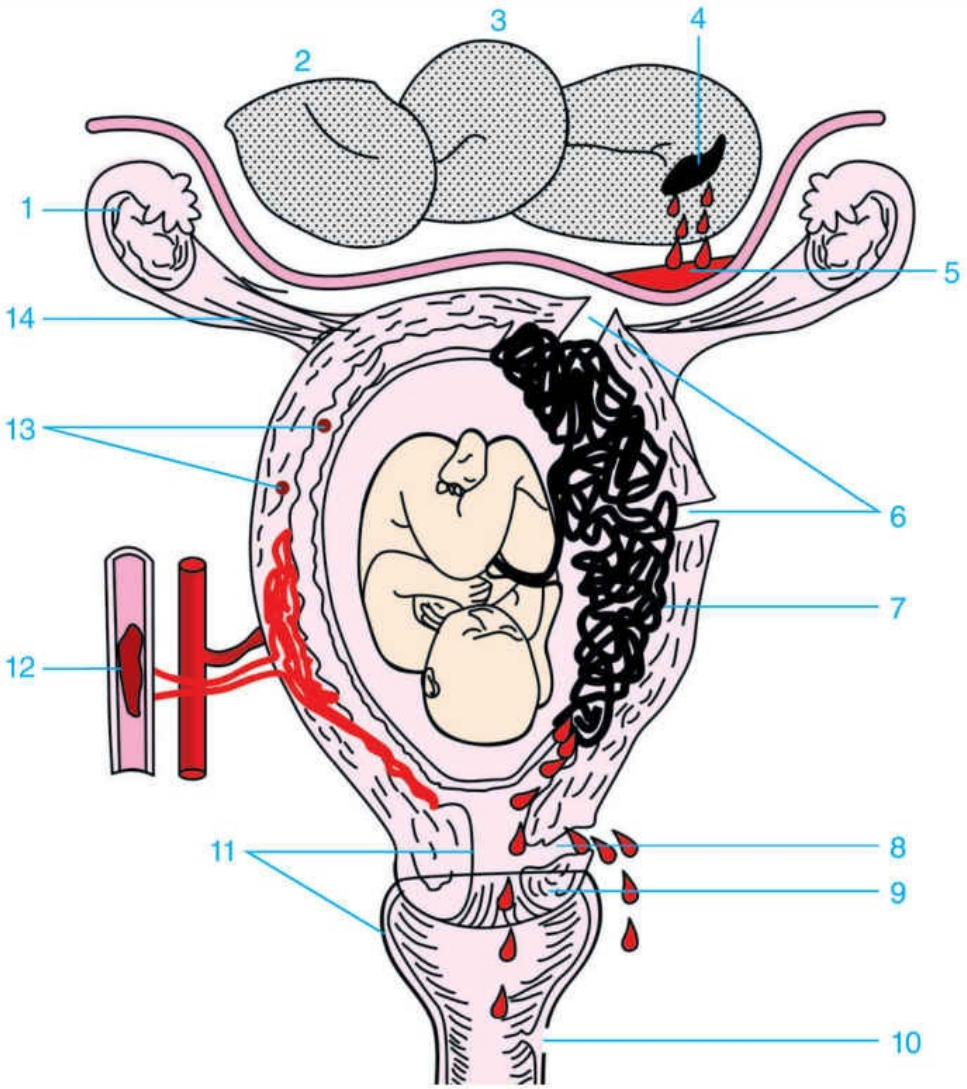


Fig. 26.4: Causes of death in **criminal abortion**. (1) Oophoritis (2) Septicemia (3) Peritonitis (4) Damage to intestines (5) Intraperitoneal haemorrhage; Shock (6) Perforation; Haemorrhage (7) Infected placental site; Retroplacental blood-clot; Septicemia; Air embolism (8) Laceration; perforation; haemorrhage into Broad ligament and Pouch of Douglas (9) Cervical necrosis and hemorrhage; cervical perforation (10) Vaginal laceration and perforation; haemorrhage (11) Infected abrasions (12) Pyaemia; Thrombophle bitis (13) Septic Endometritis and Metritis (14) Salping is. **All infections are due to:** Anaerobic streptococci; Cl. weichil; E. coli; Staph. pyogenes; Str. pyogenes

C. Remote Deaths

Occur after **weeks**. (1) Bacterial endocarditis (2) Jaundice (3) Pulmonary

embolism (4) Renal failure.

XII. ML ASPECTS OF ABORTION

A. Fabricated Abortion

Fabricated abortion is false and malicious claim of an abortion by producing a dead animal fetus. Women who have suffered trauma due to motor-vehicle accidents, or physical assault may try to exaggerate the offence [to get more compensation] by alleging that the trauma caused her to abort. A dead animal fetus, or even a dead human fetus, acquired from a hospital may be produced to support claim. Detailed examination of fetus and DNA profiling is necessary.

B. Trauma and Abortion

(1) If trauma is caused to the pregnant mother [eg motor vehicular accident, fall from height, intentional blow], abortion may be induced. In such cases it becomes necessary for the doctor to determine if trauma indeed resulted in abortion. If intention was to kill mother and abortion was caused instead, it would attract **S.316, IPC**.

(2) **Difficulties in establishing a causal relationship between trauma and abortion** -

(i) Abortion may already be in progress when trauma occurred and it merely hastened the process

(ii) Embryonic injury caused by trauma may not result in an abortion for several weeks or even months.

(3) **Criteria suggesting a causal relationship** -

(i) Fetus and placenta should be normal before trauma

(ii) Factors known to cause abortion should be absent. These include (a) Chronic infections in the mother [eg syphilis, toxoplasmosis] (b) Exposure to abortifacients [folic acid antagonists, lead, X-rays] (c) Physical attempt to induce abortion (d) Repeated h/o abortions without any cause (e) Uterine abnormalities [eg congenital defect of uterine development, endometrial polyps, incompetent cervical os, leiomyomas]

(iii) Abortion should occur within 24 h of trauma

(iv) The physical development of fetus and placenta should be compatible with

the period of pregnancy at the time of trauma

(v) There should be an adherent clot or a depression on the placental surface.

(vi) Injuries to the fetus should be compatible with the time period of trauma.

C. Miscellaneous

(1) If abortion is due to occupational exposure to poisons [benzene, gasoline, H_2S], employer is liable for compensation. Many toxic agents causing abortions [including 3 mentioned above] are included in Schedule III of Workmens' Compensation Act 1923 [WCA 1923] [**ch 2**].

(2) Wrongful life suits [please see **ch 23**].

XIII. DOCTOR'S DUTIES IN CASES OF CRIMINAL ABORTION

(1) **History taking** – Must encourage the patient to give correct history. If she is hesitant, should not force her to make a statement

(2) **Consultation** - with obstetrician

(3) **Treat her** - to the best of his ability. Should not view her as a criminal

(4) Maintain proper day-to-day records

(5) **Preservation of evidence** – (i) All soiled clothes (ii) Any foreign material collected from the genital tract [abortifacient drugs, abortion sticks, syringes, twigs etc] should be preserved and handed over to police

(6) **Professional secrecy** – must be maintained

(7) **Arrange for dying declaration** – if patient is serious

(8) **In case of death** – death certificate should not be issued. The body must be sent for postmortem.

Table 2: Differences between natural and criminal abortion

S.No	Feature	Natural abortion	Criminal abortion
1.	Cause	Predisposing disease	Unmarried girl or widow becoming pregnant
2.	Subsequent infection	Rare	Frequent
3.	Marks of violence on abdomen	Not present	Present
4.	Injuries on genital organs	Not present	Injuries [abrasions, contusions, lacerations, perforations etc] may be in vulva, vagina cervix and uterus
5.	Toxic effects of drugs	Absent	If abortifacient drugs have been used, toxic effects [local as well as systemic] would be seen. (i) **Local**"Erosions and inflammation of vagina and cervix due to local application of irritants and caustics (ii) **Systemic**"specific to the abortifacient used
6.	Foreign bodies in the genital tract	Not present	May be present
7.	Injuries and wounds on fetus	Absent	Present

27. Infant Deaths Including Battered Baby Syndrome

I. INTRODUCTION

A. Infanticide

Infanticide is unlawful destruction of a child under the age of one year by anyone. Infanticide does not include the death of fetus during labor, or when it is destroyed by craniotomy or decapitation.

B. Feticide

Feticide is the killing of fetus at any time prior to birth (**Main instances** – MTP, prenatal sex determination followed by selective abortion of female fetuses).

C. Neonaticide

Neonaticide is killing of an infant within ***24 hours*** of birth. The term was first coined by **Resnick** in 1970. The term is paradoxical, because although the term

"neonate" in obstetrics is used to denote an infant up to 28 days after birth, the term neonaticide in psychiatry and forensic medicine is used for killing of an infant within 24 hours of birth.

Table 1: Differences between medical and legal definition of infanticide

S.No	Criteria	Infanticide - Medical definition	Infanticide – Legal defintion
1.	Age of child	<1 y	<1 y
2.	Killing done by	Anyone	Only the mother, with the proviso that she must be under the effect of childbirth or lactation.
3.	Gender specificity	Person of any gender can commit infanticide	Infanticide is a gender specific law. Only a female can commit the crime of infanticide.
4.	Punishment	As for murder	As for manslaughter
5.	Verdict passed on perpetrator	Verdict of homicide	Verdict of homicide in India. In UK, jury may pass a verdict of guilty but insane.
6.	Relevance in India	In India, legal definition is identical with medial definition. All cases of infanticide, be it by the mother or by anyone else are treated as homicide. If mother is so grossly under the effect of childbirth or lactation that "she did not know what she was doing", she can take benefit of S.84, IPC [ch 28].	

II. STILLBIRTH

A ***stillborn child*** is one which is born after 28th week of pregnancy and which did not breathe or show any other signs of life at any time after being completely born [WHO]. In the UK, according to the Still-Birth (Definition) Act 1992, this period is reduced to 24 weeks.

Salient features:

(1) The child was alive in utero but dies during the process of birth.

(2) Stillbirths occur more frequently among illegitimate and immature male children in primiparae.

(3) **Incidence** – 5%

(4) **Causes** – (i) Anoxia (ii) Birth trauma [especially intracranial hemorrhage due to excessive moulding] (iii) Congenital defects (iv) Erythroblastosis fetalis (v) Placental abnormalities (vi) Prematurity (vii) Toxemia of pregnancy.

III. DEAD BIRTH

A ***dead born child*** is one, which has died in utero, and shows one of the following signs after it is completely born (1) maceration (2) rigor mortis (3) putrefaction (4) adipocere (5) mummification [**Table 2**].

Table 2: Differences between a stillborn and a dead born fetus

S.No	Feature	Stillborn fetus	Dead born fetus
1.	Period of gestation	>28 weeks	Any
2.	Condition in uterus	Was live just before start of labor pains. Died during labor in birth canal. Did not show any signs of life after being born	Was lying dead in uterus
3.	Predominance	Seen mostly among illegitimate and immature male children in primiparae	No such predominance
4.	Cardinal features	Signs of prolonged labor [bleeding into scalp, caput succedaneum, edema, severe moulding of head]	Any one or more of following signs - Maceration, mummification, Rigor mortis at delivery, ch. radiological signs
5.	Cause	Anoxia, aspiration, birth trauma, prematurity, strangulation on cord, toxemia	Congenital anomaly, ABO and Rh incompatibility

A. Maceration

Maceration (Latin macerare, to soften by soaking) is degenerative change occurring in a fetus retained in utero after death. It occurs due to the softening effect of soaking on solid tissues (**pulpy fetus**).

Salient features:

(1) The **earliest sign** of maceration is **skin slippage** (**12 hours after IUD**) [**Fig 27.1**]. The epidermis can be easily separated from the dermis by applying oblique pressure. Loss of the epidermis exposes a red, shiny, moist dermal surface, particularly noticeable over bony prominences.

(2) **24 hours**"fluid-filled bullae are formed between dermis and epidermis [Skin blebs].

(3) **48 hours**"(i) Sweetish, disagreeable odor (ii) internal fetal organs and connective tissue show increasing purple discoloration due to hemolysis and breakdown of red cells. (iii) Dark, red-stained fluid accumulates in the serous cavities. This should be distinguished from serous effusions acquired antemortem. (iv) Proteolytic digestion by **kallikrein** contributes to - vascular permeability. (v) Similar color changes are observed in the amniotic fluid, which has a deep dark red appearance ("**tobacco juice**"), or in the event of passage of meconium, a thick brown appearance. (vi) The volume of the amniotic fluid .s following fetal death, and the level of **α-fetoprotein** in it may be -ed because of concentration effect and -ed fetal skin permeability. (vii) **Autolysis of connective tissue** - contributes to laxity of joints and lack of definition of cut surface margins of the solid organs. Body is soft, flaccid and flattens out when placed on a level surface. (viii) **Bones** are flexible and readily detached from soft parts. (ix) **Joints** become abnormally mobile. (x) **Abdominal organs** may show green discoloration due to leakage of bile pigments from the gallbladder.

(4) **3 to 4 days**" (i) Fetal epidermis loses integrity. (ii) **Umbilical cord** is red, soft and smooth

(5) **5th day**"S/c edema up to 5 mm.

(6) **>1 week**" (i) Meconium released into the abdominal cavity through the dissolving bowel wall. (ii) Protruding autolyzed liver mass produces an abnormal ultrasonographic appearance. May be mistaken for omphalocele. (iii) **Dystrophic calcification** may develop in the autolyzed liver tissue. (iv) **fluid accumulation** may mimic hydrops fetalis. (v) fluid accumulation at the nape of the neck may be mistaken for a cystic hygroma or occipital

encephalocele. (vi) distortion of skull occurs during vaginal delivery. May lead to an erroneous diagnosis of hydrocephalus. (vii) Squeezing of the autolyzed brain tissue into the spinal cord and along the spinal nerve roots into the retropleural and retroperitoneal areas may produce tumorous masses of neural tissue. May be mistaken for a neurogenic neoplasm. (viii) further changes in color occur, from purple to brown discoloration.

(7) **Several weeks** - (i) a fading of color to yellow–gray occurs. (ii) Progressive loss of fluid from the fetus eventually results in the formation of "fetus papyraceous." (iii) Dehydration results in shrinkage and compaction of tissues and organs. (iv) Very occasionally an extrauterine pregnancy may be retained for years forming so-called **lithopedion** (Greek lithos, stone, paidion, small child), a calcified fetus retained in the abdominal cavity.

Fig. 27.1: Maceration in a 5 month old fetus. The mother sustained road side accident (RSA). Ultrasonography found intrauterine death just after RSA. Spontaneous expulsion was awaited and it occurred after 3 days. All findings of maceration are present.

B. Rigor Mortis, Putrefaction, Adipocere and Mummification

Rigor mortis is a usual change after death. Putrefaction occurs if air enters the liquor amnii after death. Adipocere would occur if sufficient liquor amnii is there. Mummification would occur if all liquor amnii has drained out.

C. Radiological Signs of IU Deaths

The radiological sign of fetal death help to diagnose IU deaths, when the baby is still ***in utero***. They are in essence a study of the processes of maceration.

1. Robert's sign (1944)

Earliest sign to appear. **12 hours after death**. Air in aorta, pulmonary vessels or abdomen.

2. Spalding's sign (1922)

Appears **2 days after death**. Overlapping of fetal skull bones seen on x-ray (or ultrasound) examination. Due to shrinkage of cerebrum after intrauterine death. [D/d. May also be positive (i) during or just prior to labor (ii) scanty liquor amnii and diminished fetal vitality (iii) microcephaly (iv) craniostenosis.]

3. Deuel's halo sign (1947)

3 days after death. Separation of the subcutaneous fat of the fetal scalp from the cranial bones. Seen as a halo on radiography and USG. First described by **Deuel** in **1947**.

IV. VIABILITY OF THE FETUS

Viability is the ability of a fetus to survive outside the uterus.

Salient features:

(1) **Indian Law** - Age of viability has not been defined in **Indian law**.

(2) **English law** - Age of viability is **28 weeks [s1(2)** Infant Life (Preservation) Act, 1929 of UK**]**. Since India does not have explicit law on viability, by derivation, **28 weeks** may be taken as age of **viability in India** too. Some books wrongly mention it as **210 days**. If that fact is accepted, the definition of stillbirth would have to be changed, which asserts that a stillborn child is one which is born after 28th week of pregnancy [age of viability]. According to the wrong "**210 day viability standard**", babies born between **196 days**

and **210 days** would not be viable, yet would be called **stillborn**. A classical criteria of stillborn child is that it is “capable of being born alive.”

(3) **Physiology** - at age of viability pulmonary surfactant appears in the alveoli of lungs ".es surface tension of alveoli"alveoli become able to inflate.

(4) **Pathology** - (i) When the pulmonary surfactant is deficient, **Infant respiratory distress syndrome** [IRDS, neonatal respiratory distress syndrome, earlier called hyaline membrane disease (HMD)] may result. Pulmonary surfactant is rich in lecithin [syn, disaturated phosphatidylcholine, dipalmitoyl phosphatidylcholine]. For composition, please see **ch 19**. (ii) Lecithin synthesis - by type II alveolar cells -es dramatically as the lung matures. Lecithin levels are expressed as a ratio against sphingomyelin, a non-pulmonary lipid whose concentration is relatively constant in amniotic fluid

Memory Aid 1: Lecithin levels and lung maturity

L - **l**ung, **l**ecithin

L/S > 2 is rarely associated with IRDS. L/S < 2 indicates -ed risk of IRDS. (iii) pathogenesis of hyaline membrane - .ed surfactant ".ed lung compliance"hypoventilation"hypoxemia + CO_2 retention" acidosis"pulmonary vasoconstriction"pulmonary hypoperfusion"epithelial and endothelial damage" plasma leak into alveoli"fibrin + necrotic cells [hyaline membrane].

V. LIVE BIRTH

[A] **L*egal definition*** of live birth in **Indian criminal law** - A child is “***live born***”, if any part of that child has been brought forth, even though the child may not have breathed or been completely born [**Explanation 3** of **S.299, IPC**]. [B] **L*egal definition*** of live birth in **Indian civil law -** “live-birth” means the ***complete expulsion*** of fetus from its mother, irrespective of the duration of pregnancy, and who after such expulsion breathes or shows any other evidence of life **[S.2(d) of *The Registration of Births And Deaths Act, 1969*]**. Possible evidences of life are (1) Cry. (2) Movement of body or limbs. Muscle twitching is not safe to take as evidence of life as muscles may twitch for sometime after death.

A. Signs of Live Birth

1. Shape of the chest

(1) **Before respiration** – (i) Chest is flat. (ii) Circumference 1-2 cm less than that of abdomen at the level of umbilicus

(2) **After respiration** – (i) Chest expands. (ii) Drum shaped.

2. Position of diaphragm

(1) **Before respiration** – at the level of 4th or 5th rib.

(2) **After respiration** – at the level of 6th or 7th rib.

Memory Aid 2: Position of diaphragm before and after respiration

Remember 4, 5, 6, 7

(3) **Precautions** - (i) Abdomen must be opened before thorax - Highest point of diaphragm must be noted. If thorax is opened first, air can enter thorax and push diaphragm to a lower level giving a false indication of live birth. Converse cannot happen, i.e. diaphragm going up on opening abdomen, because lungs would prevent it. (ii) In putrefaction - gases may spuriously alter position of diaphragm.

3. Lungs

Breathing causes significant and permanent changes in the lungs [**Table 3**]. Their extent depends upon the period and strength of respiration.

a. Volume

Please see **Table 3**.

b. Margins

Please see **Table 3**.

c. Consistency

Crepitant after respiration, but also crepitant after putrefaction and after artificial respiration. Please also see **Table 3**.

d. Color and expansion of air vesicles

(1) **Before respiration** – On section, interior of lung is uniform in color and texture.

(2) **After respiration** -

(i) Alveoli are distended with air, initially at anterior surfaces and margins, and then in the remaining portions.

(ii) With expansion, the air vesicles are slightly raised above the surface "seen as

angular or polygonal areas on the surface of lungs.

(iii) As blood becomes oxygenated in the expanded areas, the entire surface has a mosaic, mottled or marbled appearance. Aerated and expanded areas appear rose-colored and these areas alternate with the collapsed unaerated dark bluish areas.

(3) **Precautions** -

(i) **Exposure to air of unexpanded lungs** - may brighten color, but air cells would not be distended

(ii) **Artificially inflated lungs** - Mottling may be absent. On cut section, the exposed surface with exude little blood, but no froth. Please also see **Table 3**.

e. Gas

(1) **Respired lungs** – If lungs are dipped in water, part of air within the bronchi and alveoli will be expelled and will rise to the surface as a stream of minute dots.

(2) **Gas due to putrefaction** – Bubbles of gas, instead of being within the alveoli, are under the pleura and within the substance of lungs [interstitial blebbing]. The bubbles underneath the pleura can be moved from place to place by stroking with a finger.

f. Blood in the lung beds

Blood within the lungs after respiration is twice than that present before respiration [**Table 3**].

g. Weight of lungs

i. Fodéré's test [syn, Static test]

(1) **Procedure** - Lungs are ligated across their hila, separated and weighed.

(2) **Wt of both lungs combined** - (i) before respiration - 35 g (ii) after respiration - 70 g

(3) The -in weight is due to -ed flow of blood [**Table 3**].

ii. Ploucquet's test

(1) Wt of lungs is calculated as a ratio of infant's total weight to even out differences due to wt of infants

(2) **Wt of both lungs combined** -

(i) before respiration – $1/70^{th}$ of body wt

(ii) after respiration – $1/35^{th}$ of body wt. Takes into account differences in lung weights on account of different weights of body. Named after **Wilhelm**

Gottfried Ploucquet [1774-1814] of Tübingen.

Memory Aid 3: Fodɛrê and Ploucquet's tests

Remember **35** and **70**. These numerals just get transposed in the two tests. It is because of the coincidental fact that the wt of newborn is roughly 2450 g [35x70]. Thus wt of both lungs before respiration [35g] turn out to be $1/70^{th}$ of BW and after respiration [70g] turn out to be $1/35^{th}$.

Table 3: Differences in lungs before and after respiration

S. No	Feature	Before respiration	After respiration
[A] Gross			
1.	Color	Uniformly reddish brown, bluish or deep violet depending on the degree of anoxia	Mosaic, mottled or marbled
2.	Volume	Small; do not cover heart. Collapsed on the hilum	Larger; cover heart
3.	Surface	Smooth	uneven
4.	Margins	Sharp	rounded
5.	Lowermost margin	At the level of 4-5 ribs	At the level of 6-7 ribs
6.	Consistency	Liver like, dense, firm, non-crepitant	crepitant, elastic, soft, spongy
7.	Blood within lungs	Less	More. Twice that present in unrespired lungs
8.	Weight of both lungs [absolute]	35 g	70 g
9.	Weight of both lungs [in relation to body weight]	1/70 of body wt	1/35 of body wt
10.	Specific gravity	1.04-1.05. Heavier than water	0.94. Lighter than water
11.	Hydrostatic test	-ve. Sinks in water	+ve. Floats in water
12.	Thoracic cavity	Not occupied fully by lungs	Almost occupied fully by lungs
13.	Pleura	Loose, wrinkled	Taut, stretched

[B] Cut section			
14.	Oozing of blood	Little frothless blood exudes on pressure	Abundant frothy blood oozes on cut section
15.	Bronchi and bronchioles	Empty	Contain blood stained froth
16.	Alveoli	Not inflated	inflated
[C] MLI			
17.	MLI	Indicates stillborn or deadborn child	Indicates live born child

h. Hydrostatic test (Raygat's test)

i. Principle

Upon breathing, both wt and vol of lungs are -ed. Wt is -ed due to inflow of blood and vol due to inflow of air. The - in vol is much more than - in wt due to which specific gravity of lungs is .ed.

Salient features:

(1) **Sp gr of lungs** – (i) Before respiration "1.04-1.05 (ii) After respiration "0.94

(2) **Procedure** - Hydrostatic test is performed in following four stages: (i) Stage 1 - Whole thoracic pluck consisting of both lungs and heart are placed in a bucket of water. If the pluck floats"indicates air in lungs"indicates that the infant may have born alive. If the pluck sinks, move to second stage. A piece of liver is also sunk in water as control. It must sink in normal conditions. If it floats, it may indicate putrefaction, making the test of dubious value. (ii) Stage 2 -Each brochus is tied, and lungs severed above the ligature. Each lung is then placed separately in water. If either lung floats, it indicates that the infant may have born alive. If either lung sinks, move to third stage. (iii) Stage 3 - Cut each lung in 12-20 pieces (a) Roll a piece of lung gently between a finger and thumb near the ear and see if there is a crackling crepitant noise. (b) Dip

each piece under water and squeeze between thumb and finger and see if any bubbles are escaping (c) observe if they float independently. For moving to the next stage same criteria applies as above (iv) Stage 4 - Each piece is now taken out of water, wrapped in a piece of cloth and squeezed by putting a weight. This is an important step as it is supposed to remove the expiratory reserve volume air, and tidal air. Residual air still remains within the alveoli, which can not be taken out by any means. This residual air is responsible for keeping the lungs afloat in infants who have been born alive and respired. If the lungs were floating in earlier stages due to artefacts (such as gases introdued by artificial respiration), the pieces of lung would not continue to float till the last stage.

ii. Fallacies

There are **two major fallacies** of this test.

(a) Child respired after birth yet lungs sink

Causes:

(1) **Absorption of air** – Circulation continued after stoppage of respiration for sometime"air from lungs is absorbed in circulation"No air remains in lungs.

(2) **Atelectasis** (non expansion) of lungs. Sequence of events is Feeble respiration"Air does not enter lungs, but remains up to tracheal and bronchial level"Oxygenation of blood occurs through tracheal and bronchial mucosa"Lungs remains unexpanded, but children was alive.

(3) A**lveolar duct membrane** - Causing obstruction to entry of air in alveoli

(4) **Diseases** - (i) Acute edema (ii) Congenital syphilis (iii) Pneumonia

(5) **Feeble respiration** – More air is expelled from the lungs during expiration, than what is inhaled during inspiration.

(b) Child did not respire after birth yet lungs float

Causes:

(1) **Artificial respiration** - given via a tube, catheter or cannula passed into the trachea or by mouth to mouth respiration. Lungs are inflated partially. In case of mouth to mouth respiration, air may be found in stomach too.

(2) **Putrefaction** - Putrefactive gases will make the lungs float. Distinctive features

(i) Body - shows signs of decomposition

(ii) Lungs - (a) soft and greenish (b) Bubbles of gas - (I) large bubbles seen on lung surface. (II) Unequal in size. (III) Project considerably from the

surface. (IV) Gas within them can be pushed readily from place to place. (V) Collapse on pricking.

(3) **Respiration within the womb** [**vagitus uterinus**]- Fetus may respire within the womb if membranes have ruptured, but may die from natural causes within the birth canal and may not breathe once completely born.

(4) **Respiration within the Vagina [vagitus vaginalis]** - Similar to above. Respiration occurs within vagina, but not when completely born.

iii. Hydrostatic test is not necessary in following conditions

(1) **One is sure fetus was born dead** - Fetus is

(i) Born before age of viability [28 wks]

(ii) Macerated or mummified

(iii) Monster [eg anencephalic]

(iv) Bruising on lungs - indicating efforts at artificial respiration.

(2) **One is sure fetus was born alive** -

(i) Stomach - contains milk

(ii) Umbilical cord - has separated and a scar has formed.

i. Radiography of lungs

May reveal air within them.

j. Microscopic examination of the lungs

(1) **Value** - is limited. Cannot provide clear evidence of extrauterine respiration, if child has lived only for a few minutes.

(2) **Procedure** -

(i) Removal - Thoracic contents are removed intact by cuts with a scalpel by a **"no touch" technique** first described by **Osborn** [Pathologist and Medical Superintendent, Derbyshire Royal Infirmary] in 1953. The technique aims to eliminate artifacts.

(ii) Fixation - for 48 h

(iii) Sections - taken from the whole lung

(3) **Appearances** -

(i) at 4 months pregnancy - Parenchyma of the lung shows gland like structure with cuboidal or columnar lining

(ii) at 5th month - air cells are filled with amniotic fluid

(iii) at full term - (a) thin walled adult type alveolus is formed. (b) Completely

atelectatic, but many of its terminal bronchioles and vesicles are partly expanded by amniotic fluid. (c) Fetus makes some respiratory movements near full term, which fill its alveoli with amniotic fluid. This material is not stained with H&E giving a spurious impression that air is present in lungs. As respiration takes place, further expansion of alveoli occurs. Fluid present in alveoli is partly absorbed back into the pulmonary circulation and partly expelled through the bronchi. (d) Tardieu's spots - are no indication of live birth. They may be seen in stillbirth, live birth and in bronchopneumonia.

k. Tests for fetal lung maturity

Presence of creatine, lecithin and fat cells.

4. Changes in stomach and intestines

a. Air in stomach and intestines

(1) **Breslau's second life test**; **stomach bowel test** **[Fig. 27.3]** - Stomach and intestines are removed after tying double ligatures at each end, and put in water. They float if respiration had taken place; otherwise they sink

(2) **Principle** - Air is swallowed into the stomach and intestines during respiration, making them buoyant

(3) **False +ve** –

(i) Resuscitation attempts

(ii) Bacterial gas formation [putrefaction]

(4) **Survival period** – can be calculated: (i) Immediate after birth – gas in stomach. Sometimes may take up to **15 min**. In such cases, stomach bowel test may be –ve even in live births, if death takes place before this period. (ii) 1-2 h – Gas reaches small intestines (iii) 5-6 h – colon (iv) 12 h - Rectum.

(5) **Drawbacks and Fallacies** –

(i) Useless in putrefaction

(ii) Air may be swallowed by the child in attempting to free the air passages of fluid obstructions in cases of stillbirth.

b. Other material

(1) Other material present in the stomach may be (i) Amniotic fluid (ii) Blood [maternal] (iii) meconium (iv) milk (v) Mucus and Mucins [glycoproteins in

mucus] (vi) Saliva.

(2) Presence of milk is the only surest indication that the child was born alive.

(3) All the other indicate swallowing movements within the uterus or during passage through the birth canal. No reliance may be made on them.

5. Changes in the middle ear

(1) **Wreden's test** –

(i) Regular procedure – Skull cap is removed and base of skull submerged in water. Petrous part of temporal bone [which forms roof of the middle ear] is opened. If a bubble of air escapes from middle ear, the child was born alive.

(ii) Simpler method – Dip ears in water. Puncture tympanic membrane"Bubble of air escapes" Live birth. Both procedures reveal presence of air in middle ear.

(2) **Principle** - During embryonic life, the middle ear contains gelatinous tissue. During efforts of breathing, the sphincter at the pharyngeal end of Eustachian tube relaxes and some air enters the middle ear. Named after **Roberto Wreden** of St. Petersburgh. Sometimes wrongly spelt as "Wredin's test". Also recommended by **H. Schmaltz** in **1877**.

(3) **Reliability** – Very low.

6. Other signs of live birth

Many of these also help in determination of time of survival.

a. Blood

(1) **Nucleated RBC**"disappear in 24 h

(2) **Fetal Hb** [synthesized mainly in liver]"(i) Before birth"80-90% (ii) 3rd month"7-8% (iii) 6 m"disappears completely.

b. Meconium

(1) **Characteristics:** (i) It is a green viscid substance consisting of thickened bile and mucus (ii) Stains are brownish green (iii) Stiffen the cloth (iv) Reaction is acidic

(2) **Expulsion** -

(i) Completely excreted from the large intestine in 24-48 h after normal birth

(ii) May be completely excreted before birth in (a) breech presentation (b) severe

anoxia.

c. Caput succedaneum

Caput succedaneum [CS] is an area of soft swelling that forms in the scalp over the presenting part of the head in vertex presentations [**Fig 27.2**].

Salient features:

(1) **Causes** - (i) Juxtaposition of scalp to the opening of the dilated cervix during labor, especially during prolonged or difficult delivery. (ii) Vacuum extraction.

(2) **Mechanism** – rigid cervical ring [or ring of extractor] presses on presenting part [scalp] ".ed venous return"Localized area of congestion and edema

(3) **Swelling** – (i) Site (a) vertex presentations - Most commonly occurs asymmetrically over the parietal region (b) Breech – over buttocks, scrotum, labia (ii) crosses suture lines and is soft (iii) Thickness is 3-4 times the normal thickness of scalp

(4) **Fate** – starts diminishing soon after birth. Completely disappears in a week.

(5) **D/D** – Cephalhematoma [**Fig 27.2; Table 4**].

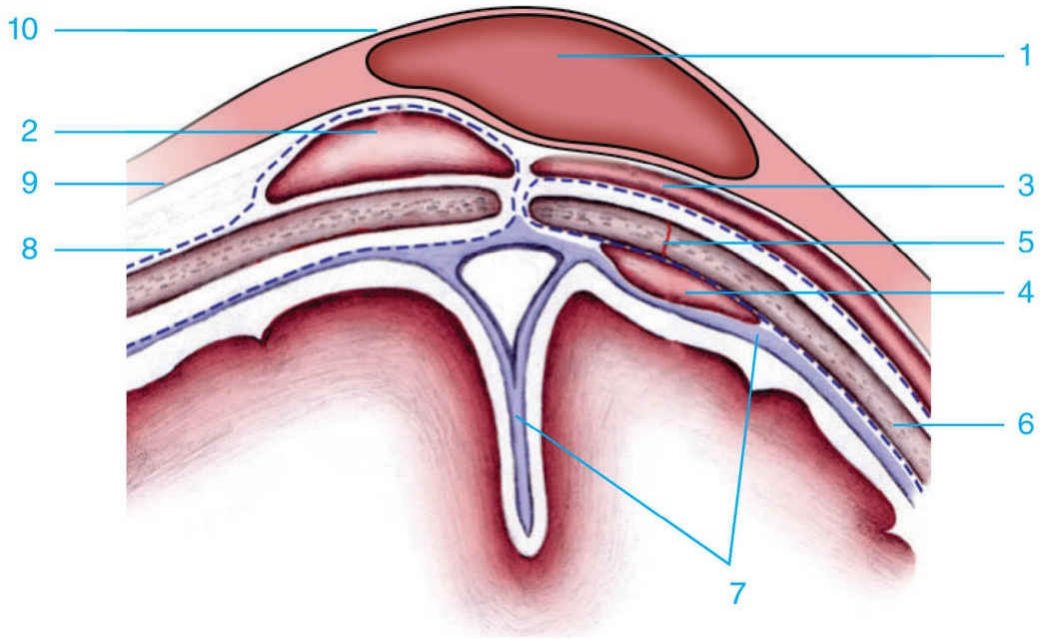


Fig. 27.2: (1) **Caput succedaneum** (CS) and (2) **Cephalhematoma** (CH) (3) Subgaleal hemorrhage and (4) Extradural hemorr-hage are also shown for better comprehension of relationship between various cranial hemorrhages. (5) Fracture of the skull (6) Skull (7) Dura (8) Periosteum (9) Galea or epicranial aponeurosis (10) Skin. CH does not extend across suture lines, because it is limited by periosteum over it; CS has no such limitation.

Table 4: Differences between Caput succedaneum and cephalhematoma

S. No	Feature	Caput succedaneum	Cephalhematoma
1.	Cause	Edematous swelling between the skin and galea due to pressure of presenting part, and impaired venous return	localized accumulation of blood between periosteum and skull due to *rupture of a periosteal capillary* [pressure of birth, instrumental delivery, vacuum delivery]
2.	Contents	Sero-sanguinous fluid	Blood
3.	Occurrence	Common	Rare [<1%]
4.	Suture lines	Extends across them	Never extends across them
5.	Size	Larger	Smaller [1-5 cm]
6.	Situation	May be bilateral	Unilateral; present over parietal bone
7.	Color	No discoloration	Discoloration of the swollen site due to presence of coagulated blood. Looks like a bruise
8.	Impulse on crying	No impulse [because no connection with CSF]	No impulse
9.	Development	Immediately after birth	Not present at birth. Develops after 24-48 h.
10.	Disappearance	Starts .ing from birth. Full disappearance in 1 wk	Often -es during first 1-2 days as more blood oozes. Then starts .ing. Full disappearance in 6-8 wks
11.	Complications	Usually none	(1) Jaundice, because of extra load of blood pigment (2) calcification or ossification may occur with a subsequent bony swelling that may persist for months"Significant deformities of the skull. May have to be surgically excised.
12.	MLI	Generally taken to be a sign of live birth. **Exc** - (i) Has been found even before birth in oligohydramnios (ii) In prolonged labor, fetus may die before birth	Regression helps in timing the survival period

d. Skin

(1) **Initially** -

(i) Skin is bright red.

(ii) Vernix caseosa covers skin mostly in axilla, inguinal region, folds of neck, buttocks.

(iii) Rarely absent. It may also have been washed away

(2) **2 days** – darker, brick red.

(3) **3rd day** –

(i) Yellow due to physiological jaundice [due to relative insufficiency of enzymes required for conjugation and excretion of bilirubin].

(ii) Skin of abdomen exfoliates during first 3 days after birth

(4) **7 days** – color is normal.

e. Umbilical cord

i. Changes in umbilical cord proper

(1) **2h** – Blood clots in the cut end

(2) **12-24h** [Mnemonic 1 d]– Cord attached to the child **shrinks and dries** [no

relation with live birth; seen in deadborn and stillborn also].

(3) **36-48h** [Mnemonic 2 d]– An **inflammatory ring** forms at the base of stump.

(4) **2-3 d** [Mnemonic 3 d]– Cord **m ummifies** [No relation to live birth; seen in deadborn and stillborn also].

(5) **5-6 d** – Cord **falls** off, leaving an ulcer

(6) **10-12 d** – Ulcer heals, leaving a scar.

Memory Aid 4: Changes in umbilical cord

1, 2, 3, ~~4~~, 5-6"**SIM Falls**

(1) 1d"**S**hrinks

(2) 2d"**I**nflammatory ring

(3) 3d"**M**ummifies

~~4~~ d"Nothing happens

(4) 5-6d"**Falls**

(5) **Healing** occurs in twice as much time"10-12 d [twice of 5-6d]

ii. Vascular changes

(1) **10h–3d** – UA closes [**Table 5**]

(2) **3-5 d** – Left UV closes [**Table 5**].

iii. Signs of human interference

(1) Whether ligature has been applied or not [generally indicates live birth].

(2) Whether cut by scissors [sharp edges] or torn [ragged edges]. Latter indicates self delivery or unskilled delivery.

(3) Note length of remaining cord.

f. Placenta

If placenta found with the body, it must be examined. Placental dimensions at full term [**ch 26**]. Umbilical cord dimensions at full term [**ch 3**].

g. Circulation

Various circulatory structures close at fixed intervals [**Table 5**].

Table 5: Circulation changes in a newborn

S.No	Structure	Closure period
1.	Umbilical arteries	10 hours to 3 days
2.	Left Umbilical vein	3-5 days
3.	Ductus venosus	3-5 days
4.	Ductus arteriosus	7-10 days
5.	Foramen ovale	2-3 months

Memory Aid 5: Order of closure of fetal structures

A very **ve**ry **art**istic **for**um

Artery (umbilical)"Vein (left umbilical)"Venosus"Arteriosus"Foramen ovale

Memory Aid 6: Umbilical vein

Q. Which vein is left?

A. Left is left [Right begins to obliterate at **4th wk** and disappears by **7th wk**]

Memory Aid 7: Umbilical artery

Umb **il i** cal artery is branch of **il**iac **i** nternal

Sometimes fossa ovalis can take longer to close, and sometimes remains open throughout life [morbus ceruleus]. Patent foramen ovale is detectable in about **30%** of general population.

h. Miscellaneous

Subdural hematoma – Although rare, but if present, indicates live birth. Due to molding [**ch 17**].

VI. CAUSES OF DEATH

i. Precipitate Labor

Precipitate labor is the expulsion of fetus within less than 3 h of commencement of contractions.

Salient features:

(1) All three stages of labor are merged in one

(2) Delivery occurs suddenly and rapidly even without the knowledge of mother

(3) **Delivery may occur even unconsciously** during (i) Anesthesia (ii) apoplexy (iii) asphyxia (iv) coma (v) Deep drunkenness (vi) delirium (vii) eclamptic convulsions (viii) fits (ix) Hypnosis (x) hysteria (xi) Influence of narcotic or intoxicating drugs (xii) sleep (xiii) syncope. Such conditions are thought to bring about deep lethargy and complete loss of sensation, preventing the mother to sense the ongoing parturition.

(4) **Risk factors** are

(i) Multipara [extremely rare in primi]

(ii) Placental abruption

(iii) Roomy pelvis

(iv) Small premature baby. In babies weighing >2500 g, incidence is 3%.

(v) Mother not being able to distinguish the sense of fullness produced by the descent of child from the feeling of bulky evacuation.

(5) **Associated with** - an -ed risk for maternal complications

(i) cervical and grade 3 perineal tears

(ii) need for packed-cells transfusions

(iii) post-partum hemorrhage

(iv) retained placenta

(6) **Child may die from** -

(i) Suffocation - if falls into lavatory pan containing feces

(ii) Drowning - if falls into a pan containing water [**ch 19**"Death of newborn infants]

(iii) Head injury - with fracture of skull and intracranial hemorrhages [bilateral SDH]. (a) Bones involved – (I) one or both parietal bones; (II) fracture may radiate into the frontal and squamous portion of temporal bone (b) Especially likely to occur (I) if mother was standing (II) There was hard floor beneath. The cord is torn more commonly at the fetal end than at the maternal end, but is rarely torn at the middle.

(iv) Hemorrhage - from torn end of the cord.

(7) **PM findings** -

(i) Placenta – is attached to newborn

(ii) Umbilical cord – lacerated, torn [details as above].

(iii) Hair and injured scalp – show grass, gravel, mud, sand.

(iv) Head - (a) Caput succedaneum and moulding of head - absent (b) fracture of skull. (c) IC hemorrhages [please see above].

(v) Air passages and lungs – contain blood, meconium, vaginal mucus, mud, gravel, sand, grass, urine, feces etc depending on where the child fell or drowned.

(8) **MLI** -

(i) **Infanticide** - Plea of precipitate labor is sometimes raised in cases of infanticide.

(ii) **False allegation** - Alternatively in a true precipitate labor case, mother may be accused of infanticide. Differentiation in **Table 6**.

Table 6: Differences between head injury due to precipitate labor and blunt force

S.No	Feature	Head injury due to precipitate labor	Head injury due to blunt force [as in infanticide]
1.	Manner	Accidental	homicidal
2.	Mechanism	Sudden dropping of newborn infant from pelvis to the floor due to sudden precipitate labor	Intentional hitting over head to kill
3.	Bruises	May be present on vertex – the area directly coming in contact with floor	Found anywhere on scalp
4.	Lacerations on scalp	absent	Present
5.	Fractures	Fissured fractures of parietal bones usually. Run upwards at right angles to meet sagittal suture	Extensive comminuted and depressed fractures of vault or base
6.	Brain	Usually not injured	Contusions, hemorrhages, lacerations

Criminal

a. Acts of commission

Acts of commission are acts done to cause death, eg suffocating the child.

Salient features:

Since the child was unwanted, there may have been attempts at criminal abortion, attempts at self delivery [to cause immediate destruction] etc. These may cause multiple circumferential abrasions around the whole surface of the neck [due to fingernails].

i. Asphyxia

(a) Drowning

(1) Rare form of child murder

(2) Child may be thrown in tank, well or other reservoir of water [**antemortem drowning**], or first killed and then thrown similarly [**postmortem submersion**].

(3) **Woman in parturition** - may sit in a large tub of water and directly deliver the baby in water"child delivered underneath water"dies of drowning before any respiration takes place"Signs of asphyxia not evident

(4) **Drowning in milk** - At one time, in Varanasi and other neighboring places, submersion of child's face into a cauldron of warm milk used to be a common mode of infanticide.

(5) **Precipitate delivery**"there may be mixture of urine, blood, meconium and water in the infant's air passages [please see above].

(b) Strangulation

(1) **Manual strangulation** [throttling] - nail marks may be found on child, but they may also be due to frantic efforts of mother to deliver herself.

(2) **Ligature strangulation** - Ligature may be found in situ. Sometimes umbilical cord may be used as a ligature material to mimic natural accident.

(c) Suffocation

(1) **Methods:**

(i) Smothering - (a) Closure of child's nose with fingers and pushing up lower jaw to occlude mouth. (b) Placing pillow or towel over infant's face (c) Pushing infant's face down into bed clothing or against breast

(ii) Gagging - Forcing cotton-wool, mud, rag etc into the mouth.

(2) **Autopsy** - Force needed is so small, that no evidence of struggle or trauma etc may be present. Only associated signs eg presence of cotton wool etc may give away method of murder. If these have been removed, there may be no clue left.

ii. Burning

(1) Rare form of child murder

(2) Application of dry heat, or more commonly immersing child in a cauldron of boiling water. Usual plea is that child accidentally spilled hot water over him, which was placed near him for bath.

iii. Fracture and dislocation of cervical vertebrae

Caused by twisting the neck.

iv. Poisons

Rarely poisoned milk may be given or poison may be injected or instilled in the eye.

v. Trauma

(a) Head injury

(1) **Inflicted by:**

(i) Dashing the head against a wall or floor by holding feet [rare] - Bruising of ankles and feet, where feet were gripped may be seen.

(ii) Blows on head with blunt weapon

(iii) Head pressed forcibly - under the leg of a bedstead [charpoy]

(2) **Injuries produced:**

(i) Contusions, lacerations - of scalp, brain.

(ii) Fractures of skull– comminuted, depressed. D/d – injuries produced by forceps delivery [as above].

(iii) EDH in neonates - [**ch 17**] (iv) SDH, SAH.

(b) Injury to other organs

Abdominal injury, crushing of testes, thrusting of needle within brain through fontanelles. Cut throat, incised wounds, stabs etc may be seen

(c) Throwing from height

Uncommon, but well-known method.

b. Acts of omission

Acts of omission are acts intended to overlook basic needs of the child, eg not giving food to child.

Salient features:

(1) **Examples of acts of omission** – Failure to

(i) Clear the air passages – these may be obstructed by amniotic fluid or mucus

(ii) Protect child – from heat or cold

(iii) Provide proper assistance – during labor. May cause death by suffocation or head injury

(iv) Supply child with proper food

(v) Tie the cord – after it is cut. May cause death by hemorrhage

(2) Person in-charge of the infant [mother, father, guardian etc] can be charged

u/s 302, IPC if they fail to take care of child and allow him to die.

i. Abandoning of infants

(1) An abandoned child is called a **foundling**

(2) If father, mother or guardian of a child <12 y exposes or abandons him"7y imprisonment or fine or both [**S.317, IPC**]

(3) If death of such exposed child occurs, parent or guardian would be punished for murder or culpable homicide, as the case may be.

ii. Concealment of birth by secret disposal of dead body

Whoever intentionally conceals or endeavors to conceal birth of a child by secretly burying or otherwise disposing of the dead body of a child whether such child died before, after or during its birth, is punished with 2 y imprisonment or fine or both [**S.318, IPC**].

VII. CHILD ABUSE

Child abuse is the physical, sexual or emotional mistreatment or neglect of a child.

Salient features:

(1) Child abuse can occur in a child's home, or in the organizations, schools or communities the child interacts with.

(2) **Five categories** of child abuse: (A) Child neglect (B) Emotional and psychological abuse (C) child sexual abuse and (D) physical abuse (E) Intentional poisoning [chemical abuse].

A. Physical Abuse

Physical aggression directed at a child by an adult. Further divided into those caused by mechanical trauma, and other forms of abuse [asphyxiation, burns and scalds, drowning and subtle abuse].

1. Mechanical trauma

a. Battered baby syndrome

Battered baby syndrome [syn. ***BBS***, ***Battered child syndrome***, ***Caffey's syndrome, Caffey-Kempe syndrome,*** Maltreatment syndrome**,** ***Non Accidental Injury of Childhood*** (NAIC), parent-infant traumatic stress syndrome (PITS), Tardieu's syndrome] is a clinical condition in which young children usually under 3 years of age are beaten repeatedly over the most trivial provocation. The children are often unwanted (female, mentally deficient, disabled, stepchildren).

Salient features:

(1) **Lack of provocation** - Beating is completely excusable. Provocation is always trivial

(2) **Precipitating factors** – Often child's irritating actions eg crying, refusal to be quiet, persistent soiling of napkins etc

(3) **Deprivation of nutrition**, care and affection is present

(4) **Delay** - There is often delay between onset of injuries and medical attention sought. It is sought, only when the child becomes critical and parents lose nerve. The explanation is lack of money.

(5) **Cerebral palsy** - 10-15% cases of cerebral palsy may be the result of the battered baby syndrome.

i. Epidemiological features

(1) **Age** – usually <3 years. But can occur at any age

(2) **Sex** – slightly more in males

(3) **Status of child in family** – unwanted, disabled, stepchild, failure of contraception, pregnancy before marriage

(4) **Socioeconomic factors of parents** – (i) Age - Young [20-30 y] (ii) Family – isolated (iii) Guilt amnesia (iv) Have criminal records (v) .educated, .IQ, .socioeconomic class (vi) Psychiatric problems (vii) Socially unstable (viii) Unemployed (ix) Parents themselves brought up in (a) disharmonious families (b) families having long standing emotional or financial problems (c) chaotic, violent family (d) They were themselves battered children (e) Wife often battered by husband, with battering continuing during pregnancy (f) unhappy childhood experiences.

ii. History

(1) Incompatible with injuries. Repeated trauma over time is the hallmark of BBS. This causes abrasions, bruises, burns, fractures and other injuries of

different ages. The classical explanation offered by parent is that the child fell down the stairs. If that were the case, the age of all injuries would be the same

(2) Parents change history as they go to different doctors; even with the same doctors, their history may have serious contradictions.

iii. Injuries

Injuries are due to direct manual violence [commonest].

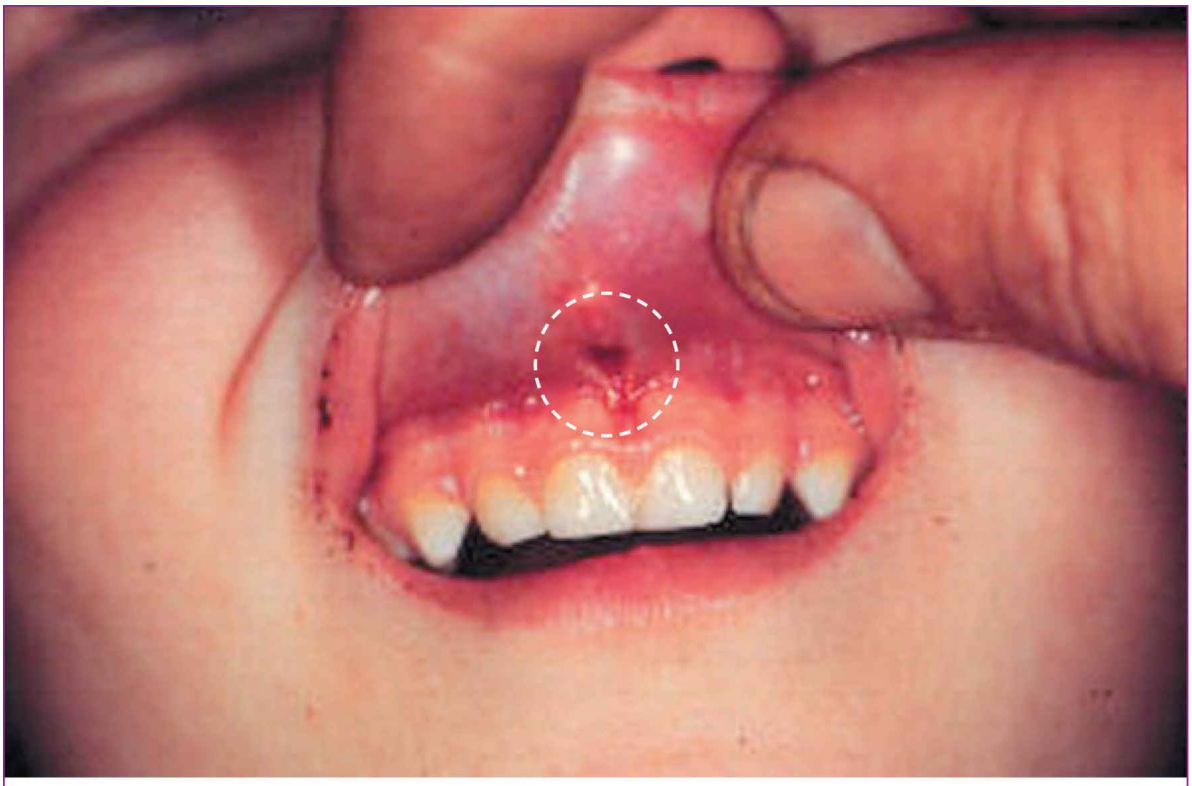


Fig. 27.3: Tear of frenulum in a case of battered baby syndrome.

(a) External injuries

(1) **Tear of the frenulum** – the most typical injury. Laceration of mucosa of upper lip is also seen. Laceration may be so large as to cause separation of inner surface of lips from base of gums. Typically caused by (a) efforts to silence screaming (b) blow on the face (c) may also be produced by trying to suffocate child [please see below].

(2) **Soft tissue injuries** –

(i) Types, site and shape – Almost anywhere on the body (a) Abrasions – on

back. Caused by dragging (b) Bite marks [**ch 3**] – on cheek, neck, back (c) Bruises (I) on either side of chest, behind the axillae, down the anterior chest wall and around the large joints eg elbow and joint"Indicate places where child was gripped strongly **[Six penny bruises - ch 12**] (II) on buttocks and thighs – caused by belts, canes, common household objects eg wooden ladles, hair brushes, pieces of wood, straps etc (d) Graze abrasions - on back"Indicate dragging (e) Knuckle punches – on the back. Show as rows of 3-4 round bruises [represent metacarpals of the offending fist] (f) Lacerations – beating by hard blunt objects, eg canes and sticks (g) Pinch marks – on face, neck, back, buttocks, thighs. Appear as butterfly shaped bruises with one wing caused by thumb larger than the other caused by opposing finger (h) slap marks – show as clear bruises [or as lines of petechial hgs] resembling fingers [of the slap] on cheeks, neck and back. (i) Scalp injuries – very characteristic. Caused by vigorous pulling on scalp hair (I) Subgaleal hematoma (II) Traumatic alopecia – bald patches on scalp

(ii) Cause - rough handling, beating, kicking or throwing the infant around.

(iii) Age - Typically the injuries are of different ages, as they are inflicted at different points in time.

(3) **Eyes -** (i) Black eye (ii) Hemorrhages [Retinal, Subconjunctival, Subhyaloid, Vitreous] (iii) Lens displacement (iv) Retinal separation.

Fig. 27.4: Six penny bruises on the body of a child in a case of battered baby syndrome.

(b) Internal injuries

Extensive internal injuries may be present with minimal or no external signs of injury.

1st. Skeletal injuries

(1) Lesions due to direct beating -

(i) Skull fractures, especially in occipito-parietal area. They are depressed,

multiple and wide

(ii) Transverse and spiral fractures of long bones, especially due to twisting of arms and legs etc.

(2) Traction lesions - These injuries result from jerking of shaking a child's limbs. May also result if, say, a toy is jerked away from a child's hands. Injuries of the periosteum of long bones are seen without fractures.

(i) Periosteal hematomas (a) If fresh – periosteum bulging with blood seen (b) If old – Seen radiologically as an extra line of opacity running alongside the affected length of bone. Due to calcification of bleeding which occurred under the periosteum.

(ii) Periosteal shearing – because periosteum is easily stripped off in infants

(iii) Epiphyseal separation – seen radiologically as small fragments separated from the main bone

(iv) Avulsion of metaphysis or chipping of the edges of metaphysis. These are known as **corner fractures** and **bucket handle fractures**. Bucket handle fracture is essentially the same as corner fracture, but this term is used where the loose bone is rather wide at the distal end, making it end in a crescent shape **[Fig. 27.5]**.

(3) Anteroposterior compression of chest - Fractures of ribs in midaxillary line

(4) Side-to-side compression of chest -

(i) Fractures at the costochondral junctions

(ii) Fractures along posterior angles of ribs. After 1-2 wks, callus forms. On X-rays these are seen as a "**string of beads**" [syn, **rosary bead appearance**] in the paravertebral gutter [**Nobbing fractures**] **[Fig. 27.6]**.

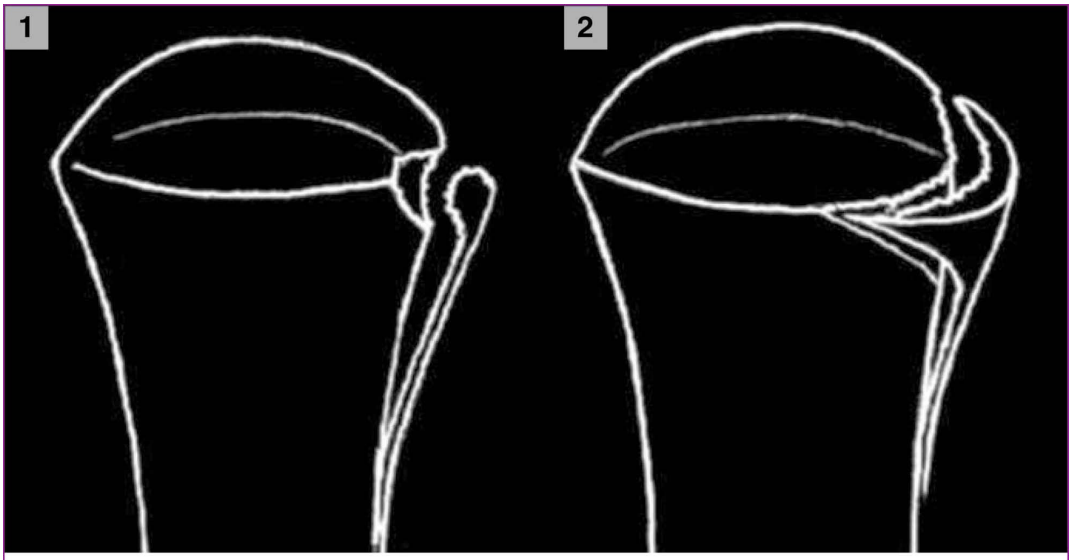


Fig. 27.5: **(1)** Corner fracture and **(2)** Bucket handle fracture.

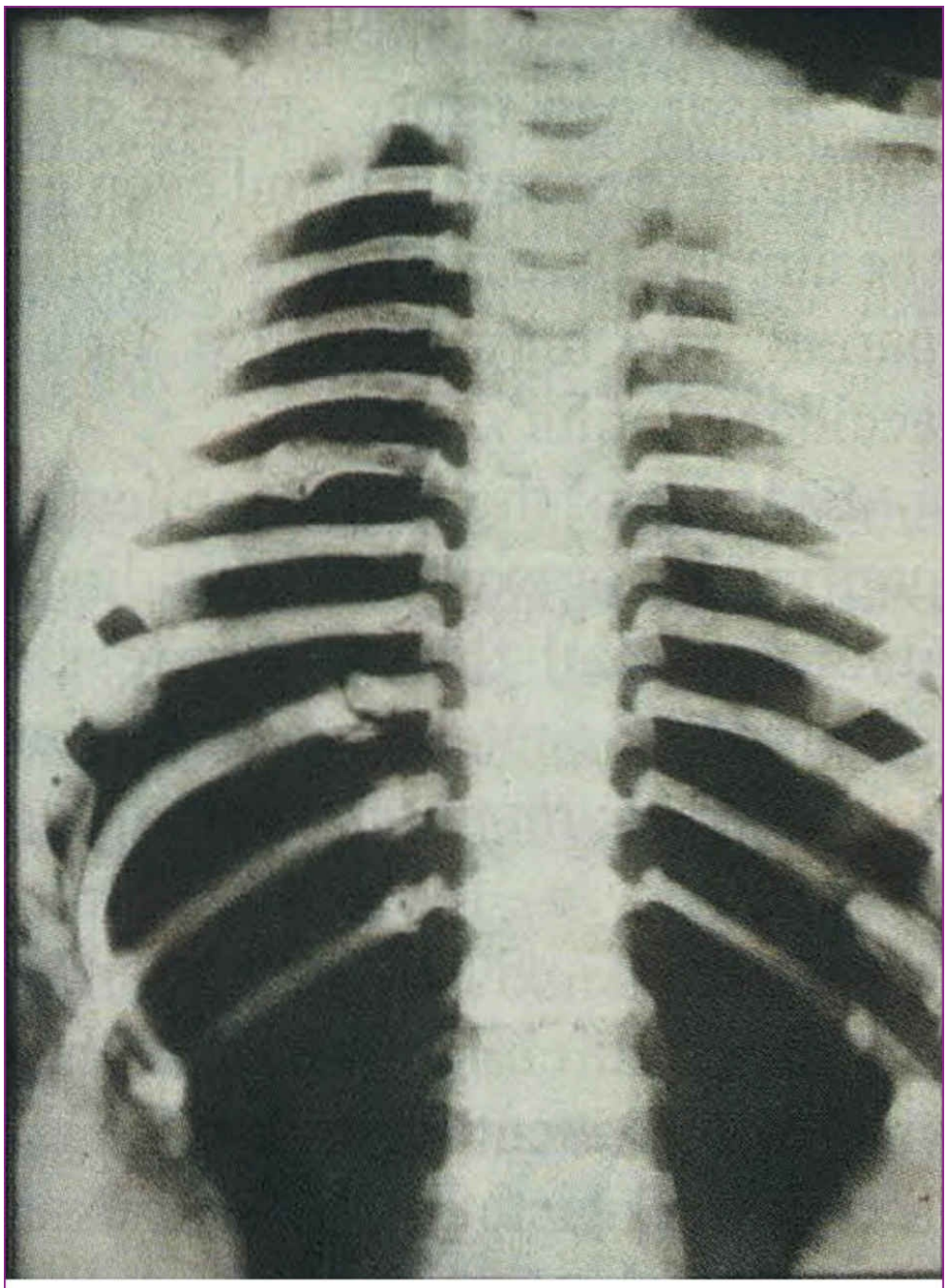


Fig. 27.6: Multiple fractures along posterior angle of ribs. As they would heal, the multiple calluses would give a "string of beads" or "rosary bead" appearance.

2nd. Visceral injuries

(1) Brain and meninges - SDH – Found in about 40% of fatal cases

(2) Lungs - Posttraumatic pulmonary pseudocysts [PTPPCs] – ch 17"Lung

injuries.

(3) Liver - Bursting injuries – Another name for lacerations. Crushing or compressing force on abdomen, as by fists

(4) Spleen - Bursting injuries – cause same as in liver

(5) Hollow viscera -

(i) Stomach, intestines and urinary bladder – may be ruptured, especially if compressing force [fists etc] was applied when the viscera were distended

(ii) Second part of duodenum and jejunum – may be completely transected

(iii) Tearing of mesentery – may cause complete disruption of the small intestine.

iv. Diagnosis

(a) In the living

Can be done by

(1) Time taken to seek medical advice

(2) nature of injuries

(3) recurrence of injuries

(4) Imaging studies (i) X-rays of the entire body [**babygram**] (ii) MRI (iii) Technetium 99m (^{99m}Tc) methylene diphosphonate (MDP) bone scintigraphy. (iv) Fluorine 18–labeled sodium fluoride (^{18}F-NaF) positron emission tomography [PET].

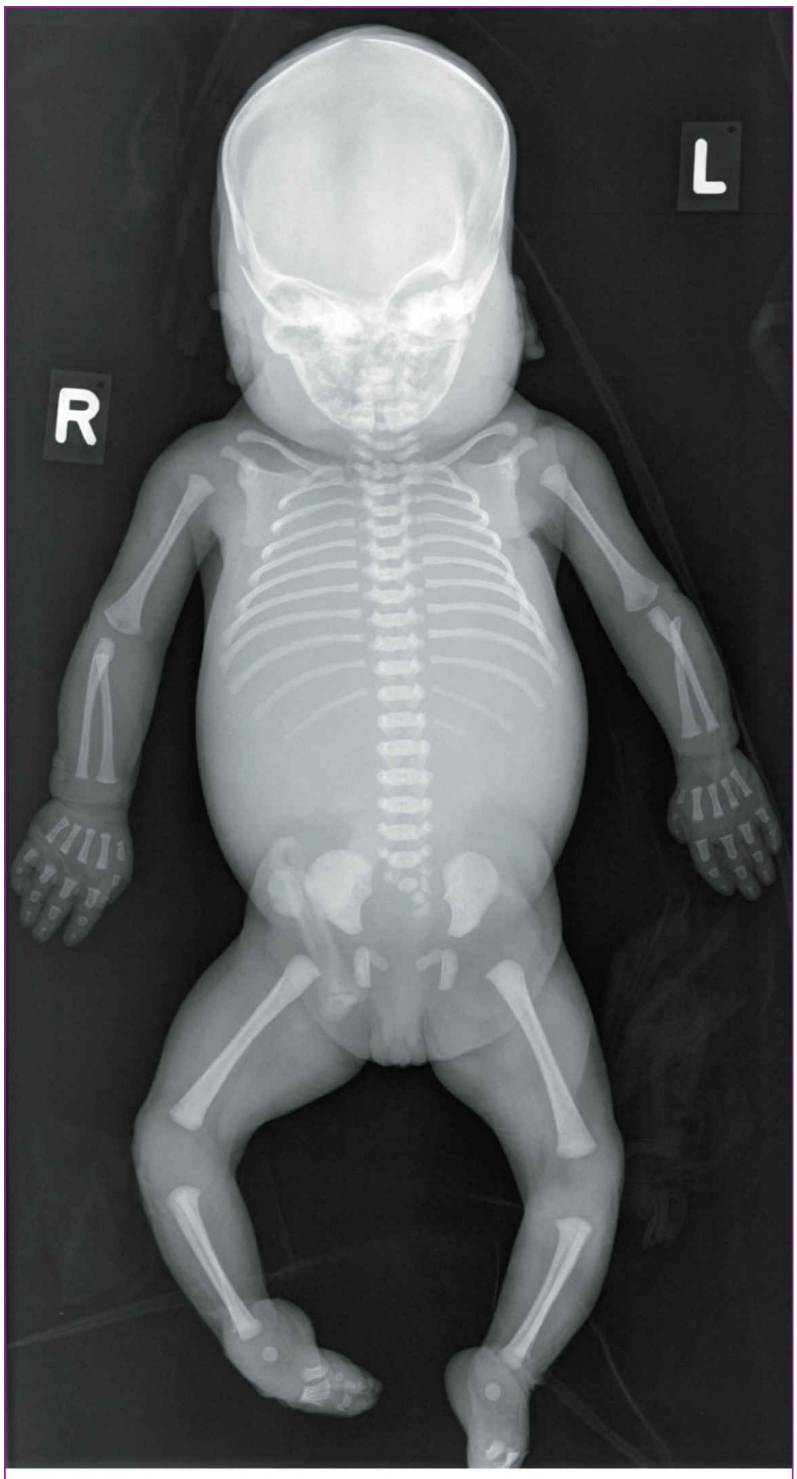


Fig. 27.7: A Babygram

(b) In the dead

(1) **Imaging studies** [as in the living] - before autopsy. Detects (i) old fractures

(ii) Epiphyseal and metaphyseal injuries in various stages of healing

(2) **Autopsy** – shows external and internal injuries as mentioned above

(3) **Cause of death** –

(i) Head injury [most common]

(ii) Rupture of abdominal organ [2nd most common].

v. D/d

D/b stands for "Differentiate by".

(1) **Acute abdomen** will occur in (a) Intrinsic gastrointestinal disease (eg. peritonitis, obstruction, inflammatory bowel disease, Meckel's diverticulum) [D/b Radiographs, stool tests] (b) Intrinsic urinary tract disease (infection, stone) [D/b Culture, ultrasound, intravenous pyelogram] (c) Genital problems (e.g., torsion of spermatic cord, ovarian cyst) [D/b History, physical examination, ultrasound, radiograph, laparoscopy] (d) sickle cell crisis [D/b Angiography, sickle cell studies]

(2) **Bruising** will occur in (a) Hemophilia [D/b Prothrombin time, partial prothromboplastin time] (b) Von Willebrand's disease [D/b Von Willebrand's panel] (c) Henoch Schonlein purpura [D/b Typical distribution of lesions] (d) Purpura fulminans [D/b Ruling out sepsis] (e) Ehlers-Danlos syndrome [D/b Hyperextensibility] (f) Mongolian spot - Not a true bruise [see ch 12 for details]

(3) **Fractures** will occur in (a) Osteogenesis imperfecta [D/b Radiology and blue sclerae] (b) Rickets [D/b Nutritional history] (c) Birth trauma [D/b Birth history] (d) Hypophosphatasia [D/b Decreased alkaline phosphatase] (e) Leukemia [D/b Complete blood count, bone marrow] (f) Neuroblastoma [D/b Bone marrow, biopsy] (g) osteomyelitis or septic arthritis [D/b History]

(4) **Local erythema or bullae** will occur in (a) Bacterial Cellulitis, Pyoderma gangrenosum and Staphylococcal impetigo [D/b Culture, Gram stain] (b) Herpes, zoster or simplex [D/b Scraping, culture] (c) Epidermolysis bullosa [D/b Skin biopsy] (d) Contact dermatitis, allergic or irritant [D/b Clinical characteristics]

(5) **Metaphyseal lesions, epiphyseal lesions**, or **both** are seen in (a) Scurvy [D/b Nutritional history](b) Menkes syndrome [D/b .copper, .ceruloplasmin] (c) Syphilis [D/b serology] (d) "Little League" elbow [D/b History] (e) Birth trauma [D/b History]

(6) **Physiological striae** - Linear red to purple marks may occur in teenagers due to sudden growth spurt.

vi. Investigation

Family history – Record any previous infant deaths in similar circumstances. A similar previous death strongly goes in favor of child abuse.

b. Shaken baby syndrome (SBS)

Shaken baby syndrome [***SBS***, ***infantile whiplash syndrome, whiplash Shaken Infant Syndrome***] refers to a constellation of signs and symptoms resulting from violent shaking of the head of an infant or small child.

VIII. SUDDEN INFANT DEATH SYNDROME (SIDS)

Sudden Infant Death Syndrome [syn. cot death, crib death, SIDS, sudden unexplained infant death, SUID] is defined as the sudden death of a seemingly healthy infant [age <1y], whose death remains unexplained even after (i) thorough case investigation (ii) death scene examination (iii) review of clinical history (iv) complete autopsy and (v) serological, toxicological, histopathological and microbiological examinations.

Salient features:

(1) **History** – typically child is quite well, when put to bed, or may only have a minor URI [common cold, sneezing] or minor GIT disturbances

(2) **Simultaneous sudden infant death syndrome** [SSIDS] – Unexplained death of 2 or more infants takes place within 24 h.

A. Theories of Causation

No one is sure why SIDS occurs, but there are several theories. Death may result from a number of causes, which combine and act via a common pathway of cardiorespiratory failure, while the child is passing through a vulnerable period of development. The main theories are:

(1) **Allergies** -

(i) Cow's milk protein allergy

(ii) House mite allergy

(2) **Critical Diaphragm Failure** [CDF] - Respiratory failure caused by CDF. Four principal risk factors contributing to CDF are

(i) undeveloped respiratory muscles

(ii) non-lethal infections

(iii) prone resting position

(iv) REM sleep. Even relatively minor infections can cause an acute and significant reduction in diaphragm force generation capacity that in conjunction with other risk factors precipitate CDF.

(3) **Deficiencies** – of (i) Antibodies (ii) Biotin (iii) Calcium, magnesium, selenium (iv) gamma globulins [hypogammaglobulinemia] (v) Vitamins B,C,D,E.

(4) **During pregnancy** – certain factors were present that inhibited fetal circulation [eg cocaine use (**ch 41**] "damage of fetal brain"medulla could not control breathing properly

(5) **Genetic:**

(i) Brainstem abnormality of the serotonergic system – giving rise to defective respiratory and autonomic responses.

(ii) Cardiac channelopathies – Genes coding for faulty ion channels or ion channel-associated proteins"(a) long QT syndrome, (b) short QT syndrome (c) Brugada syndrome (d) catecholaminergic polymorphic ventricular tachycardia.

(iii) Faulty Interleukin-1 receptor antagonist (IL-1RA) protein – IL-1RA modulates a variety of interleukin 1 related immune and inflammatory responses, and is encoded by the IL1RN gene. There are 3 known variants – Allele 1 is normal. Alleles 2 and 3 are associated with -ed risk of SIDS.

(iv) Left ventricular hypertrabeculation – This condition is characterized by a highly trabeculated, “spongy” myocardium"causes heart failure, arrhythmia, thromboembolic events. A wide variety of mutations are responsible for hypertrabeculation.

(v) MAO-A molecular polymorphisms.

(6) **Hormonal** - Deficiency of parathyroid.

(7) **Infections** - Respiratory [bacterial - S. aureus; viral]

(8) **Inflammatory reactions** – In a small group of SIDS cases very high cytokine [IL-1ß, IL-6, TNF**α**] concentrations are seen [“cytokine storm”]. May be a possible explanation for SIDS.

(9) **Obstruction of airways** - (i) Anatomical abnormalities (ii) Nasal edema (iii) Nasal obstruction or inflammation or both (iv) Excessive mucus in respiratory passages (v) Flaccid pharynx in hypotonic babies (vi) Faulty neck posture (vii) Laryngeal spasm

(10) **Prone sleep position** – Children who are made to sleep in prone position have 3 times - risk. Parents are now advised a supine sleep position for infants. Following factors may trigger death in this position

(i) Asphyxia due to airway compression

(ii) Rebreathing of exhaled gases in the face-down position

(iii) Impaired heat loss with subsequent hyperthermia when the face is pressed against bedding

(iv) impaired cardiorespiratory regulation related to heat stress

(v) More bacterial growth – There is heavier growths of S. aureus and E. coli in nasal samples from infants who sleep on their stomachs, compared with babies who sleep on their backs.

(11) **Sharing of beds** - Approximately 50% of sudden infant deaths occur when infants are sharing a bed with another person [co-sleeping].

(12) **Sleep apnea** – Prolonged sleep apnea in certain infants"hypoxia"death. Infant is at the highest risk for SIDS during sleep [reason for calling it cot death or crib death].

(13) **Sleep-induced arrhythmias**

(14) **Toxic** - Mattress related – Most mattresses contain P, As, or Sb compounds as fire retardants"warmth and perspiration of the sleeping infant"infection by microorganisms [esp the fungus Scopulariopsis brevicaulis]"generating phosphines, arsines and stibines from materials containing P, As, or Sb" Poisoning to death.

(15) **Miscellaneous** -

(i) Adrenal insufficiency

(ii) Conduction system anomalies

(iii) Deficient liver enzymes

(iv) Gastroesophageal reflux"bradycardia

(v) Hyperthermia

(vi) Hypothermia

(vii) Impaired glial-neuronal interactions in the cardio-respiratory centre of the brainstem"stoppage of heart and respiration.

(viii) Metabolic disorders
(ix) Neurogenic shock
(x) Sodium overload in feeds
(xi) Suffocation by bedclothes and pillows.

B. Epidemiological Features

(1) **Incidence** – 0.6/1000 live births
(2) **Age** – Peak incidence at 2-4 months
(3) **Sex** - male predominance
(4) **Geographical distribution** - Worldwide
(5) **Environmental** – Exposure to cigarette smoke or use drugs of abuse while in the womb [**ch 46**] or after being born.
(6) **Child related:**
(i) Birth weight - Risk is higher in preterm infants or those born with low birth weight
(ii) Immunizations - (a) General - SIDS cases are immunized less frequently and later than controls. Early and more frequent immunizations may reduce the risk of SIDS. (b) Hexavalent vaccines – Some workers have found an -ed risk of SIDS in children receiving hexavalent vaccines [diphtheria, tetanus, acellular pertussis, Haemophilus influenzae type B, poliovirus and hepatitis B (DTaP-Hib-IPV-HepB)]. These were introduced in Europe in 2000.
(iii) Multiple birth babies –incidence is 3 times in members of a twin pair and still more in triplets. Could be because multiple birth babies are generally premature and of low birth weight
(7) **Parent related** -
(i) Age of mother - - risk to those born to young mothers.
(ii) Alcoholic mother – Incidence of SIDS is -in children having alcohol-consuming mothers than in those non-alcohol-consuming mothers. Furthermore even in alcohol-consuming mothers, the incidence -es significantly on weekends and New Year, when there is -ed consumption of alcohol. It is hypothesized that alcohol impairs parental capacity to care for their children.
(8) **Socioeconomic** – occurs more in poor families
(9) **Time of death** – Death always occurs during sleep. At all times of night, with a moderate -in the early morning hours

(10) **Season** – More common in winters
(11) **Latitude** – More prevalent in countries with higher latitudes
(12) **Miscellaneous factors** – Risk is -ed in following conditions
(i) Having a brother or sister who had SIDS (ii) Heads covered with bedclothes. (iii) Late or no prenatal care (iv) Premature birth (v) Short time period between pregnancies (vi) Soft bedding in the crib.

C. Symptoms

Almost all SIDS deaths occur without any warning or symptoms when the infant is thought to be sleeping.

D. Autopsy

All changes are not seen in any given individual case.
(1) **Face** -
(i) Facial pallor
(ii) No petechial hemorrhages in face or eyes
(2) **Hands** –
(i) Clenched
(ii) Contain fibres from bed clothes
(3) **Definite pathology** – found in about 15% brought as SIDS, eg (i) Congenital heart disease (ii) Down's syndrome (iii) Pneumonia (iv) tracheobronchitis. But by definition, once significant pathology is discovered, they no more remain cases of SIDS, and autopsy report should not mention this term. The term SIDS should be entered as a cause of death, only when either no findings are discovered, or very minor findings insufficient to explain death.
(4) **Milk or blood stained froth** – in child's nostrils, mouth or bedding
(5) **Respiratory passages** – (i) froth (ii) milky vomit (iii) shedding of individual tracheobronchial epithelial cells
(6) **Inflammatory signs** - (i) Laryngitis (ii) Tracheitis (iii) Bronchitis (iv) Bronchiolitis (v) Pneumonitis (vi) pleuritis, either individually or in various combinations
(7) **Lungs** – (i) Surface - Patchy or uniform purplish discoloration (ii) Consistency – firm (iii) wt--ed (iv) pathology – congestion, edema
(8) **Petechial hemorrhages** – on visceral surfaces of heart, lungs and thymus.

Most common finding [seen in about 75% cases)]. Could be due to terminal inspiratory efforts against a closed glottis.

(9) **Histopathology** –

(i) Bronchi – peribronchiolar cell infiltration

(ii) Lungs - (a) patchy alveolar collapse (b) Alveolar walls - thickened (c) aggregations of (I) platelets (II) neutrophil polymorphonuclear granulocytes and (III) lymphocytes (IV) monocytes in the lung capillaries. Indicative of early inflammation In about 1/5th cases, inflammatory cells are also found in alveolar spaces [localized areas of bronchopneumonia] (d) capillary thrombosis [producing ischemia during life].

(iii) **Brain** - (a) brainstem - bilateral hypoplasia of the arcuate nucleus.

(iv) **Heart** - (a) Conduction system - resorptive degeneration.

(10) **Biochemical** - -ed levels of cytokines [IL-1ß, IL-6, TNF**α**] in blood and CSF.

(11) Genetic testing to exclude genetic disorders.

E. ML Importance

(1) **Infanticide** – May be misdiagnosed as SIDS due to lack of evidence. **Meadow's dictum** states that "one sudden infant death is a tragedy, two is suspicious and three is murder, until proved otherwise".

(2) **SIDS infants as organ donors** – A lot of confusion existed earlier regarding the validity of using heart and other organs from SIDS infants because of unclear etiology. Studies have now proved that heart and other organs from SIDS infants can be transplanted in other infants with no -ed incidence of death in recipients.

(3) **Civil and Criminal cases involving SIDS** – have generally failed in courts, because courts recognize that this is a natural phenomemon.

28. Forensic Psychiatry

I. INTRODUCTION

[A] **Psychiatry** - Deals with the study diagnosis and treatment of mental illness. [B] **Forensic Psychiatry** - Application of psychiatry for the purposes of law and administration of justice. [C] **Mental Health Act, 1987** (MHA)- Law passed by the govt of India in 1987 relating to treatment and care of mentally ill persons. This Act does not mention the words ***insane, insanity, lunatic*** and ***lunacy*** anywhere. Instead the term 'mentally ill person' is mentioned and defined. These terms should best be avoided. According to S.2(l) of MHA, a ***mentally ill person*** is one who is in need of treatment, by reason of any mental disorder other than mental retardation.

II. KEY TERMS IN FORENSIC PSYCHIATRY

A. Abreaction

Abreaction is reviving and bringing into consciousness traumatic experiences of the past, which the patient has repressed subconsciously.

B. Actus Reus (guilty action)

Actus reus refers to ***criminal act***. See also mens rea.

C. Affect

A term related to mood, feeling or emotion. It is described as mood, or inner feelings at a particular moment. If mood is emotional 'climate' [more pervasive and sustained], affect is emotional 'weather' [more fluctuating changes].

D. Affective Disorder

A disorder in which ***affect*** is disturbed (e.g. bipolar disorders, depression).

E. Amnesia

Amnesia is loss of memory. May be seen in head injury. **Retrograde amnesia** - Extends backwards in time from the point of head injury. **Anterograde amnesia** – Extends forwards in time from the point of head injury.

F. Compos Mentis

Having a sound mind [from Latin, composed mind]. For recording certain statements eg dying declaration, making a valid will, it must be certified that the declarant was compos mentis.

G. Confabulation

Confabulation refers to false answers given by ***amnesia*** patients without a deliberate attempt to mislead. Typically seen in Korsakoff's psychosis [please see ch 40]. Confabulation is different from lying, but may be very difficult to differentiate from it.

H. Delirium

Delirium [Latin de lira; "out of the furrow", "off the track"] is a common and severe neuropsychiatric syndrome with core features of (1) acute onset (2) fluctuating course (3) attentional deficits and (4) reduced clarity of awareness of the environment.

Salient features:

(1) **Commonest organic mental disorder seen in clinical practice**. 5-15% of all medical and surgical inpatients develop delirium. This percentage is higher in postoperative patients.

(2) Earlier known as acute **b**rain syndrome, acute **c**onfusional state, acute **o**rganic reaction, metabolic encephalopathy and toxic psychosis.

(3) **Causes:** (i) Medical conditions - [especially when there is continuous - temp] e.g. CNS infections. Also in head trauma (ii) Toxic substances e.g. datura

(4) **MLI:** (i) person may become impulsive, violent and may commit homicide or suicide (ii) A person in delirium is **not responsible** for his acts (iii) Wills made in delirium are not valid.

I. Delusion

Delusion is a false belief in (i) something which is not a fact, and (ii) which persists even after its falsity has been clearly demonstrated, and (iii) which is not widely prevalent in persons of similar socioeconomic and educational status. Dislikes, hatred and prejudices however ill-found are not insane delusions.

1. Types of delusions

(1) **Delusional Misidentification Syndromes** - include **four** types of syndromes:

(i) Capgras syndrome (Delusion of doubles, Illusion des sosies) - Person holds a delusion that a friend, spouse, parent, or other close family member has been replaced by an identical-looking impostor [stranger]. Familiar person is thus seen as a stranger.

(ii) The syndrome of subjective doubles - The patient's own self is perceived as being replaced by a double.

(iii) The syndrome of intermetamorphosis - Familiar persons are believed to have exchanged identities [no stranger is involved]. A person misidentifies his wife as his deceased mother and, later, as his living sister.

(iv) Fregoli syndrome ***(Illusion de Fregoli*, The Fregoli delusion, the delusion of doubles**) – Delusional belief that one or more familiar persons, usually persecutors following the patient, **repeatedly change their appearance**.

Memory Aid 1: Fregoli Syndrome

In **F**regoli syndrome, person thinks that **f**amiliar persons **f**ollow him and change their **f**igure [appearance].

(2) **Erotomania** - One believes that another person, usually someone of higher status, is **in love with him or her** [**de Clérambault's syndrome**]. The affected individual attempts to contact the other person (through phone calls, letters, gifts, and sometimes stalking).

(3) **Grandeur** (syn. Inflated self-esteem and self-image)- The affected person thinks that he is very rich, while in reality he may be a pauper.

(4) **Hypochondriacal** (syn. Somatic) – The person believes he is ill, although he is perfectly healthy

(5) **Infidelity** (syn. Jealousy) – Person thinks that his wife is unfaithful to him while she is chaste. Also known as **morbid jealousy**, **delusional jealousy**, **sexual jealousy**, **Othello syndrome** or **Othello psychosis**. (In the Shakespearean play 'Othello', the main character Othello thinks that his wife Desdemona is infidel and strangles her. He then kills himself with a sword!). The syndrome may appear by itself or in the course of paranoid schizophrenia, alcoholism, or cocaine addiction.

(6) **Influence** – Also known as

C

lérambault-Kandinsky syndrome [**Memory Aid 2**]. The person complains that his actions, feelings and thoughts are being

C

ontrolled or influenced by some outside agency – sometimes even with the help of hypnosis, telepathy etc.

(7) **Nihilistic** – The person feels he does not exist [**Cotard syndrome**].

Memory Aid 3: Cotard Syndrome

In Cotar **d** syndrome, person thinks he is **d**ead

(8) **Persecution** (paranoia) - The person thinks that people around him are trying to kill him [most common type of delusion]. The affected individual is frequently highly litigious.

(9) **Reference** – The person believes that everyone is looking at him or talking about him, or that items in newspapers or radio are referring to him

(10) **Self-reproach** – The person blames himself for past failures and misdeeds which are of no significance

(11) **Miscellaneous:** delusions of jealousy, religion etc.

2. Medicolegal importance of delusions

(1) **Criminal responsibility** [CR]– To determine CR, the delusions must be presumed to be real, and then it must be determined if the act done was criminal or not [for details, please see below - McNaghten rule " examples].

(2) **Testamentary capacity** – A person affected by an insane delusion can make a valid will, if the delusion is not related in any way with the disposal of property or the persons affected by will.

J. Fugue

Fugue (pronounced - Fyoog) is a state of altered awareness during which the affected person forgets his identity, assumes a new identity, leaves home and wanders. It occurs in depressive illness, epilepsy, hysteria and schizophrenia.

K. Hallucination

Hallucination is a false sense perception without any external object or stimulus to produce it.

Salient features:

(1) **Hypnagogic hallucination** – is a vivid dreamlike hallucination which occurs as one is falling asleep. The opposite is **hypnopompic hallucination**, which occurs as one is waking up. [g " p]. don't indicate pathology.

(2) **Pseudohallucination** - is one in which the patient knows the stimulus is in the mind.

(3) **Types** - They can occur in any sensory modality: (i) **Auditory** – Also known as Paracusia. Person hears voices and imagines that someone is speaking to him, when no one is present. (ii) **Command hallucinations** – Also known as command automatism [please also see **automatism** below]. Patient is ordered by hallucinatory voices to do unwanted and undesirable things. May even commit suicide or homicide at the order of such imaginary voices. Found closely in association with delusions of influence [please see above] (iii) **Gustatory** – Feeling of tastes when no substance is in mouth (iv) **Olfactory** – perceiving smells, when none is there. Also known as **phantosmia** (v) **Psychomotor** –Person has feeling of movement of some part of the body, where there is none. (vi) **Tactile** – Also known as haptic hallucinations. One may feel as if insects are crawling underneath the skin, while there are none. Seen in chronic cocaine abuse (vii) **Visual** – Person may see something, which does not exist (God, imaginary object, fearful and attacking animal, snakes etc).

L. Illusion

Illusion is a false interpretation by the senses of an external object or stimulus which has a real existence. **Ex**. (i) A rope is hanging from a tree and the person perceives it to be a snake. There is a real stimulus (rope), which is false perceived as snake. In hallucinations, a person may see snakes hanging from the tree, where even the rope is not there. A normal person may have illusions but on proper explanation, he will realize his mistake (as when another person goes and touches the rope to tell him it is not snake). But a mentally ill person may insist it is a snake. (ii) Stem of a tree " patient thinks it is a ghost standing (iii) Birds singing " patient thinks some person is speaking about him (iv) Person comes with a dog to meet him " Patient thinks he has brought a lion to kill him.

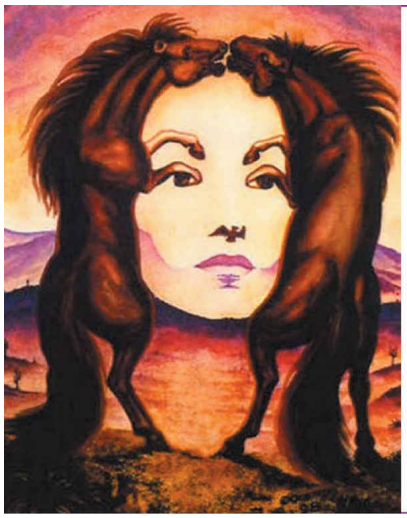


Fig. 28.1: "Horses or face" illusion. The figure may be interpreted in two ways – one can see horses or face. While a normal person realizes after some time that he is looking at an illusory figure, a psychiatric patient may not realize that, and may keep believing in the existence of both.

M. Impulse

An ***impulse*** is a sudden and irresistible urge, compelling a person to perform some action without motive or forethought.

(1) **Types:**

(i) **Dipsomania** – An irresistible desire to consume alcohol

(ii) **Kleptomania** – An irresistible desire to steal (stolen objects may be of little value)

(iii) **Mutilomania** - An irresistible desire to mutilate

(iv) **Pyromania** - An irresistible desire to set buildings on fire

(v) **Sexual impulses** - An irresistible desire to perform a perverted sexual act

(vi) **Suicidal** and **homicidal impulses** - An irresistible desire to commit suicide or homicide

(vii) **Trichotillomania** - An irresistible desire to pull out body hair.

(2) **ML aspects** – In India person is criminally responsible, because he knows what he is doing [please see **M'naghten rule** and **S.84, IPC** below]. Under the more advanced **irresistible impulse test** [not followed in India], person is not criminally responsible.

N. Lucid Interval

Lucid interval (L. lucidus, clear) is a period of clarity of thought between two subsequent bouts of mental illness [**Fig 28.2**].

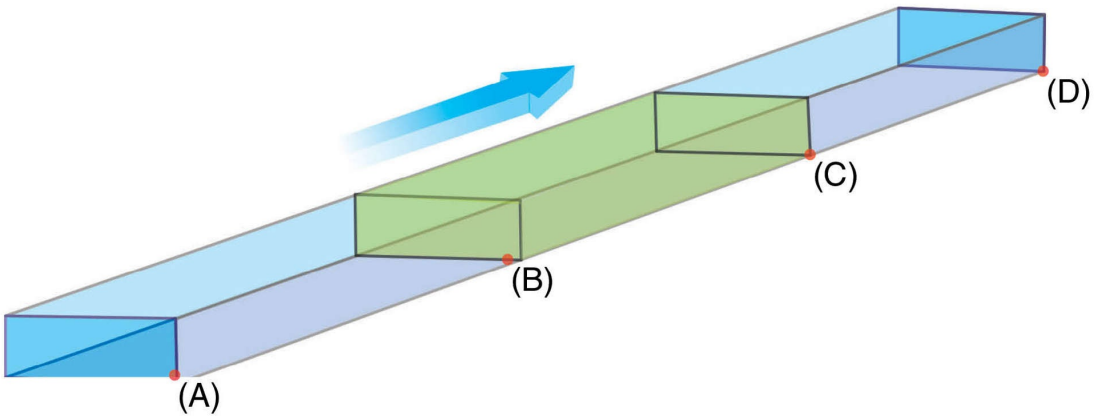


Fig. 28.2: Lucid interval seen in mental illness. AB & CD = Manic phase; BC = Depressive phase (Lucid interval).

Salient features:

(1) Most commonly seen in bipolar disorders [between two subsequent manic attacks].

(2) It is also seen in **head injury**, where it refers to a period of consciousness between two successive periods of unconsciousness [**ch 17; Table 1**].

(3) **MLI** – The person is responsible for all civil and criminal acts done during lucid interval, eg making a will, entering into a contract (civil), committing a murder (criminal).

Table 1: Differences between lucid interval of head injury and insanity

S.No	Feature	Head injury	Insanity
1.	History	Of injury	Of mental disease
2.	Lucid interval preceded by	phase of concussion	Phase of symptoms of insanity
3.	Lucid interval followed by	Phase of cerebral compression	Recurrence of symptoms of insanity
4.	Number of episodes	Only once	Recurrent

O. Mania

Mania is one of the episodes of bipolar (manic-depressive) disorder (the other being depressive), characterized by an elevated, or irritable mood state, an increase in energy, decreased need for sleep and impaired judgment.

P. Mens Rea (guilty mind)

Mens rea refers to criminal intent.

Salient features:

Legally speaking, **crime = *mens rea* + *actus reus***. If either actus reus or mens rea is absent, crime does not take place legally.

Q. Oligophrenia

Oligophrenia is another name for feeblemindedness, mental subnormality or mental retardation.

Salient features:

1. Older system of classifying oligophrenia used terms like **idiot, imbecile** and **moron.** Because of stigma attached to such terms, this classification is not

used now. Both older and modern classifications are compared in **Table 2**.

Table 2: Modern classification of subnormal IQs compared with older system

IQ Range	Modern Classification	Older classification	Mental age [in yrs]
< 20	Profound	Idiot	<3 y
20-34	Severe	Imbecile	4-7
35-49	Moderate		
50-69	Mild	Moron	8-15
70-80	-	Borderline deficient	>15

Memory Aid 4: Modern and older classification of subnormal IQs

P S M^2 "**P**rofound, **S**evere, **M**oderate, **M**ild

I I M , **B**angalore"**I**diot, **I**mbecile, **M**oron, **B**orderline

Memory Aid 5: Imbecile

Mental age of an **imbecile** [several and moderate intellectual disability] is below the **age of criminal responsibility** [**7 y**]

2. Classification of individuals with higher IQs was given by Lewis Terman (1877-1956) [**Table 3**].

Table 3: Classification of various supernormal IQs

IQ Range	Classification
90-110	Normal or average intelligence
110-120	Superior intelligence
120-140	Very superior intelligence
140 and over	Genius or near genius

R. Oneiroid States (Oneirophrenia)

Oneirophrenia is a hallucinatory dream-like state [Gk 'oneiros' (dream); 'phrenos' (mind)]. Shares some characteristics with simple schizophrenia (e.g confusional state and clouding of consciousness). **Causes** - prolonged sleep deprivation, drugs (e.g. ibogaine).

S. Phobia

A ***phobia*** (Gk: phobos, fear) is an irrational, intense and persistent fear of certain situations, activities, things, animals, or people. The main symptom is the excessive and unreasonable desire to avoid the feared stimulus. The fear is beyond one's control, and interferes with daily activities. Phobias may develop to almost any object or situation. **Some important phobias** are: (i) Acrophobia, Altophobia – fear of heights. (ii) Agoraphobia – fear of large open places (from where escape is impossible) (iii) Aviophobia – fear of flying. (iv) Claustrophobia – fear of confined spaces. (v) Hemophobia – fear of blood. (vi) Mysophobia – fear of germs, contamination or dirt. (vii) Nosophobia – fear of contracting a disease. (viii) Xenophobia – fear of strangers, foreigners, or aliens.

T. Psychosis

Psychosis [Gk psyche, mind; osis, disease] is an abnormal condition of the mind, when there is a loss of contact with reality. People suffering from psychosis are described as psychotic. Neurosis is now no more used as a diagnostic term, but

when it was used, it was usual to differentiate it from psychosis (**Table 4**).

Table 4: Difference between true insanity and feigned insanity

S. No.	Trait	True insanity	Feigned insanity
1.	Onset	Gradual	Sudden
2.	Motive	Absent. There is no history of any crime	Present. Has committed some crime. Wants to take benefit of S.84, IPC
3.	Predisposing factors	Usually present. History of insanity in parents may be present. Sudden grief, loss of a dear one, monetary loss,	Absent
4.	Signs and symptoms	Uniform and present whether the patient is being observed or not	Present only when he knows he is being observed. Symptoms are variable and haphazard. Do not fit in any definable illness. May be excessively exaggerated
5.	Facial expression	Peculiar. Vacant look	No peculiarity. Frequently changing. Exaggerated
6.	Insomnia	Usually present. May not sleep for several days.	Absent
7.	Exertion	Patient can stand exertion, fatigue and hunger.	Cannot stand exertion for more than a few days. Breaks down after that
8.	Habits	Dirty. Filthy	Not so
9.	Skin and lips	Dry, harsh	Normal
10.	Reaction to frequent examinations	Does not mind, as he has nothing to conceal	Resents repeated examination, as he fears detection of malingering

III. THE MENTAL HEALTH ACT, 1987

The **Mental Health Act** was passed by the Indian Parliament in **1987**. The **aims and objectives** were to consolidate and amend the law relating to mentally ill persons, their better treatment and care, better management of their property, and their overall better protection. It is a social welfare legislation. It repealed the **Indian Lunacy Act, 1912.** It changed certain objectionable and stigmatic terms into more acceptable terms (**Table 5**).

Table 5: New terms used by the Mental Health Act, 1987, in place of older terms used in Indian Lunacy Act, 1912.

Older terms used in Indian Lunacy Act, 1912	New terms used in Mental Health Act, 1987
Mental hospital	Psychiatric hospital
Lunatic	Mentally ill person
Criminal lunatic	Mentally ill prisoner

Salient features:

(1) **Mentally ill person** is **defined** as a person who is in need of treatment by reason of any mental disorder other than mental retardation [**S.2(l)**]

(2) **License** – (a) Establishment and maintenance of psychiatric hospitals or psychiatric nursing homes can only be with **license** [**S.6**], which has to be renewed every 5 years [**S.94**.] (b) The license will be **revoked** if the hospital is not maintained according to provisions of the Act [**S.11**] (c) if hospital is run without license, the penalty is **imprisonment of 3 months** and/or **fine of `200** for first offence, **6 m** and/or **`1000** for second offence and if hospital is continued to run without license even after that, a penalty of **`100 per day** [**S.82**]

(3) **Regular inspection of psychiatric hospitals by Inspecting Officers** – In order to know that psychiatric hospitals are working well and in accordance with the Act, the State Government or licensing authority will appoint an '**Inspecting Officer**' who at any time can enter and inspect any psychiatric hospital, inspect its records and talk to patients in private. If working of hospital not found satisfactory, he will report to the licensing authority who will take appropriate action [**S.13**]

(4) **Appointment of visitors and monthly inspection by them** - For every psychiatric hospital, the Govt shall appoint 5 or more visitors (of these at least **one** should be a **medical officer**, preferably a psychiatrist, **two** should be **social workers** and two others) [**S.37**]. Every month three or more visitors

will make a joint inspection of every part of the psychiatric hospital and examine every minor admitted as a voluntary patient, and preferably every other mentally ill person admitted [**S.38**]. Regular visits by inspecting officers and visitors ensure that psychiatric hospitals are run smoothly.

(5) **Proper admission and discharge of patients** – Please see below under 'restraint of the mentally ill'

(6) **Penalty for improper reception of mentally ill person** – If a mentally person is improperly admitted or detained in a psychiatric hospital, the punishment is **imprisonment** up to **2 years** or with **`1000** fine or both [**S.83**].

IV. RESTRAINT OF THE MENTALLY ILL

Restraint of the mentally ill refers to lawful restraint of a mentally ill person [MIP], who is a danger to himself or others. It can be (A) Immediate restraint or (B) Admission in a psychiatric hospital.

A. Immediate Restraint

Immediate restraint means taking a mentally unsound person into immediate control whose illness has exacerbated acutely, or who has suddenly become violent. There is no specific provision in MHA 1987 for immediate restraint. It is done **U/s 81, IPC** [**act done to prevent other harm**].

Salient features:

(1) **Conditions when immediate restraint is required** - Illnesses where a person (i) may become dangerous to himself or (ii) to others (iii) may wastefully spend his own property or (iv) that of others.

(2) **Such illnesses are:** (i) Delirium tremens (ii) mania (iii) Organic delirium

(3) **Purpose:** (i) To prevent such injury to himself, others and to the property (ii) To prevent wasteful expenditure of his own or others' property.

(4) **Prerequisites** – informed consent of guardian must be obtained. If consent not obtainable for any reason, person can be restrained without consent, but only as long as danger exists.

(5) **Method** – by safely locking up in a room under immediate personal care of attendants.

B. Admission in a Psychiatric Hospital

Elaborate procedures for admission in a psychiatric hospital have been made so no person may exploit the law. If a person is aggrieved over an unfavorable will made by a relative, he may try to prove him mentally ill by getting him admitted to a psychiatric hospital. A criminal may himself get admitted in order to claim benefit of **S.84, IPC**.

1. Admission on a voluntary basis

(1) **Major** – Any person >18 y who considers himself a mentally ill person [MIP] and desires to be admitted to any psychiatric nursing home for treatment, may request the doctor in charge for being admitted as a voluntary patient [**S.15**].

(2) **Minor** – In case of persons <18 y, request is made by guardian [**S.16**].

(3) **Procedure** – The doctor in-charge shall make such inquiry [examination etc] as he may deem fit within 24 h and if satisfied that the applicant or minor requires treatment he may admit him [**S.17**].

(4) **Comments** – A straightforward procedure. The possibility of patient malingering illness is prevented by doctor himself examining such person. Very few patients are admitted under this law.

2. Admission under special circumstances [application by relative or friend]

If mentally ill person [MIP] is *unable to express his willingness* for admission

(i) Patient may be admitted for up to 90 days on an application by relative or friend [**S.19(1)**] (ii) Application should be on prescribed form (iii) It should be accompanied by certificates from 2 medical practitioners [allopath, homeopath, or ayurveda (**S.2k**)], one of whom should be a Govt doctor (iv) each doctor should have examined the MIP separately within 10 days prior to application [**S.30**]. (v) If above certificates are not attached, the doctor in charge may appoint 2 doctors working in the hospital to examine him [**S.19(2)**].

3. Reception order on application

(1) **Reception order** [RO]– means a magisterial order for MIP to be “received”, admitted and kept in hospital.

(2) **Application by doctor in charge** – If doctor thinks that treatment is needed for >6 m or detention is needed for patient’s own safety and that of others, he

would make an application to magistrate for a reception order [**S.20(2)**].

(3) **Application by husband, wife or relative:**

(i) Husband or wife of patient can make an application to magistrate.

(ii) If there is no husband or wife or if they are ill or absent or can not make an application for whatever reason, any relative of patient can make an application [**S.20(3)**].

(iii) He must state why the husband or wife is not making the application. He should also indicate his relationship with the patient, and the circumstances under which the application is being made [**S.20(4)**].

(iv) All persons making application under this provision [husband, wife, relative] must be >18 y and must have seen the patient within 14 days of making application [**S.20(5)**].

(v) The application must be in prescribed form and must be accompanied by certificates from 2 medical practitioners, one of whom must be a Govt doctor [**S.20(6)**].

(vi) Each medical practitioner should examine the patient independently [**S.21(a)**]

(vii) The certificate should state that the MIP is suffering from mental disorder of such a nature and degree that his treatment in the psychiatric hospital is required and that such detention is necessary in the interests of the health and personal safety of patient or for the protection of others [**S.21(b)**].

(4) **Procedure:**

(i) When application is made by doctor – Magistrate would pass an RO, if he is satisfied [**S.22(1)**]

(ii) When application is made by husband, wife or relative – (a) magistrate would consider statements made in application and also the medical certificates [**S.22(2)**] (b) he may personally examine **A**lleged **M**entally **I**ll **P**erson (AMIP) [**S.22(3)**] (c) If he is satisfied that the patient needs treatment, he would immediately pass RO; if not, he shall fix a date for further consideration [**S.22(4)**] (d) He will give notice of date fixed to the applicant, and to any other relevant person [**S.22(5)**] (e) Till the application is disposed, magistrate would pass order for proper care and custody of AMIP [**S.22(6)**] (f) On the date fixed for disposal of application, the magistrate would consider the application ***in camera***, in presence of (i) the applicant (II) AMIP himself (III) a representative appointed by AMIP (IV)

any other person whom magistrate thinks is fit to be there [eg a doctor, a neighbor, friend etc]. After listening to all parties, the magistrate may either pass an RO [no time limit of detention] or dismiss the application. The magistrate would decide whether the cost of such enquiry would be charged from applicant or from the mentally ill person [**S.22(7)**]. (g) If application is dismissed, the magistrate shall record reasons for dismissal and shall give a copy to the applicant [**S.22(8)**].

4. Reception order on production of mentally ill person before Magistrate

a. Wandering or dangerous mentally ill person

(1) **Detention -** An police officer in charge of a police station may detain any wandering mentally ill person (WMIP) [who is wandering aimlessly] or dangerous mentally ill person (DMIP) [who is dangerous to himself or others due to his violent behavior][**S.23(1)**].

(2) **Information** - The Police officer would immediately inform him why he is being detained. If WMIP or DMIP is incapable of understanding, their relatives or friends must be informed [**S.23(2)**].

(3) **Production before magistrate** - Such WMIP or DMIP must be produced before a magistrate within 24 h of detention. The period of journey is not included [**S.23(3)**].

(4) **Procedure:**

(i) In respect of examination - Magistrate shall (a) examine the person personally (b) cause him to be examined by a doctor and (c) make any other relevant enquiries [**S.24(1)**].

(ii) In respect of admission – Doctor must certify the person to be mentally ill. If he feels that patient may be malingering and needs sustained observation or there is some other difficulty in on-the-spot diagnosis, he may say so to the magistrate and he would pass a temporary order for detention for observation of a period of up to **10 days** [**S.28(1)**]. If diagnosis cannot be made during that period, two more such orders can be passed. The doctor must make a diagnosis within **30 days** [**S.28(2)**]. If after medical certificate from doctor the magistrate is satisfied of the need of his treatment or for protection of himself or others (a) he may make an RO. (b) If any relative or friend wants him to be admitted to a particular licensed psychiatric

hospital and agrees to bear cost, and doctor in-charge of that hospital consents, an RO for that particular hospital is made. (c) If relative or friends furnishes a bond ensuring that WMIP or DMIP would neither injure himself nor others, and agrees to keep him, the magistrate, instead of making an RO, would hand over such person to the relative or friend [**S.24(2)**].

b. Cruelly treated mentally ill person

(1) **Information to magistrate:**

(i) Police officer – may inform magistrate if an MIP is in the custody of relative or guardian, and is being cruelly treated by him [cruelly treated mentally ill person, CTMIP] [**S.25(1)**].

(ii) Any private person – can similarly inform magistrate [**S.25(2)**].

(2) **Procedure:**

(i) The magistrate would call CTMIP, relative who is cruelly treating him and person who is legally bound to maintain such CTMIP [**S.25(3)**].

(ii) Magistrate would order the person who is legally bound to maintain such CTMIP to take his proper care. If he willfully neglects to comply with the order, he can be fined up to `**2,000** [**S.25(4)**].

(iii) If it appears to magistrate that despite fine etc, the CTMIP would not be properly looked after, he would make an RO, under same procedural guidelines as with WMIP and DMIP above [**S.25(5)**].

(3) **Powers of Police Commissioner [PC]**– In an area where PC has been appointed, he would discharge all powers and functions of magistrate in respect of WMIP, DMIP and CTMIP [**S.36**].

C. Discharge of a Mentally Ill Person

(1) **Voluntary patient:**

(i) If patient, or minor was admitted voluntarily, normally he should be discharged within 24 h of a discharge request [**S.18(1)**]

(ii) If minor during admission attains majority, the doctor would intimate him of this fact, and if no application from him for continuation of admission is received within 1 month of such intimation, he would be discharged [**S.18(2)**]

(iii) If doctor thinks more treatment is required, he would make a Board of 2 doctors within 72 h of receipt of application of discharge and seek its

opinion. If Board recommends continuation of treatment, the patient can be kept for 90 days against his will [**S.18(3)**].

(2) **When admission was on application by relative or friend:**

(i) Mentally ill person himself, or any of his relative or friend may apply to Magistrate for discharge.

(ii) Magistrate would give a notice to the person on whose application patient was admitted (iii) On hearing all parties he would either allow or dismiss application [**S.19(3)**]

(3) **Powers of officer-in-charge of a psychiatric hospital:**

(i) He can discharge any patient other than a voluntary patient on the recommendation of 2 doctors, 1 of whom should preferably be a psychiatrist. But a mentally ill prisoner cannot be so discharged [**S.40(1)**]

(ii) A copy of discharge order must be sent to such authority [eg magistrate] on whose reception order the patient was admitted [**S.40(2)**].

(4) **Discharge of mentally ill persons on application** – A mentally ill person detained under RO made on application shall be discharged if the person on whose application the admission order was made, applies in writing to the medical officer in-charge. However if the medical officer certifies in writing that the person is dangerous and unfit to be at large, he cannot be discharged [**S.41**].

(5) **Discharge of a person on his own request** – Any MIP (not being a mentally ill prisoner) who feels he has recovered from his illness may make an application to a magistrate. He must attach a certificate from the medical officer in-charge of the hospital where he undergoing treatment, or from any other psychiatrist. Magistrate may pass an order for his discharge if he deems fit [**S.43**].

(6) **Person detained on reception order** – Subsequently found by judicial inquisition to be of sound mind, he shall be discharged [**S.44**].

V. MENTAL ILLNESS AND RESPONSIBILITY

Responsibility means legal liability of a person for his actions. It can be in civil or criminal matters. Law presumes that every person is mentally sound until proved otherwise.

A. Civil Responsibility

1. Competence as a witness

(1) Generally speaking a mentally ill cannot testify

(2) However he can testify under following special circumstances:

(i) **Understanding not affected** - if he can understand the questions put to him and give rational answers to them [S.118 IEA]

(ii) During **lucid interval**

(iii) Understands obligations of an **oath**

(iv) Understands the **necessity of telling the truth**

(v) if able to tell coherently what he has seen.

2. Consent

(1) **Consent to any act mentioned in IPC** [eg hurt, medical examination, sexual intercourse, surgery etc] - Not valid, because the mentally ill does not understand the nature and consequences of the act [**S.90, IPC** - please also see **ch 2**].

(2) **MTP** – consent invalid [**ch 26**].

3. Contracts

(1) Only a person of sound mind is competent to contract [***S.11*** Indian Contract Act, 1872].

(2) **When is a person said to have sound mind:**

(i) If at the time when he makes the contract, he is capable of understanding it and of forming a rational judgment as to its effect upon his interests [***S.12*** Indian Contract Act, 1872].

(ii) If other party can show it did not know the mental condition of the other party, and the contract was fair " Contract may be held valid

(3) **If mental illness develops after contract is signed** – Contract is valid, until and unless mental illness makes the other party unable to perform service relevant to contract.

(4) Contract can be made during lucid interval [***S.12*** Indian Contract Act, 1872].

(5) A sane man, who is delirious from fever or who is so drunk that he cannot understand the terms of a contract or form a rational judgment as to its effect on his interests, cannot contract whilst such delirium or drunkenness lasts

[***S.12*** Indian Contract Act, 1872].

4. Government service

A mentally ill person cannot enter a govt service.

5. Inheritance

Mentally ill person cannot be disqualified from inheritance to any property [**S.28**, Hindu Succession Act 1956]. However **S.25** of HAS 1956 disqualifies a murderer [or an abettor of a murder] from inheritance. The situation is less clear if a mentally ill person commits murder.

6. Management of property and affairs of mentally ill

This is guided by **chapter VI** of the MHA [**S.50-S.77**]

(1) **Application** [**S.50**]:

(i) If a person has become incapable of managing his property due to mental unsoundness, an application for holding an enquiry into his mental condition [judicial inquisition] may be made to the District Court [DC] by (a) any of his relatives [not friend or unrelated person].

(ii) Alleged mentally ill person is personally examined either by the presiding officer of the court. Court may call any other person [psychiatrist] for opinion and report.

(2) **Findings given after inquisition** [**S.51**]:

(i) Whether the person is indeed mentally ill

(ii) if yes, can he take care of (a) himself (b) his property (c) neither. This fact must be clearly stated by the doctor in his certificate.

(3) **Appointment of guardian** – if person incapable of taking care of himself, a guardian is appointed [**S.53**].

(4) **Appointment of manager by Court**– if person is found incapable of taking care of his property, a manager is appointed to take care of his property [**S.54**].

(5) **Appointment and remuneration of guardians** [**S.57 MHA 1987**]: Legal heirs of mentally ill person cannot ordinarily be appointed guardians or managers.

(6) **Duties of guardian or manager** [**S.58**]:

(i) To take care of mentally ill persons and those persons dependent on him

(ii) To take care of his property.

(7) **Powers of manager** [**S.59**]:

(i) Has all powers in relation to property, as the mentally ill person would have had, if he was not ill

(ii) Sale, mortgage, lease of property for purposes of settlement of debts - Manager can pay all debts and liabilities of the mentally ill persons by mortgaging or selling any of the immoveable property of the mentally ill, after making an application to the DC and taking permission from it.

(8) **Admission to a psychiatric hospital** – If District court thinks that the condition of mentally ill person is such that he must be admitted in a psychiatric hospital, an order is made to that effect. The detention would be till such period the order is revoked [**S.26**].

(9) **Cessation of mental illness** [**S.75**]:

(i) If DC is informed that the person who was found to be mentally ill, is now free from such illness, it may direct a subordinate court to inquire into the matter.

(ii) A second inquiry is held following the same procedures. If person found to the mentally sound, all actions taken regarding him would be set aside [eg removal of guardians and managers etc].

7. Marriage

Condition (ii) of a valid Hindu marriage states that neither party must be mentally ill at the time of marriage. If any party is mentally ill, the marriage is voidable [for details, please see **ch 23**].

8. Partnerships

Mental illness of a partner does not automatically dissolve the partnership until court is moved for its dissolution.

9. Testamentary capacity

Testamentary capacity is a person's legal and mental ability to make or alter a valid will.

Salient features:

(1) **Definition of a will:** (i) Any testamentary document [**S.31, IPC**] (ii) legal declaration of the intention of a testator with respect to his property which he desires to be carried into effect after his death [**S.2(h)** Indian Succession Act,

1925]

(2) Person executing a will is a **testator** [male] or **testatrix** [female].

(3) **Holographic will** - It is a will that has been entirely handwritten and signed by the testator. Normally, a will must be signed by witnesses attesting to the validity of the testator's signature and intent, but holographic wills that have not been witnessed are treated equal to witnessed wills. They need not be signed or attested [**S.66(2) (a)** Indian Succession Act, 1925]

(4) **Requirements of a valid will:** (i) It must be properly written or typed and properly witnessed and signed by testator as well as at least 2 witnesses. (ii) Age - Testator must be major [>18 years] (iii) Sound mind - He must be of sound mind at the time of making the will [compos mentis]. A person is said to possess a **sound mind** if he has (a) a capacity of recollecting memories and recognizing his relatives and friends, (b) understanding their relations to him and (c) judging his obligations to all. (iv) Testator should not be under (a) undue force and (b) undue influence (v) There should be no dishonest representation of facts

(5) **Physical examination** – (a) Specifically exclude influence of drug or drink, excruciating pain etc (b) Conditions which do not disqualify (I) agraphia [failure to communicate through writing] (II) alexia [failure to understand by reading] (III) blindness, deafness, dumbness if person can communicate properly through signs or writing (IV) diseases not affecting mind (V) eccentricity [having unusual likes, dislikes and habits] (VI) Extreme age (VII) feeble health (VIII) motor or sensory aphasia (IX) physical infirmity

(6) **Sound mind** – Doctor can certify soundness of mind after (i) history (ii) Whether well-oriented in time, place and person (iii) psychiatric and psychological examination including **intelligence testing.**

In particular determine and ask (a) questions about his family, relatives, friends, business and social partners, their number, his degree of contact with them, whether he can recognize them through pictures, his opinions about all of them. (b) Does he appear to have delusions about anyone? (c) Extent, nature and value of his properties, and manner of its distribution desired by him (d) In an unjust or unusual distribution is being made, find out if it is intentional and if there are any reasons for doing so (e) Can he repeat the main provisions of his will (f) Power of concentration – by asking simple arithmetic sums (g) To determine undue pressure or influence – Ask all persons to leave room and

question him in isolation (h) Presence of any mental strain. (i) Impairment of memory (iv) Lab investigations.

(7) **Delirium and will** – please see MLI of delirium above.

(8) **Delusions and will** – please see MLI of delusions above.

(9) **Drunkenness and will** – invalid if he did not understand what he was doing

(10) **Lucid interval and will** – Will is valid made during lucid interval

(11) **Suicide after making a will** – Will is valid, if suicide was not as a result of mental disorder.

(12) **Wills *in extremis*** – Wills made by a dying person. Valid [cf **ch 1** " dying declaration].

(13) **Nuncupative Will** - A verbal will; a "deathbed" will. Valid, but shall be null at the expiration of one month after the testator, being still alive [**S.66**2. **(h)** Indian Succession Act, 1925]

(14) **Video taped (filmed) will** – Indian law is silent on this. However such video wills are seen as a good supplement to a valid will as they may serve to demonstrate mental competence.

B. Criminal Responsibility

[A] A person may plead mental illness to avoid (1)Inquiry [**S.328, CrPC**] (2) Trial [**S.329, CrPC**]. (3) Conviction [**S.84, IPC**] (4) Capital punishment. None of these can proceed in the presence of mental illness. [B] **Legal tests of insanity** – Insanity [or more correctly mental illness] has different medical and legal definitions. Several tests have been adopted over the years to judge the criminal responsibility of the mentally ill.

1. McNaghten rule (1843)

McNaghten [M'Naghten] rule - An accused person is not legally responsible for his crime if it is clearly proved that (i) at the time of committing the crime, he was suffering from such a defect of reason from disease of mind (ii) that he did not know the nature and quality of the act he was doing (iii) or that what he was doing was wrong.

Also known as **'right or wrong test'.**

(1) **Facts of the case - Daniel M'Naghten** (1813 – 1865), a Scottish woodworker was suffering from paranoid schizophrenia [his chief symptoms - delusions and hallucinations]. He believed that spies sent by Tory party – the

party in power in Britain at that time – were following him, and hatching a conspiracy against him (delusions of persecution), and accusing him of several crimes (auditory hallucinations). On **20 Jan, 1843,** he decided to kill Tory prime minister, Robert Peel, but by mistake shot his private secretary **Edward Drummond**. He died 5 days later [Other imp forensic case involving British Prime Minister - **ch 42**].

(2) **Trial and Judgment** – M'Naghten was found not guilty on grounds of insanity. Not sent to jail, but to Bethlem Mental Hospital for life.

(3) **Reasoning -** There was agreement on both sides that M'Naghten suffered from delusions of persecution. Ten physicians [1 for prosecution and 9 for defense] found him mentally ill. M'Naghten's delusions had led to a breakdown of moral sense and loss of self-control, which had left him in a state where he was no longer a "reasonable and responsible being". He could not differentiate right from wrong.

(4) **Significance and Outcome** – The verdict led to an unprecedented outcry in parliament, press and public. **Queen Victoria** (1819–1901), who had herself been the target of assassination attempts, wrote to the prime minister expressing her concern at the verdict and the House of Lords revived an ancient right to put questions to judges. Lord Chancellor **Lord Lyndhurst** put before 12 judges, 5 hypothetical questions. The answers given to the House of Lords on **19 June 1843** formed the basis of M'Naghten rule. M'Naghten rule dominated the law on criminal responsibility in many countries throughout the British Commonwealth for over 100 years, till it was set aside by Durham rule. **In India, M'Naghten rule has been incorporated in S.84, IPC.**

(5) **Criticisms:** (i) It concentrates only on the cognitive aspect **[Fig 28.3]**. It does not take into consideration the volitional aspect or the issue of control [major criticism]. A mutilomaniac knows that what he is doing is wrong, but still cannot control himself from mutilating a man, because of lack of control. By M'Naghten rule, he is guilty. But actually should not be.

(6) **Examples** – The question to be asked is "if delusions were real, was the person entitled to kill"? If he was, he is exempt from punishment, if he was not, he is not exempt

(i) Exempt from punishment under following circumstances – Under an insane delusion, the accused thought that (a) Person in front of him is attempting to kill him (b) Person in front of him is a wild animal going to attack him (c)

He is the state executioner

(ii) Not exempt from punishment under following circumstances - Under an insane delusion, the accused thought that (a) person in front of him was causing his character assassination (b) Some person was having affair with his wife (c) Some person had stolen money from his house. Under all these conditions – even if they occurred actually – a person must complain to police and not take law in his own hands [MLI of delusions].

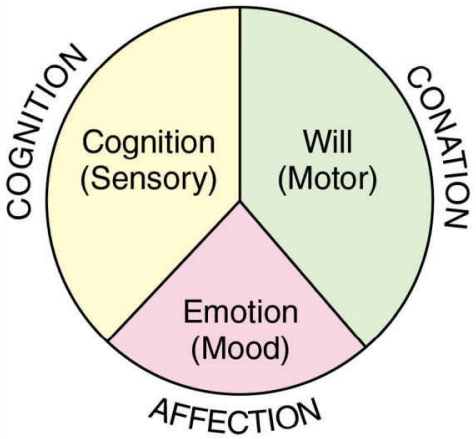


Fig. 28.3: Mind has three broad areas of functioning according to classical psychiatry – cognition (thoughts), conation (actions) and affection (feelings). In this figure they are depicted simplistically as cognition (sensory), will (motor) and emotion (mood) respectively. M'Naghten covered disorders of only one compartment – cognition. Irresistible impulse test covered the second compartment – will. Durham's rule, in trying to cover all compartments went overboard and had to be abandoned in 1972.

2. Irresistible impulse test (1887)

(1) First formulated by the Alabama Supreme Court in the 1887 case of Parsons v. State [Parsons v. State, 81 Ala. 577, 2 So. 854 (1887)].

(2) Attempted to answer the major criticism of M'Naghten rule, that it does not take into consideration the volitional aspect.

(3) It states – An accused person is not criminally responsible, even if he knows the nature and quality of his act and knows that it is wrong, if he is incapable of restraining himself from committing the act, because the free agency of his will has been destroyed by mental disease.

(4) Not followed in India.

(5) Acts done under irresistible impulses like dipsomania, kleptomania, mutilomania and pyromania are not punishable [please also see above under the heading "impulse"].

3. Durham's rule (1954)

Durham's rule [syn, 'pro***d***uct test'] states that an accused person is not criminally responsible, if his unlawful act is ***the pro*****d*****uct of mental d*****isease of mental d*****efect***. **[Memory Aid 6]** It was formulated in Durham v. U.S. 214 F.2d 862.

4. Currens rule (1961)

Currens rule states that an accused person is not criminally responsible if at the time of committing the act, the defendant, as a result of mental disease or defect, lacked substantial ***c*** apacity to ***c***onform his ***c***onduct to the requirements of the law which he is alleged to have violated. **[Memory Aid 7]** It was based on United States of America, v. Donald Kenneth Currens, Appellant, 290 F.2d 751 (3rd Cir. 1961).

5. American Law Institute (ALI) Test (1962)

American Law Institute ("ALI"), published its **Model Penal Code** in 1962. S4.01 of this Code forms the ALI test [**Table 6**].

Table 6: S.4.01 of MPC [ALI test]

Model Penal Code S.4.01 states that:

1. A person is not responsible for criminal conduct if at the time of such conduct as a result of mental disease or defect he lacks substantial capacity either to appreciate the criminality [wrongfulness] of his conduct *[cognitive insanity]* or to conform his conduct to the requirements of law *[volitional insanity]*.
2. The terms "mental disease or defect" do not include an abnormality manifested only by repeated criminal or otherwise anti-social conduct.

6. Other solutions

Above tests of insanity are "All or none" tests. Either a person is guilty or not

guilty. Other “midway” solutions have been suggested as below.

a. Doctrine of diminished responsibility

Doctrine of diminished responsibility states that a person is not fully responsible for his actions, but only partially, if by virtue of his unsoundness of mind, he did not premeditate, deliberate or formed a specific intent to kill.

Salient features:

(1) **Partial defense** - It is a partial defense available to the accused. In India, S.84 is an ‘all or none’ law, i.e. either is person is fully responsible or not responsible at all for his actions. Some countries, most notably Scotland, provide a middle path also – the doctrine of diminished responsibility – whereby he is neither fully responsible, nor acquitted. Instead he is charged for a lesser offence - ‘voluntary manslaughter’ [**ch 11**]. **Mental diseases** – This defense requires evidence of a state of mind bordering upon, but not amounting to complete insanity. (i) Mental diseases most commonly qualifying for this defense - Arrested development of mental faculties, depressions, injuries to head, obsessions, paranoia and psychopathic personalities (ii) Mental diseases or states which do not qualify for this defense - Bad temper, drug use, hatred, jealousy, low intelligence, mild character defects, political fanatism, poor judgment, racial murders and unfavorable personal feelings

(2) **Countries:** England - **S.2** of **Homicide Act 1957** provides this defense.

b. Guilty but mentally ill [GBMI]

If the court returns a verdict of GBMI, it must impose sentence, but it can only be executed after the defendant has been given psychiatric treatment and discharged. He then serves the remainder of the sentence.

VI. MEDICOLEGAL ASPECTS OF MENTAL UNSOUNDNESS

A. Inquiry and Trial of Mentally Unsound Persons

CrPC

(1) **S.328, CrPC** - Inquiry being held against a person " He claims unsoundness

of mind " Magistrate will inquire into such soundness " He is found to be mentally unsound " Inquiry will be postponed and person released. Because he is incapable of making his defense.

(2) **S.329, CrPC** - Trial going on against a person " He is found to be mentally unsound " Trial will be postponed and person released. Reason same as above.

(3) **S.330, CrPC** - Person undergoing inquiry or trial" found to be mentally unsound " He shall be released on bail. Sufficient security being given by relatives or friends that he shall be properly looked after, and not cause injury to himself or others. If bail cannot be given, he will be detained in safe custody of a proper place [name of place is not mentioned, but S.336, CrPC implies, it could be jail also]. If a person is declared mentally unsound u/s 328, 329 or 330 CrPC, and at any stage it is claimed that accused has become mentally sound, proceedings will be according to S.332, CrPC.

(4) **S.331, CrPC** - Person had earlier been released u/s 328 or 329, CrPC " becomes normal " Inquiry or trial will start.

(5) **S.332, CrPC:**

(i) Person undergoing inquiry or trial " Claims mental unsoundness " brought before magistrate" Magistrate finds him mentally sound " Trial will proceed

(ii) Person already found mentally unsound u/s 328, 329 or 330, CrPC " It is claimed he has become mentally unsound " He will be brought before magistrate " magistrate will decide whether accused is mentally fit or not.

(6) **S.335, CrPC** - Person committed a crime " given benefit of **S.84, IPC** [**NGRI**] " Court will order safe custody of such person in a proper place. [name of place is not mentioned, but **S.336, CrPC** implies, it could be jail also]. Such person can also be delivered to a relative or friend, if an undertaking is given that he will be prevented from doing injury to himself or any other person.

B. Mental Unsoundness and Murder

(1) A murderer may allege mental unsoundness in order to get benefit of **S.84, IPC**. If opinion is sought from doctor regarding mental unsoundness of a murderer, he must administer him a full battery of psychological and psychiatric tests.

(2) In addition he must make note of following facts

(i) **History** – of accused and his family regarding mental illness. Does it appear

to have arisen suddenly after murder, or were there documented visits to doctor earlier

(ii) **Accomplices** – not present

(iii) **Prearrangement, Preparation, Preplanning** – absent in mental illness

(iv) **Motive** – There should be no motive in mental illness

(v) **Victims** – may be multiple in mental illness. Some may be his own relatives and friends

(vi) **Conduct of accused at the time crime** – No secrecy involved

(vii) **Conduct of accused after crime** – mentally unsound person would not destroy pieces of evidence, and would notify even the police

(3) **Procedures regarding enquiry and trial** – guided by **S.328-339,** CrPC as above.

C. Automatism

Automatism [from Gk, automatismos, self-action] is mechanical, repetitive, undirected behavior that is not consciously controlled.

Salient features:

(1) Also called automatic behavior.

(2) **Classification** – May be classified into

(i) insane automatism [associated with psychiatric disease] and

(ii) sane automatism [not associated with psychiatric disease].

(3) **Etiology:**

(i) Cerebral disease (ii) Epilepsy (iii) Hypoglycemia (iv) hysterical states (v) psychomotor epilepsy (vi) sleepwalking (vii) Toxic [drug automatism] - ***Drug automatism*** is repeated intake of a drug because of drug induced drowsiness and amnesia. (viii) Trauma [eg concussion, posttraumatic automatism]

(4) **Types of automatism:**

(i) Ambulatory automatism - aimless wandering or moving about [fugue, poriomania]

(ii) Command automatism – please see command hallucinations above.

(iii) Mimetic automatisms - consist of facial expressions [grimacing, smiling, pouting]

(5) **Medicolegal aspects** – Indian law does not have any special provision for

automatism. He can get benefit of **S.84, IPC**, if he can show he was not aware of the act.

D. Somnambulism

Somnambulism [sleepwalking], is a sleep disorder in which the sufferer performs activities that are usually done during a state of full consciousness.

Salient features:

(1) Activities during somnambulism may be as benign as sitting up in bed, walking to the bathroom, and cleaning, or as hazardous as cooking, driving, extremely violent gestures, grabbing at hallucinated objects, suicide or even homicide. Activities are similar to automatism. Even during hazardous activities, he rarely injures himself

(2) Somnambulism belongs to the **parasomnia** family of disorders, which involves a number of abnormal and unnatural behaviors during sleep eg sleep terrors, bruxism (teeth grinding) and restless legs syndrome

(3) **Etiology** – acute anxiety, stress, concealed mental conflict

(4) Sleepwalkers arise from the **slow wave sleep (SWS) stage** [Stage 3 sleep] in a state of low consciousness and perform activities as if they are awake.

(5) **Psychological profile** – such patients are usually

(i) Socially well behaved

(ii) Unaggressive

(iii) Well adjusted in life.

E. Somnolentia

Somnolentia, semisomnolence or ***sleep drunkenness*** a condition of prolonged transition from sleep to waking, with partial alertness, disorientation, drowsiness, poor coordination, and sometimes excited or violent behavior.

Salient features:

(1) Patient may commit a crime, when suddenly aroused during sleep, especially when he was dreaming

(2) Not criminally responsible.

F. Miscellaneous

1. Hypnotism

Hypnosis is a trance state characterized by extreme suggestibility, relaxation and heightened imagination. Also known as **mesmerism**, because first suggested by German physician **Franz Anton Mesmer** [1734 –1815]. **MLI:** (i) Crime [murder etc] under hypnosis is possible, but the perpetrator cannot take the defense that he was under hypnosis. No one can be hypnotized against his will, and it is presumed that when a subject consented for being hypnotized, he knew of all consequences.

2. Kidnapping from lawful guardianship

Taking away a person of unsound mind away from a guardian without his consent is punishable u/s **S.361, IPC [ch 3]**.

3. Self-inflicted injuries

Mentally ill persons may inflict injuries on their bodies [ch 12]. They must be restrained, otherwise doctors and paramedical staff may be sued for negligence.

29. Blood and Biological Stains

I. BLOOD

A. Examination of Suspected Blood Stains

1. Whether the given stain is blood or not?

a. Presumptive tests

A ***presumptive test*** has a high sensitivity but low specificity.

Salient features:

(1) If a presumptive test is -ve, one is almost sure blood is absent [because of high sensitivity. Blood can be detected in samples with 10^5-fold dilutions or more. But if the test is +ve, confirmatory tests need to be done [because of low specificity].

(2) **Principle:** (i) Heme group of Hb has a peroxidase like activity, i.e. it can split off a nascent oxygen from an oxidant [usually 3% H_2O_2]. $H_2O_2 \xrightarrow{\textit{peroxidase}}$

H_2O+[O] (ii) The nascent oxygen can either (a) change the color of a chromogen or (b) cause a substance to emit light. Based on this, presumptive tests are divided into two – [A] color tests [B] Chemiluminescent and Fluorescent tests. Most frequently used reagents are **phenolphthalein** and **luminol** tests.

i. Color tests

(1) **False +ve** – Color reaction with a substance other than blood.

(i) Chemical oxidants

(ii) Plant materials – eg horseradish contain peroxidases"false +ve results.

(iii) Materials of animal origin [including human], which are not blood but may contain contaminating traces of blood.

(2) **False -ve** – No color reaction with blood.

(i) Extremely low blood dilutions - tetramethylbenzidine [1:400,000]; o-tolidine and leucomalachite green [1:200,000]; phenolphthalein [1:2000]. Washing, rain, heat and time may reduce the conc of blood in the sample.

(ii) Contamination with reducing agents – (a) several detergents and foods (b) ascorbic acid. Lesser contaminants are required in lower blood dilutions [to elicit false -ve]; thus both factors present together elicit false –ve easily.

(3) **Chromogens** – Over 20 have been used. 6 common ones are discussed below.

(a) Benzidine [Adler test]

(1) **Historical** - Originally described by **Adler** in 1904

(2) **Procedure:**

(i) Take stained material"cut out a small piece of stain or tease out some fibers containing stain"Place them on a porcelain tile"Add a drop of saturated sol^n of benzidine in glacial acetic acid"A dark blue color indicates a chemical oxidant and the test should be considered inconclusive. If there is no color change"Add a drop of 3% H_2O_2"If blood is present, a **dark blue** color is produced immediately"may eventually turn to brown.

(ii) Alternatively swab the suspected blood stain with clean filter paper or a swab, which may be moistened if necessary with deionized water, ethanol or saline and proceed as above.

(3) **+ve reaction** – given by even (a) very old blood stains, (b) stains that have been exposed to heat or cold, (c) stains that have been treated with cleaning

agents (d) Very dilute blood stains [up to a dilution of 1:300,000]

(4) **Weak reaction given by** – bacteria, formalin, milk, oxidizing agents, pus, rust, saliva and certain vegetable and animal juices

(5) **Current status** - Was once considered one of the best presumptive tests, but because of proven carcinogenicity of benzidine, was replaced by o-tolidine test.

(b) Phenolphthalein [Kastle-Meyer test]

(1) First described by **Kastle** and **Shedd** in **1901**, for the detection of plant peroxidases. In **1903**, **Meyer** used it for the determination of peroxidase in blood and pus.

(2) **Procedure** – Make stain extract with distilled water"add 20 drops of phenolphthalein reagent [phenolphthalein 2 g + NaOH 20 g + Zn + Distilled H_2O 100 ml]"add 2 drops of H_2O_2"If blood present, a **characteristic pink** color develops immediately.

(3) **Comparison with benzidine** – Specificity more than that of benzidine, but sensitivity is lesser.

(4) **Disadvantages** -

(i) Amount of recoverable DNA - is reduced when this test is used.

(ii) False +ve – with traces of Cu. They are not really considered false +ve in true sense, because the color produced is not the color produced is not the characteristic pink, but some other color.

(c) O-tolidine test [Kohn test]

(1) Reaction is similar to that of benzidine and is conducted under acidic conditions

(2) Produces **blue color** resembling benzidine

(3) Not to be confused with **o-tol**

u

idine test [please note extra “u”] which is a test for chloride ions in water (as in swimming pool water) and not for blood.

Memory Aid 1: O-toluidine test

Under **W**ater - Test with an “u” [tol **U**idine] is used for **W**ater chloride.

(d) Leucomalachite green [LMG]

(1) Also referred to as **McPhail’s Reagent** [Hemident]

(2) **Procedure** -

(i) Take stained material as in benzidine test.
(ii) Apply 1-2 drops of the LMG reagent.
(iii) Other steps are similar as in benzidine test
(iv) An immediate **blue-green** color change indicates blood.
(3) **Disadvantages** -
(i) Use of LMG obstructs DNA recovery from stain.
(ii) Sensitivity is 10 times less than that of Luminol.

(e) Tetramethylbenzidine [TMB]

All steps are same as in LMG test, except that the reagent used is **TMB**. Color change is **blue-green**.

(f) Guaiacum test

(1) **Reagent used** - **α**-guaiaconic acid, a phenolic compound, that is extracted from the wood resin of **Guaiacum trees**, which grow in subtropical and tropical regions of the Americas.
(2) All steps are same as in LMG test, except that the reagent used is **Guaiacum**. Color change is **deep blue**.

Memory Aid 2: Presumptive tests for blood

1. **B**enzidine is **B**lue
2. **P**henolphthalein is **P**ink
3. **L**eucomalachite is **b**lue **G**reen because **L**eaves are **b**old **G**reen
4. All other colors are **blue or blue green**.
5. **G**uaiacum is not **g**reen. Thus it has to be blue.
6. **B**lue **G**reen is in TM**B** and LM**G**

ii. Chemiluminescent and fluorescent tests

Salient features:

(1) **Luminol** is the luminescent dye and **Fluorescin**, the fluorescent dye that is used for determination of blood.
(2) **Done in dark room** - Blood stains which are normally invisible to eye shine in the dark.
(3) **Advantages** –
(i) They can be sprayed over large suspect areas
(ii) Patterns eg bloody footprints, fingerprints, splatters etc can be made out
(iii) Can be sprayed in inaccessible nooks and corners and crevices of a room, or

in a vehicle etc to know where exactly stains are present

(iv) Can determine blood at crime scenes, even if they are thoroughly cleaned and washed.

(4) **Disadvantages** - if sample is very little, the spraying of these reagents causes dilution. This may cause problems later if DNA profiling has to be done.

(a) Luminol test

(1) Luminol gets oxidized in presence of Hb and H_2O_2"Luminesces with a blue-white light.

(2) **Advantages** - (i) Considered the best overall presumptive blood test because it has the greatest sensitivity and specificity of all. Sensitivity is 20 times more than other tests – Greatest advantage. Hb has been detected in 1:10 million dilutions. Bloodstains have been detected under 8 layers of paint. (ii) does not destroy the DNA, (iii) Can be reapplied (iv) easy to use, (v) non-toxic, (vi) Detection up to 7 wks [minimum] (vii) most resistant of all presumptive tests to the possible influence of contaminants [eg detergents etc].

(3) **Drawbacks** -

(i) Operators must work in dark, making manipulations difficult.

(ii) Identification relies on one's eyes becoming accustomed to the dark.

(iii) The reaction is short-lived [30 s].

(iv) Cannot differentiate between human and animal blood.

(v) Not specific for hemoglobin

(vi) Interferes with later determination of enzyme and protein genetic marker systems [especially Erythrocyte Acid Phosphatase (EAP), Esterase D (EsD), Peptidase A (PEP A) and adenylate kinase [AK] (vii) luminol is suspected to be a mutagen.

(b) Fluorescein test

(1) Test first suggested in 1910 by **Fleig**.

(2) **Fluorescin** is the reduced form of fluoresc**e**in. Fluorescin is the prepared reagent for analysis; on oxidation by heme it forms Fluoresc**e**in – which fluoresces [Fluorescin/Fluorescein system]. Fluorescin sprayed stains are exposed to light in the range of **425-485 nm** [usually] using an alternate source device [this is not necessary with luminol, which works on chemiluminescence].

b. Confirmatory tests

Hb molecules are detected. An average RBC contains about 280 million molecules of Hb. An ideal test [which does not exist] must be able to detect a single molecule.

i. Microcrystal assays

(1) In microcrystal assays, blood crusts from a bloodstain are treated chemically to convert heme into heme derivatives, which form crystals with distinctive morphologies.

(2) The crystal morphology is examined with a microscope

(3) **Drawback** –Although specificity is high, sensitivity is very low.

(a) Hemin [Teichmann] crystal test

Described in **1853** by **Ludwig Teichmann** of Poland.

(1) **Procedure** –

(i) Place suspected stain or material on a glass slide

(ii) Add a small crystal of NaCl and glacial acetic acid [2-3 drops]

(iii) Cover with a cover slip

(iv) Apply little heat with a flame placed below the slide

(v) Cool and examine under microscope

(vi) If blood is present, faint yellowish-red to brownish-black rhombic crystals of hemin [Ferriprotoporphyrin chloride (syn, hematin chloride)] are seen. They are arranged singly or in clusters.

(vii) Confirmation of the nature of crystals - add a drop of H_2O_2 under the cover slip"bubbles of gas are seen.

(2) **False negative** –

(i) Old stain

(ii) Fabric washed or treated with chemicals

(iii) Defective chemicals used [moisture in acid]

(iv) Wrong application of test [addition of too much NaCl, overheating].

(3) **False positive** - None.

(b) Hemochromogen [Takayama] crystal test

Described in **1912** by **Masaeo Takayama** of Japan.

(1) **Procedure** -

(i) Place suspected stain or material on a glass slide

(ii) Add 2-3 drops of Takayama reagent [Water: Saturated glucose sol^{n}: NaOH (10%): pyridine = 2:1:1:1]

(iii) Cover with a cover slip

(iv) If blood is present, pink feathery crystals of hemochromogen [Pyridine ferroprotoporphyrin (syn, reduced alkaline hematin)] arranged in clusters or sheaves appear within 5 min [**Fig 29.1**]. However to be sure that the suspected stain is not blood, one must wait for 30 min. If crystals do not form up to this period, the test is declared negative

(v) Slide warming [up to 37°C in an incubator, especially in cold weather] hastens the reaction, but is not necessary as in Teichmann test.

(2) **Advantages** -

(i) He**mo** chromogen crystal test is **mo**re reliable [with **memory aid 3**].

(ii) Gives good results even with old stains

(iii) Heating is not necessary.

(c) Wagenaar test

Also called acetone-chlor-hemin test. Developed in **1935**. Suspected bloodstain + Few drops of acetone + one drop of dilute HCl. If hemoglobin is present, characteristic crystals form, which are observed under a microscope.

ii. Microscopic examination

(1) **Presence of RBCs** etc -

(i) Suspected stain extracted with Vibert's fluid [or any of the solvents mentioned above]" Examined under microscope"presence of cellular materials [RBC, WBC and platelets] confirms blood. In a dried stain, all cellular material becomes unrecognizable [cells are broken down]. In such a case, other confirmatory tests are employed

(ii) Intact RBCs seen only in (a) fresh wet stains (b) in blood clots

(2) **Identification:**

(i) Mammals – biconcave, circular, non-nucleated. Exceptions - camel and yak – RBCs are biconvex, elliptical, non-nucleated.'

(ii) Mammals showing nucleated RBCs [nRBCs] - Small numbers of nRBCs are seen in (a) alpacas [0-3/100] and (b) some breeds of dogs [Miniature Schnauzers and Dachshunds] (c) Humans (I) Newborns (II) in certain diseases and poisonings [lead poisoning (plumbism); Bone marrow

neoplasia, Pernicious anemia]

(iii) Other animals – in amphibia, birds, fish, reptiles – biconvex, oval, nucleated.

iii. Spectroscopic Examination

(1) It is essentially **absorption spectroscopy** [for principle, please see **ch 31**].

(2) **Most delicate and reliable test** for detecting the presence of blood in both recent and old stains

(3) **Quantity required** is <0.1 mg

2. If the stain is blood, whether it belongs to a human or not (species origin)?

Currently done by (1) **immunological,** (2) **enzymological or** (3) **DNA tests**.

Immunological

Principle [of immunological tests]

(1) Human serum contains proteins in colloidal suspension

(2) If it is injected in animals, they would be immunized against these proteins and produce antibodies [precipitins]. The serum is called antihuman precipitin serum

(3) If human serum is brought in contact with the serum of immunized animal [diluted up to 1:1000], the two would react forming a **visible precipitate** [within a minute].

Table 1: Characteristic blood proteins with functions and genes coding for them

S.No	Body fluid	Gene Characteristic of fluid	Characteristic Protein	function
	blood	SPTB	β-spectrin	**β-Spectrin** is part of erythrocyte membrane skeleton, **spectrin**
		PBGD	porphobilinogen deaminase	**Porphobilinogen deaminase** is the **3rd enzyme** of the heme biosynthesis pathway [Pl see **ch 36** " Fig showing **MOA of Pb**]

Table 2: Possible and impossible blood phenotypes of children if phenotypes of parents are known

S.No	Phenotypes of parents	Phenotypes of children	
		Possible	Impossible
1.	O×O	O	A, B, AB
2.	O×A	O, A	B, AB
3.	O×B	O, B	A, AB
4.	O×AB	A, B	O, AB
5.	A×A	O, A	B, AB
6.	A×B	O, A, B, AB	None
7.	A×AB	A, B, AB	O
8.	B×B	O, B	A, AB
9.	B×AB	A, B, AB	O
10.	AB×AB	A, B, AB	O
11.	M×M	M	N, MN
12.	M×MN	M, MN	N
13.	M×N	MN	M, N
14.	MN×MN	M, N, MN	None
15.	MN×N	N, MN	M
16.	N×N	N	M, MN

3. Human leukocyte antigen (white cell antigens, HLA typing)

Human Leucocyte Antigens are a group of highly polymorphic glycoproteins

found on the surface of leucocytes, which can be used for paternity testing because of their high degree of polymorphism.

Salient features:

(1) **Dominant period** - HLA typing started in 1970, and was predominantly used until 1985, when it was superseded by DNA profiling

(2) **Exclusion of paternity** – can be done up to 80% just by HLA alone [**Table 3**].

Table 3: Expected chance of excluding a man wrongly accused of paternity by means of blood groups and HLA

System used to exclude	Expected %age exclusion rate based on individual system	Expected %age exclusion rate combined with all previous systems
Red cell antigens		
ABO	17.6%	17.6%
MNSs	32.1%	44.1%
Rh	28.0%	59.8%
Kell	3.3%	61.1%
Duffy	4.8%	63.0%
Lutheran	3.3%	64.2%
Kidd	4.5%	65.8%
Serum Proteins		
Haptoglobins (Hp)	18.1%	72.0%
Gc	15.9%	76.5%
Ag	14.2%	79.8%
Gm1 Gm2	7.8%	81.4%
Km1	4.1%	82.2%
Red Cell Enzymes		
Phosphoglucomutase (PGM)	24.5%	86.6%
Erythrocyte Acid Phosphatase (EAP)	21.0%	89.4%
Glutamate Pyruvate Transaminase (GPT)	18.7%	91.4%
Esterase D (EsD)	9.0%	92.1%
Adenosine deaminase (ADA)	4.5%	92.5%
Adenylate Kinase (AK)	4.2%	92.8%
Glyoxalase 1(GLO)	18.0%	94.1%
HLA system		
HLA	80%	97%

4. Serum protein polymorphism

Human serum contains many proteins, many of which show polymorphism. In 1950s, it was discovered that these polymorphisms could be used for individualization.

a. Serum haptoglobins

(1) **Serum Haptoglobin** (Hp) was the most widely used polymorphic serum protein [before the advent of DNA].

(2) **Function** - To bind and transport Hb from the blood stream to the liver for the **recycling of the iron** contained in the Hb.

(3) **Genetics** - Two alleles are known – **Hp1** and **Hp2**. They combine to form three genotypes **Hp1-1**, **Hp2-2** and **Hp1-2** (or **Hp2-1**, which is identical). The resulting phenotypes are **Hp1**, **Hp2** and **Hp1-2** respectively.

(4) **MLI** - Hp is a relatively stable protein in dried blood stains. This makes it very valuable for forensic work.

b. Group specific component (Gc)

(1) Gc is a **vitamin D binding glycoprotein** in the **α**-2 globulin fraction of serum proteins.

(2) With **electrophoresis**, 3 different forms can be identified (Gc 1-1, Gc 2-2 and Gc 2-1), but with **isoelectric focusing** (a more refined technique), 9 Gc subtypes can be distinguished.

c. Ag Groups

(1) The Ag groups were discovered in **1961** in the serum of a multitransfused patient.

(2) Family studies showed that the antigen was inherited in an **autosomal dominant fashion**.

(3) Later, the serum protein was identified as **LDL** (low-density lipoprotein).

(4) **Genetics** - Two alleles are present"Ag(x) and Ag(y). The possible phenotypes are Ag(x+y+), Ag(x+y-), Ag(x-y+) and Ag(x-y-).

d. Gm and Km systems

(1) ***Immunoglobulins*** are highly polymorphic and account for approximately 15% of the serum proteins.

(2) Two immunoglobulins which were widely used in forensic work were **Gm**

and **Km**.

Location -

(i) **Gm** resides on the **heavy chain** (**G**amma) of immunoglobulin G (IgG)

(ii) **Km** resides on the light chain (**K**appa) of all immunoglobulins [IgG, IgA, IgM, IgE, IgD]

(3) **Inheritance** - Both **Gm** and **Km** allotypes are inherited independently of each other as autosomal, co-dominant alleles.

(4) **Advantages** -

(i) Stability - The antigens are stable at moderate heat, and may be stored at room temperature for extended periods and frozen for years

(ii) Variety of types - Especially with Gm, which has more than **20 allotypes**; Km allotypes are only **3**

(iii) They exhibit a high concentration in dried blood.

5. Red cell enzyme polymorphism

Several red cell enzymes show polymorphism. (1) **Phosphoglucomutase** (PGM) has been most commonly used in forensic work. Others that have been used for paternity are (2) **Acid Phosphatase** [AP] (3) **Adenosine deaminase** [ADA] (4) **Adenylate kinase** [AK] (5) **Carbonic Anhydrase** [CA II] (6) **Erythrocyte Acid Phosphatase** [EAP] (7) **Esterase D** [ESD] (8) **Glucose-6-Phosphate Dehydrogenase** [G6PD] (9) **Glyoxalase** [GLO 1] and (10) **Peptidase A**.

6. Hemoglobin (Hb) variants

(1) **More than 200 Hb variants** have been identified.

(2) **Commonest** is **Hb A**.

(3) Some people have both Hb A and another variant **Hb S** or **Hb C**.

(4) **Hb F** -

(i) It is **fetal Hb** which appears in fetal blood after **3 months** of gestation.

(ii) It disappears after birth within **6 months**.

(5) The different alleles are expressed co-dominantly, and it is possible to observe the heterozygous **Hb AS**, **Hb AC**, **Hb AF** types.

(6) **Hb S** is associated with **sickle cell trait**.

(7) Hb variants can easily be determined by electrophoresis or Isoelectric focusing (IEF).

B. MLI of Blood Groups

1. Cause of death

Incompatible blood transfusions.

2. Crime investigation

(1) Blood stains found at scene of crime, clothing and person of suspect can reveal identity of victim. Accused cannot allege it is his own blood because of different blood groups.

(2) Blood does not adhere readily to swiftly moving metallic objects. It is sometimes difficult to detect on a bullet which has passed through the body.

(3) Bloodstains on clothing may be due to crushing of bugs, fleas, louse and mosquitoes. Differentiating features - Small in size, sharply angular in outline, usually on the inside of garment, microscopy would reveal insect parts [eggs, hair, scales etc].

3. Disputed maternity

Disputed maternity arises

(1) when there has been an exchange of child at hospital [either intentionally or by accident]

(2) when two women claim the same child

(3) in case of kidnapped childzz

(4) suppositious child (please see ch 24 - Virginity, pregnancy and delivery).

4. Disputed paternity

(1) Question of disputed paternity can be solved by blood groups [**Table 3**]

(2) Bernstein's laws [please see above] are used

(3) Such questions arise under following circumstances

(i) When the child is born in lawful marriage, but the husband denies that he is the father

(ii) When a child is born out of lawful marriage, and the mother accuses a certain man of being the father of child and the man denies the accusation [Affiliation case, ch 24]

(iii) Suppositious child [ch 24]

(iv) Suits for nullity of marriage

(4) **Procedure** -

(i) Parents - 5 ml of venous blood is taken from mother and alleged [putative] father and placed in plain tube. Neither party should have had a blood transfusion within 3 months.

(ii) Infant – For taking blood, should not be <2 m; preferably >6 m. 1 ml blood is taken in a plain tube by venepuncture, or prick [ear or heel]

(iii) Testing – to avoid artifacts, testing of all subjects [mother, child, alleged father] should be done (a) by the same person, (b) in the same laboratory, (c) on the same day, (d) using the same batch of antisera and reagents.

5. Doping in sports

Packed cell transfusions are common doping methods in endurance sports. May be detected by different minor antigens eg Kidd (Jk).

6. Identification

Identification of a criminal from the blood stain found at scene of crime. Rarer a blood group is, higher are the chances of inclusion or exclusion of an alleged criminal.

7. Other biological stains

If the person is a “secretor”, blood group agglutinogens can be demonstrated in other biological stains too.

C. ML Aspects of Blood Stains

(1) **Infected blood spills** – in hospitals etc blood [and other body fluids] must be cleaned with sodium hypochlorite [NaOCl, 10,000 ppm chlorine]. Spread of infection through inability to control infection across patients makes the hospital liable for damages. In economically advanced countries sodium dichloroisocyanurate (NaDCC) may be used; however hypochlorite is more economical and easily available compared with NaDCC; hence it is used in India and other economically less developed countries. Please also see discussions under

(2) MLI of blood groups

(3) Examination of suspected blood stains

(4) Applications of DNA profiling.

D. DNA Fingerprinting (DNA Profiling)

DNA profiling [syn. DNA fingerprinting, DNA testing, DNA typing, genetic fingerprinting) is an identification technique based on the fact that each individual (except identical twins and clones) has a unique DNA. It was developed by British geneticist **Sir Alec Jeffreys** (1950-) in 1984 and published in 1985. The technique was made commercially available in 1987, when a chemical company, **Imperial Chemical Industries** (ICI), started a blood-testing centre in England. The **FBI** implemented this technique to forensic casework in **1988** after extensive validation studies.

a. Restriction fragment length polymorphism (RFLP) analysis

Restriction Fragment Length Polymorphism, or ***RFLP*** (commonly pronounced "rif-lip"), refers to multiple variations within different individuals [polymorphism] of DNA fragments [Restriction fragments] cleaved from their genome by restriction endonucleases. Because of DNA polymorphism, the length of DNA fragments obtained from different individuals is different. These DNA fragments are typically **500-30,000 bp in size** [**Table 4**]. In practice only bands between **4,000 [4kbp]** and **20,000 bp** have been used for analyses. Below the 4kbp level, the density of bands is such that reliable comparisons could not be made. Thus they belong to the minisatellite [VNTR] region of DNA [**Table 4**].

Salient features:

(1) This was the first technique used for forensic DNA analysis [by Alec Jeffreys]

(2) **Terminology** – VNTR and RFLP are sometimes used interchangeably and as synonyms. There is a minor difference though. **VNTR** refers to **V**ariable **N**umber of **T**andem **R**epeats at a given loci [**Table 4**]. **RFLP** refers to different lengths of DNA fragments that are produced as a result of these repeats. These fragments are cut on either side by a restriction endonuclease [giving rise to first two words "**R**estriction **F**ragment"]. Their lengths are different in different individuals [giving rise to last two words "**L**ength **P**olymorphism"]

(3) **Hypervariability** - VNTR loci are hypervariable, each showing 100 or more alleles [as determined by length polymorphism]. Polymorphism is so high that

typing of 5 to 8 loci [or markers] is sufficient to differentiate most individuals

(4) **Steps** - (i) extraction of DNA (ii) Digestion of DNA into fragments using a specific Restriction Endonuclease – Extracted DNA is incubated with an appropriate RE, which cleaves it into different sized fragments in different persons [**Fig 29.1**]. (iii) Electrophoretic separation of fragments based on size by agarose gel electrophoresis - (a) Fragments are placed into one end of a bed of **agarose gel** [1%] with electrodes in it. Agarose gel is a polysaccharide made from sea weeds. (b) An electric current is passed overnight through the gel, which separates fragments according to their sizes. (c) The negatively charged DNA fragments [-ve charge is due to the presence of phosphate groups] move through the gel, toward the positive electrode (anode). Smallest DNA move fastest and furthest down the gel and the largest DNAs move the least [**Fig 29.2**]. Fragments from 500 to 25,000 bp in length can be separated by this method. (iv) Denaturing the double-stranded DNA fragments in a high pH environment - Agarose gel is soaked in a weak alkaline solution. This denatures the DNA strands [Two strands of DNA separate out. It becomes single stranded]. (v) Transferring the single strands of DNA out of the gel onto a membrane support [either nitrocellulose or nylon] - This technique is known as “Southern blot”. Developed by the English molecular biologist **Edwin Southern** [1938 -] in **1975**. Somewhat akin to blotting up of ink by a blotting paper. **Plays on the name Southern blot** were later made to denote other techniques [they have nothing to do with directions]; **northern blot** is used for detection of RNA [developed in **1977** at Stanford University], **western blot** [syn protein immunoblot] to detect proteins and **eastern blot** to analyze protein post translational modifications (PTM) [chemical modification of a protein after its translation]. (vi) Hybridizing the immobilized DNA fragments with specifically labeled DNA probes - The bands are still invisible to the naked eye. A DNA probe is allowed to probe these bands. Excess probe is washed off. (vii) Detection of the hybrid products by autoradiography or chemiluminescence - The Nylon membrane is pressed against an X-ray plate for about **24 hours**. The bands appear on the X-ray plate as dark lines depending on where the probe had attached. The result is known as an ***autorad*** [short for **autorad**iograph]. These dark lines or bands are specific for each individual and called his DNA fingerprints.

(5) **Drawbacks** [of RFLP technology]: (i) **Degraded samples not useful** - As

VNTR sequences are long, nondegraded DNA samples are needed to make a profile. In degraded samples, VNTR sequences are broken. A typical allele used in RFLP is 500-30,000 bp in size [**Table 4**]. Thus fragments of DNA as large as 30,000 bp should be intact for successful RFLP. This is not possible in degraded DNA. This can best be conceptualized as imagining a large thread being broken into smaller pieces. The more pieces it is broken into, the lesser will be the average length of each piece. More degradation of DNA breaks it up into more number of pieces, and thus lesser length (bp) of each piece. (ii) Large amount of DNA needed (typically 50–100 ng). (iii) Work with radioactive substances is hazardous (iv) The procedure takes a minimum one week.

Table 4: Different types of satellite DNA

S.No.	Criteria	Microsatellite	Minisatellite (telomeric)	Minisatellite (hypervariable)	Satellite
1.	Other names	Short tandem repeats (**STR**s)	—	Variable Number Tandem Repeats (**VNTR**s)	—
2.	Size of repeat unit [in bp]	1 to 6	6 - Mainly "TTAGGG" in humans	7 to 100	100 to several thousand
3.	Total array size (or size of allele) [in bp]	10-500	10,000-15,000	500-30,000	100,000 to 100 million
4.	Location	Distributed almost ubiquitously throughout genome	All telomeres	Throughout genome, but most clustered in telomeric regions	Heterochromatin, especially centromeric heterochromatin
5.	Biological functions	Unknown, but may fine tune gene expression.	Protects chromosome ends; provides means of terminal sequence replication; possible roles in chromosome pairing and cell ageing	Very rare examples of expression, importance unknown. Otherwise unknown (probably none)	Unknown (probably none)

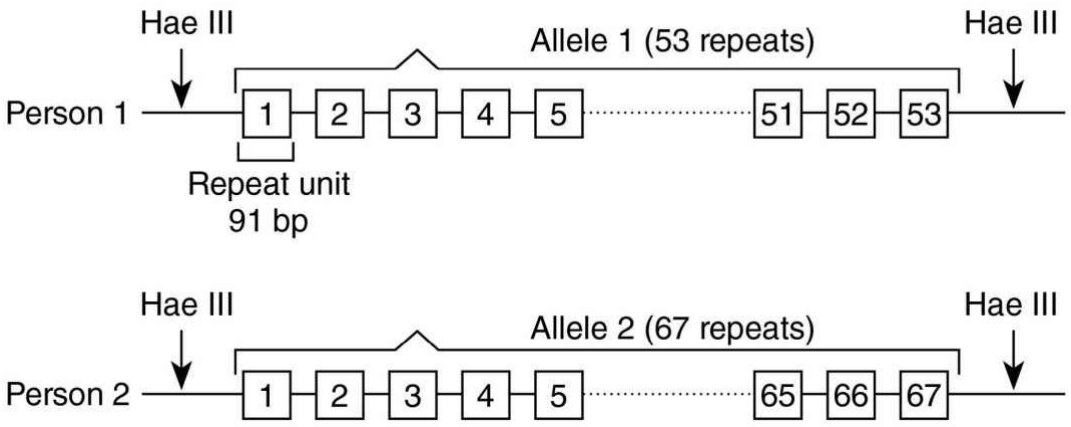


Fig. 29.1: Two persons showing VNTRs in their genome. The repeat unit is 91 bp in size. Person 1 shows 53 repeats of this region, while person 2 shows 67 repeats. The two persons are said to possess two different alleles [One having a size of 4823 bp (91×53) and another having a size of 6097 bp (91×67)]. In a population several different alleles [more than 100] may exist for a single locus. When a restriction endonuclease (Hae III) cuts the allele from both sides, the resulting fragments are different in size. They travel with different speeds in agarose gel and come to occupy different places.

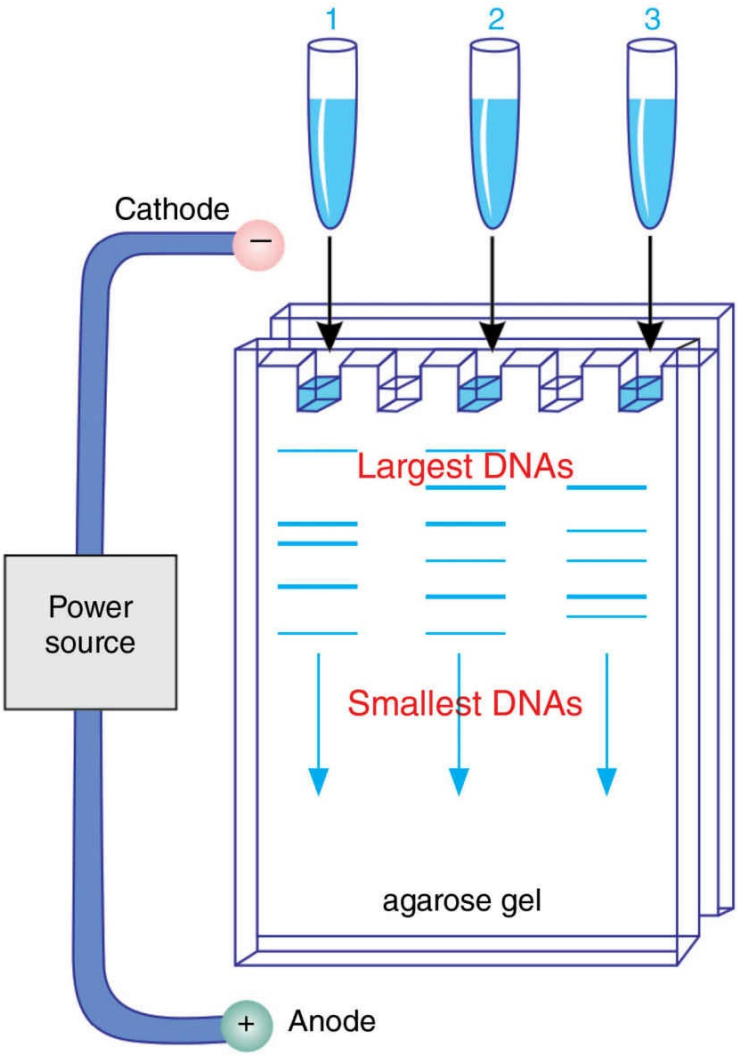


Fig. 29.2: Agar gel electrophoresis. The three tubes contain DNA fragments of different sizes from (1) Mother (2) Child (3) Alleged father

b. Short Tandem Repeat (STR) profiling [Using PCR]

Short Tandem Repeat profiling refers to preparing a profile of multiple variations within different individuals [polymorphism] of small DNA fragments less than 500 bp in size, belonging to microsatellite region [**S**hort **T**andem **R**epeats] of DNA [**Table 4**]. This technology came in 1990s, mainly to overcome the drawbacks of RFLP technology [please see above].

Salient features:

Advantages of STR analysis over RFLP – (i) The main advantage is the

smaller size of STR alleles [10-500 bp; **Table 4**].

c. Y-chromosome profiling

Y-chromosome profiling [YCP] refers to the study and analysis of forensically important loci on the Y-chromosome. Since Y-chromosomes are passed down from fathers to sons, it is essentially a study of the history of Y-chromosomes over time as they are passed from father to son to grandson to great-grandson, along an unbroken line of biological fathers and sons. The study of Y-chromosomes in many ways is similar to the study of **male surnames**, since both are passed from fathers to sons.

d. Mitochondrial DNA analysis

Mitochondrial DNA (mtDNA) is a 16,569 base pair (bp) circular strand of DNA located in mitochondria.

Salient features:

Inherited solely from the mother. **Forensic value of mtDNA** - arises from (a) sequence variability of the D-loop between individuals, (b) its efficient PCR amplification with limited biological material (c) In addition to the skin, blood, semen and saliva, from which nuclear DNA can be extracted, mtDNA can also be extracted from materials which fail to yield nuclear DNA, eg teeth, hair shafts, bone fragments and even human feces. (d) It is more robust than nuclear DNA for typing materials [due to its higher copy number per cell and its resistance to extreme environmental conditions. It is more resistant to putrefaction than nuclear DNA; thus useful in old materials. Also useful when the material is derived from dead cellular material such as hair or nail. Its circular nature confers protection from the action of exonucleases]. Ancient historical material can also by typed. Can be performed on fewer cells due to higher copy number. mtDNA can now be done from just ***1 sperm cell***. In contrast about **67 sperm cells** required for STR analysis using conventional nuclear DNA [please see above] (e) the haploid nature of mtDNA makes it easier to sequence than diploid nuclear DNA.

1. Biological samples for DNA profiling

Samples for DNA profiling must be collected using sterile uncontaminated clothes. Usual samples to be collected are:

(1) **Blood** - Bloodstains on carpets, cloth, floor tiles, food, furniture, metal, newspaper, plastic, vehicle, wallpaper, wood etc.
(2) **Fetus** – chorionic villus samples, muscle biopsy
(3) **Hair** – from head, moustache, beard, axilla, public regions.
(4) **Nasal mucus**
(5) **Saliva** - Cigarette butts, envelops, mouth swabs etc.
(6) **Semen** – suspected stains on cloth, floor tiles, fur, furniture, matted hair [generally pubic], paper, vaginal aspirate and swabs from anus, mouth, penis and vagina.
(7) **Tissue** - bone marrow, fingernail scrapings, muscle, spleen.

2. Applications of DNA profiling

a. Disputed paternity

Please see under MLI of Blood groups.

b. Extortion

(1) DNA profiling done from **saliva samples** [from envelops sent as extortion letters] can be matched with that of accused.
(2) Similar profilings done from the material on face masks, gloves, nasal secretions and saliva from cigarette butts.

c. Homicide

(1) **Blood –** DNA profiling done from blood [found on weapon or on clothes of accused] can be matched with that of victim.
(2) **Tissue** – found on bullet recovered from scene of crime [**ch 13**]
(3) **Hair roots** – Similar matching from hair on a weapon with that of victim.

d. Identification

(1) **Crime scenes** - Crucial DNA evidence may by left at crime scenes which may be matched to those to **potential suspects**. Besides usual biological materials eg semen, blood, urine and feces, unusual trace evidences that can lead to positive identification are a single human hair, lip cells left on a beer can and saliva on envelope flaps and cigarette butts etc.
(2) **Mutilated remains** as in accidents, bomb blasts, burnt bodies, mass

disasters, other catastrophes and putrefied bodies.

(i) DNA profiles from such remains may be compared with previous profiles.

(ii) DNA profiles of the deceased [during his life] may be prepared by taking cells of the deceased from toothbrush, combs etc which the deceased had been using.

(iii) If DNA profiles cannot be prepared from such material, profiles of close relatives such as parents, children, siblings etc might be taken, which will also be useful.

e. Sexual crimes

identification of accused from DNA profile of accused.

f. Other

(1) Authenticate consumables such as caviar and wine

(2) **Environmental forensics** - Detect bacteria and other organisms that may pollute air, water, soil, and food

(3) **Justice** - Exonerate persons wrongly accused of crimes

(4) **Wildlife forensics** - Identify endangered and protected species as an aid to wildlife officials (could be used for prosecuting poachers)

(5) **Immigration** - some visa applications may depend on proof of relatedness.

(6) **Organ donation** - Match organ donors with recipients in transplant programs

(7) **Pedigree tracing.**

3. Limitations

(1) **Twins** - DNA profiling of identical twins is same [fingerprints even in identical twins are different]

(2) **Expensive**; elaborate equipment. Thus facility available at limited places

(3) **Contamination** of samples can give wrong results

(4) Older DNA profiling technologies are more **time consuming** [RFLP].

(5) **Ethical** –

(i) Invasion of privacy - Holding a person's DNA profile on record is violation of that person's DNA 'ownership'.

(ii) Deletion of records - If a person has been proved innocent after DNA profiling, he has a right to demand removal of his DNA information from police data bases, but this is rarely done.

(iii) Illegal sharing of information - DNA information can be shared by police with others [eg medical researches, drug companies] without the individual's consent.

(6) **Sabotage** - DNA evidence is easily planted at a crime scene. [much more difficult or even impossible with fingerprints]

(7) **In blood and bone marrow transfusion** – DNA profile of donor will be generated and he may be indicted for a crime he did not commit.

4. DNA databases

A ***DNA database*** or DNA databank is a database of DNA.

Salient features:

(1) A DNA database is used much like a fingerprint database in identifying criminals, but can also be used in the analysis of genetic diseases, or genealogy.

(2) held electronically in the DNA database

(3) **DNA Databases around the world** -

(i) US – Has the largest DNA database in the world [**CODIS**]

(ii) UK - The United Kingdom National DNA Database [2nd largest].

(iii) Australia - The National Criminal Investigation DNA Database [NCIDD]

(iv) Canada - The National DNA Data Bank (NDDB)

(v) India – No similar DNA database till date.

(4) **Genomic sequences in DNA databases** - Full genomic sequence is not recorded - Only patterns of STRs. Different databases record different STRs. There is no unanimity.

(5) **Privacy issues** – Such databases pose ethical issues, eg threats to an individual's civil liberties. DNA databases may contain person's health-related data, including markers that identify various genetic diseases etc. This is regarded as an invasion to privacy.

Table 5: Differences between RFLP and STR analysis which uses PCR [Polymerase Chain Reaction]

S.No	Criteria	RFLP	STR profiling [Using PCR]
1.	When came into use	In 1985. First technique to be used. Not in routine forensic use now.	Relatively newer technique. Became popular in mid-to-late 1990s. Used routinely in forensic work now.
2.	Amount of sample required	Large amounts	Small amounts. Can be "multiplied" using PCR
3.	Analysis of decomposed samples [with DNA fragments <500 bp in size]	RFLP is difficult or impossible.	Can be done.
4.	Enzyme used	Restriction Endonuclease, which cleaves DNA into smaller fragments	Taq Polymerase, which [instead of cleaving DNA into smaller fragments] multiplies them manifold.
5.	Power of discrimination (P_d)	Very high	Low with single STR locus. But very high with multiplex STRs
6.	Speed of analysis	Slow. May take weeks.	Quick
7.	Automation	Not possible	Possible

II. FECAL STAINS

Identification - is by **Edelman test**. Based on the fact that **urobilinogen** is present in fecal stains. Suspect stain+ alcoholic mercuric chloride+ alcoholic zinc chloride"**green fluorescence** if fecal stain. [urobilinogen in the stain is oxidized to urobilin by alcoholic mercuric chloride. In the next stage, urobilin forms a zinc-urobilin complex, which gives the green fluorescence.]

III. LEUCORRHOEAL STAINS

(1) Contain epithelial cells
(2) Have the feel and appearance of seminal stains
(3) Ova of Enterobius vermicularis have been found in them. The worm is known to occur in female bladder and genitals.

IV. MILK

The detection of milk stains or colostrum on undergarments (eg blouse or brassiere) may be important in crimes involving pregnancy, abortion or concealment of birth.

V. NASAL SECRETIONS

(1) Nasal secretion is one of the most difficult body fluids to identify
(2) Especially difficult to discriminate from vaginal secretions and saliva.
(3) People usually blow the nose with paper tissues when the amount of nasal secretion is increased under conditions such as acute nasopharyngitis and allergic rhinitis, or rarely, vasomotor rhinitis or sinusitis.
(4) **Composition of nasal secretions** -
(i) fluid excreted from mucosal glands and cells,
(ii) plasma leaking from blood vessels,
(iii) tears excreted through the nasolacrimal duct from the lacrimal gland.

VI. SALIVA

Identification of saliva is important in alleged cases of fellatio, cunnilingus, kissing, sucking, spitting, or biting.

A. Sources from where saliva may be recovered

(1) Face mask worn by a suspect
(2) Gag or smothering device
(3) Cup or bottle used for drinking
(4) An envelop related to a crime (saliva used for sealing or affixing stamp)
(5) Cigarette butt.

B. Tests

(1) Detection of amylase [**Phadebas test**]
(2) Buccal epithelial cells [DNA profiling can be done]
(3) RNA based assays.

VII. SEMINAL STAINS

For **seminal stains**, please see chapter on sexual offences.

VIII. SWEAT

Sweat stains may become important in cases of violent struggle.

(1) **Dermcidin** is an antibiotic peptide secreted by sweat glands. A monoclonal antibody (**G-81**) has been developed which can react with dermcidin, helping in the identification of sweat stains

(2) **DNA typing** can be done from sweat stains taken from inside of undergarments.

IX. URINE

Examination of urine and its stains may be necessary in cases of murder and sexual assault.

A. General

(1) May appear **yellow.**

(2) Characteristic "**urine**" smell from the stain (due to ammonia evolved by bacterial degradation of urea).

(3) **Fluorescent** under UV light.

B. Chemical tests

Detection of urine stains is based in detection of **amines**, **phosphate**, **sulphate**, **urea** or **creatinine**, each of which exists in urine in higher concentration than in other body fluids. Urinary glycoproteins are also specific to urine.

(1) **Urea** in suspected stain is detected with **urease-bromothymol blue** paper. If the stain is urine, urea would be enzymatically decomposed to **ammonia** and carbon dioxide by urease. Liberated **ammonia shifts the pH**, changing the color of the indicator (bromothymol) from **yellow to blue**.

(2) 0.005% cent sol^{n} of **para-dimethylamino cinnamaldehyde** can be used as a colour test for urea.

(3) **Uric acid (UA) and urea nitrogen (UN) ratio multiplied by 20** (UA/UN # 20) in human urinary stains is **between 1 and 4**; in all other stains and in animal urinary stains this ratio is either < 1 or >

(4) This fact is exploited to identify urine stains.

(5) Urine contains a high molecular weight glycoprotein **Tamm-Horsfall protein** (**THP**), named after its discoverers Tamm and Horsfall (also known as **uromodulin** or **uromucoid**). This has been detected by **radioimmunoassay** to identify unknown stains as urine stains.
(6) **Jaffe reaction** - In the presence of picric acid and a weak base, **creatinine** would give a **bright-red coloration**.

C. Species origin

Precipitin test by the agar diffusion method, against the antiglobulins of various relevant species, including humans.

D. Blood group

Blood group of the person can be determined if the person was a **secretor** (individuals who secret blood group substances into body fluids such as urine).

E. DNA typing

DNA typing can also be done from urine stains.

X. VAGINAL FLUID

In cases of **suspected sexual assault**, traces of vaginal fluid may be detectable on the penis of the accused.
(1) Vaginal fluid contain **vaginal cells** which are rich in glycogen (starch)
(2) If sticking on penis in cases of sexual assault, the vaginal cells would be transferred to a moist blotting paper, if it is rubbed lightly against glans.
(3) Presence of **Döderlein's bacillus**.
(4) The blotting paper is then exposed to **iodine vapors**
(5) **Starch** inside the vaginal cells turns into **starch iodide**, which is **brown in color**.
(6) RNA based assays.

XI. VOMITUS

Vomitus can be detected by (i) **Low pH** resulting from stomach acids. (ii) Microscopic and macroscopic **identification of foodstuffs**.

30. Forensic Science Laboratory

I. LOCARD'S EXCHANGE PRINCIPLE

Locard's exchange principle states that when any two objects come into contact, there is ***always*** a transfer of material from each object on to the other. This principle is often used as a sure proof of crime. The principle was first enunciated by **Edmond Locard** (1877–1966), often referred to as the Sherlock Holmes of France.

Fig. 30.1: Edmond Locard

II. INSTITUTE OF FORENSIC SCIENCE - ORGANIZATION

A comprehensive **Institute of Forensic Science** should comprise of **3** categories of service (i) **Clinical** (ii) **Pathological** and (iii) **Laboratory services**. In addition it should have several other facilities such as (iv) **exhibit room** (where exhibits brought by police, e.g. blood stained weapons, viscera etc) are stored (v) **stores** (for equipment, chemicals etc) (vi) **workshop** (for training) and (vii)

library.

A. Central and State FSLs

(1) Central FSLs are funded by central government.

(2) **Central CFSLs** (Central Forensic Science Laboratories) – **7** in India [**Bhopal, Chandigarh, Guwahati, Hyderabad (Telangana), Kolkata, New Delhi, Pune**]. Of these, **6 CFSLs** [except the one at ND] are under the **Directorate of Forensic Science Services** (DFSS). The one at New Delhi is under CBI [Address: Block No.4, CGO Complex, Lodhi Road, New Delhi-110003]. In addition there is one **institute of serology**, located at **Kolkata**. It was established in 1912 starting with Forensic Serology as its core activity, but now has branched out into different field of Serology. CFSLs meet the demand of police from all over India.

B. Interpol

Interpol (**International Criminal Police Organization**), with headquarters in Lyon, France, was established in **1923**. It facilitates international police cooperation. **190 countries**, including India are its members. It has a comprehensive forensic science laboratory.

III. FORENSIC SCIENCE LABORATORY – INDIVIDUAL SECTIONS

The main functions of forensic science laboratory are: (i) to reconstruct a crime and (ii) to analyze trace evidence (blood, hair, fiber etc from victim, accused and the scene of crime and to link the three together. This work is carried on by different sections. The student is advised to co-relate each section with appropriate chapters in the book.

A. Lie Detection

Following techniques are mostly being used for lie detection.

Tests for lie detection

a. Brain mapping (Brain fingerprinting)

Brain mapping for ***brain fingerprinting*** is a forensic science technique that determines whether specific information (regarding a crime) is stored in a subject's brain. Scientific name of the technique is **Memory and Encoding Related Multifaceted Electroencephalographic Response** (**MERMER**). Invented and developed by **Lawrence Farwell** of US in 1990s.

i. Principle

(1) **Storage of information in brain** - If the subject has committed a crime, the information would be **stored in his brain**.

(2) **EEG responses to crime related information:**

(i) If crime related information (eg picture of the crime scene, photograph of victim, motive of crime etc) are presented to the subject on a computer screen, he may **consciously deny the information**, but his brain would recognize the information.

(ii) This would elicit **relevant EEG waves** which could be detected.

(iii) EEG response to an image, sound etc is known as **event related potential** (**ERP**)

(iv) Memory is an integral part of ERP. (a) If a subject is shown a familiar image (e.g. inside of an aircraft engine to an aircraft mechanic), his EEG would show **memory related waves** in ERP. Similarly if the suspect has killed with a weapon, his brain would be familiar with the weapon. If the weapon is shown to him, his brain would recognize it and would immediately respond with a memory related ERP. These memory related ERPs are called **P300** (because delay between stimulus and response is **300 ms**).

(b) If the subject is shown an unfamiliar image, memory related waves are absent.

ii. Procedure

(1) The subject to be tested wears a special **headband with electronic sensors** (**electro-cap**) that measure the **EEG from several locations on the scalp**.

(2) He is then shown (on a computer screen) several stimuli consisting of words, phrases, or pictures. **Stimuli** are of three types:

(i) "**irrelevant**" stimuli that are irrelevant to the investigated situation and to the test subject [an unknown person, house etc]

(ii) “**target**” stimuli that are relevant to the investigated situation and are known to the subject (face of the victim), and

(iii) “**probe**” stimuli that are relevant to the investigated situation and that the subject denies knowing (weapon of offence, scene of crime etc). **Probe stimuli** contain information that is known only to the perpetrator and investigators, and not to the general public or to an innocent suspect who was not at the scene of the crime.

iii. Results and interpretation

If the subject has killed the victim, his brain would recognize scene of crime and weapon. Even if he consciously denies information, his EEG would show **memory related P300 waves.** He is thus the criminal.

b. Hypnosis

Use of hypnosis for legal purposes is known as Forensic Hypnosis. Hypnosis may be used for retrieval of information from witnesses when it cannot be acquired through any other means. Memories long forgotten or intentionally suppressed may be recovered during sessions of hypnosis.

c. Lie detector (polygraph)

i. Principle

If a person is speaking a lie"Fear"-sympathetic activity"-**heart rate**, -**blood pressure,** -**respiratory rate**. In addition -sweating"-skin conductivity. If a person is asked a question and simultaneously these parameters are being monitored, the investigator would know when he is speaking a lie. Also known as **psychophysiological detection of deception** (**PDD**).

ii. Procedure

Two types of polygraphs (**analog** and **computerized**) are in use. Analog instruments make **actual ink tracings** of above parameters; computerized instruments make an **inkless tracing** on the computer. **Stoelting polygraphs**, both analog and computerized versions, are used commonly. (i) **Pre-test interview** - Polygraph test starts with a pre-test interview to gain some preliminary information which will later be used for “control questions”, or CQ.

(ii) **Explanation of the test** - The tester explains how the polygraph is supposed to work, emphasizing that it can detect lies and that it is important to answer truthfully. (iii) **Actual test** – Questions asked are three types (1) "**irrelevant**" or IR (Is your name Ram?) (2) **"probable-lie" control questions** (CQ) that most people will lie about ("Have you ever stolen money?") (3) "**relevant questions**" (RQ), that the tester is really interested in. The different types of questions alternate. The test is passed (i.e. the subject is not guilty) if the physiological responses during the **probable-lie control questions** (CQ) are **larger than those during the relevant questions** (RQ). (iv) **Post-test interview** - If responses to RQ are smaller than responses to CQ, the tester attempts to elicit admissions during a post-test interview. An example, "Your situation will only get worse if we don't clear this up".

iii. Results and interpretation

If responses to RQ are greater than those to CQ, the person is experiencing fear, and hence lying. Use of **beta blockers** e.g. **propranolol**, may vitiate the test.

d. Narcoanalysis (truth serum)

i. Principle

Narcoanalysis is based on the principle that a person lies using his imagination [inventing lies], which requires full consciousness. By inducing a semiconscious state [narcosis], his capacity of inventing lying is diminished.

ii. Procedure

Sedatives or hypnotics are administered to induce narcosis. Agents commonly used are

(1) **Anticholinergics:**

(i) 3-quinuclidinyl benzilate [an odorless incapacitating agent used in military also. Also known as **BZ**. It is an anticholinergic compound related to atropine, scopolamine, hyoscyamine, and other deliriants]

(ii) Scopolamine [syn, hyoscine].

(2) **Hypnotics:**

(i) Barbiturates [eg sodium amytal (intermediate acting), sodium pentothal (ultrashort acting)- **ch 40**]. Thus often known as **Amytal interview**. Long

acting barbiturates are not suitable

(ii) Ethanol

(iii) Temazepam. The drugs are used either singly or in various combinations.

iii. Results and interpretation

Person loses inhibitions, is relaxed and becomes talkative. May speak truth under loss of inhibitions. The effect is same as when a person speaks truth under the effect of alcohol [Latin In vino veritas, "in wine there is the truth"]. Large number of false negatives are common [i.e. when the person does not confess despite being guilty].

iv. Medicolegal aspects

(1) **Scientific validity** - The scientific validity of the narcoanalysis test has been questioned by medical professionals

(2) **Legal validity:**

(i) Forced use of truth drugs is considered a form of torture according to international law, and according to Declaration of Tokyo [**ch 2**], a doctor cannot participate in torture.

(ii) **Article 20(3) of Constitution of India** states that no person accused of any offence shall be compelled to be a witness against himself. **S.161(2), CrPC** states that a person cannot be bound to answer questions which expose him to a criminal charge. Narcoanalysis contravenes both laws by forcing a person to answer questions incriminating himself.

(iii) In 2010, Supreme Court of India has opined that this test cannot be forced upon any suspect.

(3) **Medical use** – Its use is scientifically and legally valid in the evaluation of certain psychiatric patients.

(4) May cause sudden death due to dose miscalculations or unpredictable reaction to barbiturates.

B. Photography

Forensic photography (forensic imaging) is the use of photography for the purposes of law and administration of justice. Forensic Photography is done for

(1) Recording evidence [e.g. scene of crime, injuries at body etc]. For photography in vehicular accident cases, please see **ch 18**

(2) To reveal what normally cannot be seen. This can be done by (a) UV light (b) IR light.

1. UV light

Memory Aid 1: Forensic applications of UV light

B[4]ritish academy of **F[4]**orensic **S[3]**ciences **G[2]**o**T[3]** a **P[3] A L**

B[4]"Bite, Blood, Bruises, Burns

F[4]"Fibers, Fingerprint, Fake currency, Forgery

S[3]"Scorpion, Semen, Skeleton

G[2]"Glass, GSR

T[3]"Tattoos, Teeth, Tetracycline

P[3]"Poisoning : Ethylene glycol, Lead, Phosphorus

A"Adipocere

L"Lead [repetition. Already mentioned]

2. IR light

Memory Aid 2: Forensic applications of IR light

GeT A B[3] a**D F an " GSR, Tattoos, Artwork, Bite, bloodstains, bruise, Document, Forgery**

31. Introduction, Classification of Poisons, Law Relating to Poisons and General Considerations

I. INTRODUCTION

(1) **Fatal dose** – Amount of drug that would kill 50% of a test population.

(i) Same as LD_{50}.

(ii) Minimum fatal dose [syn minimum lethal dose] is the smallest dose that has been recorded as fatal to a healthy person. [Please also see “Toxicity scales”].

(2) **Fatal period** – Time in which a person dies if given fatal dose.

(3) ***Toxicology*** is the study of the adverse effects of chemicals on living organisms. It includes the study of symptoms, signs mechanisms, treatments, postmortem appearances, detection of poisoning etc.

(4) ***Toxins*** are poisons produced by living cells or organisms. **Ex** - algae [phycotoxins], animals [zootoxins], bacteria [bacterial toxins], fungi [mycotoxins] and plants [phytotoxins]. They are generally small molecules (peptides or proteins), and less complex than an poison or a venom. The toxicity varies greatly in severity, ranging from **minor** (**bee sting**) to **major and severe** (**botulinum toxin**). ***Toxinology*** is the study of toxins.

(5) **Venom** - is a special category of poisons, that must be injected by one organism into another to produce harmful effect [e.g. snake venom]. Venoms are generally high-molecular wt proteinaceous substances [cf poison]. Please also see ch 38 and 39.

(6) **Xenobiotics** - are drugs and chemical compounds foreign to the body [e.g. antibiotics, metals such as lead].

A. The Poisons Act, 1919

This Act regulates the import, possession and sale of poisons. Was amended in 1958.

B. The Drugs and Cosmetics Act, 1940

This Act was passed in 1940, in order to regulate the import, manufacture, distribution and sale of drugs.

C. The Drugs and Cosmetic Rules, 1945

(1) **Framed** - Under The Drugs and Cosmetics Act, 1940

(2) **Schedules** – Contains several imp schedules at the end. (i) Schedule C – Biological and Special Products. (ii) Schedule E – Omitted since 1982. Instead there is schedule E1. which gives a list of poisonous substances under the Ayurvedic, Siddha and Unani Systems of Medicine. (iii) Schedule F – (a) Part I to XII-A - Omitted since 1999. (b) F1. prescribes specifications of following products. Part 1– Vaccines. Part II –Antisera. Part III- Diagnostic antigens [Bacterial Origin]. (iv) Schedule G – Contains about 65 drugs [eg

Aminopterin, antihistamines, L-Asparaginase, Bleomycin, Insulin etc], which must be labeled with the words "Caution: it is dangerous to take this preparation except under medical supervision". (v) Schedule H – contains several prescription drugs, which must be labeled with the symbol **Rx** and following warning: 'Schedule H drug- Warning: To be sold by retail on the prescription of a Registered Medical Practitioner only'. The label of innermost container of drugs in schedules '**G**' and '**H**' should additionally bear a conspicuous **red vertical line** on the **left side** running throughout the body of the label which should not be **<1mm** in width. (vi) Schedule J – contains a list of 51 diseases for the cure of which no drug can be advertises [eg AIDS].

Memory Aid 1: 6 important Schedules under The Drugs and Cosmetic Rules, 1945

C a B E P idemic. F i V e G e G s H elP J uD ges

Schedule C – Biological and special products

Schedule E (1) – Poisonous substances

Schedule F – Vaccines, antisera and diagnostic antigens

Schedule G – Medical supervision, drugs to be taken under

Schedule H – Prescription drugs

Schedule J – Disease for the cure of which no drug can be advertised

D. The Drugs Control Act, 1950

It provides for the control of the sale, supply and distribution of drugs.

E. The Drugs and Magic Remedies (Objectionable Advertisement) Act, 1954

The objective of this Act is to ban advertisements which offend decency or morality, and to prevent self medication and treatment, which cause harmful effects.

F. The Medicinal and Toilet Preparations (Excise Duties) Act, 1956

This Act provides for the levy and collection of duties of excise on medicinal and toilet preparations containing alcohol and narcotic drugs.

G. The Narcotic Drugs and Psychotropic Substances (NDPS) Act, 1985

The Narcotic Drugs and Psychotropic Substances (NDPS) Act was enacted in ***1985***, by the Indian Govt (i) to ***consolidate*** and ***amend*** the law relating to narcotic drugs (ii) to make ***stringent provisions*** for regulation of Narcotic drugs and psychotropic substances (iii) to provide for ***forfeiture of property*** derived from narcotic drug trafficking, and (iv) to ***implement the provisions*** of the International Convention on Narcotic Drugs and Psychotropic substances.

II. MEDICOLEGAL ASPECTS OF POISONS

(1) **S.85, IPC - Criminal act done under involuntary intoxication** - Person is intoxicated without his knowledge or against his will"becomes incapable of judgment"commits a criminal act"Did not know what he was doing"He is not responsible for the crime. **Example** – **X** administers datura to **Y** without his knowledge"**Y** murders **Z** under the effect of Datura"**Y** is not responsible.

(2) **S.86, IPC - Criminal act done under voluntary intoxication** - Person takes intoxicating drug voluntarily"looses self control"Commits a crime"taken for medical examination"Found disoriented in time, place and person [i.e. from a medical standpoint, he did not know what he was doing]"Legally speaking it would still be presumed that he knew what he was doing, but his intention to do so cannot be presumed.

(3) **S.274, IPC** - Adulteration of drugs — 6 m or `1000 fine or both.

(4) **S.275, IPC** - Sale of adulterated drugs — 6 m or `1000 fine or both.

(5) **S.276, IPC** - Sale of drug as a different drug or preparation — 6 m or `1000 fine or both.

(6) **S.277, IPC** - Fouling water of public spring or reservoir — 3 m or `500 fine or both.

(7) **S.278, IPC** - Making atmosphere noxious to health — `500 fine.

(8) **S.284, IPC** - deals with negligent conduct with respect to poisonous substance. Punishment **6m** or **`1000** fine or both. **Ex** –Housemaid leaves toilet cleaner (acid) negligently on a table"A young child drinks it believing it to be water"Housemaid can be sued u/s 284, IPC.

(9) **S.324,** IPC*"ch 11

(10) **S.326,** IPC*"ch 11.

(11) **S.328,** IPC:

(i) Person gives or otherwise administers a stupefying, intoxicating or unwholesome agent, (a) with an intention of causing hurt or (b) with intent to commit or to facilitate the commission of an offence or (c) knowing it to be likely that he will thereby cause hurt"10 y+fine

(ii) Drug facilitated sexual assault [**ch 25**] attracts provisions of this section.

(12) **Secondary poisoning** - Poisoning that results when one organism ingests [or comes into contact with] another organism that has been poisoned.

(i) Occurs typically when a predator eats an animal, [eg mouse, rat, insect] that has previously been poisoned by a commercial pesticide. It can affect humans. Minamata Bay disaster [please see ch 36] was an example of secondary poisoning. [cf Secondary drug fatalities. Please see below].

(ii) Sucking venom out of a snake bite wound [**ch 38**"MLI of snake venom].

(iii) Medical and paramedical staff can get secondary exposure to poisons during treatment of certain poisons [OP, organochlorine, Nicotine]. They should wear protective gear during entire management period [impervious gloves and gowns].

III. NATURE OF POISONING

A. Homicide

Characteristics of an **ideal homicidal poison** are

(1) should be **colorless, odorless** and **tasteless** [most important]

(2) should be **easily available**

(3) should be **cheap**

(4) its symptoms should resemble a **natural disease** [**Table 8**]

(5) **symptoms should appear late** after administration, to avoid suspicion

(6) Should not produce vomiting [expulsion]

(7) should be **highly toxic**

(8) **Fatal period** – should be less once symptoms have appeared

(9) There should be **no antidote** available against it

(10) There should be **no post-mortem** changes

(11) should **disappear** from the body after death

(12) there should be **no test available** to detect it.

B. Suicide

Characteristics of an **ideal suicidal poison** are

(1) Should produce an easy death [most important criteria. Opium and barbiturates fulfill this criteria]

(2) Easily available [organophosphorus, aluminum phosphide]

(3) Capable of being easily taken in food or drink. **Cheapness is not a criteria**, as a dying man would not worry about the cost. **Color, odor or taste are not important** on the same grounds. **Table 1** gives some importance differences between an ideal homicidal and ideal suicidal poison.

Table 1: Differences between an ideal homicidal and ideal suicidal poison

S.No	Characteristics	Ideal homicidal poison	Ideal suicidal poison
1.	Color	Colorless	Any color
2.	Odor	Odorless	Any odor
3.	Taste	Tasteless	Any taste
4.	Solubility	Should be easily soluble in food and drink, without causing any obvious change	No such requirement
5.	Signs and symptoms/clinical diagnosis	Should resemble a natural disease/clinical diagnosis should be difficult	Should cause a painless death
6.	Onset of signs and symptoms	Should be very slow, so it is very difficult to associate signs and symptoms to the poison	Should be quick
7.	Fatal dose/toxicity	Should be very low/toxicity should be high	No such requirement
8.	Fatal period	No particular requirement. Short fatal period however precludes the victim from getting effective treatment	Should be very less, so as to give a quick and easy death
9.	Availability of poison	Should be easily available	Should be easily available
10.	Availability of antidotes	Should not be available for the poison	No such requirement
11.	Detectability	Should not be detectable	No such requirement
12.	Disappearance after death/ metabolism	Should disappear after causing death/ metabolism should be quick	No such requirement
13.	PM changes	Should produce no PM changes	No such requirement
14.	Cost	Cheap	No such requirement
15.	Examples	There is no ideal homicidal poison. Some poisons which come close are (i) arsenic (ii) thallium	Opium, barbiturates, Aluminum phosphide

C. Parasuicide

Parasuicide (Gk para, “near” or “resembling”) or ***pseudocide*** refers to suicide attempts or gestures and self-harm where there is no actual intention to die.

Salient features:

(1) Mostly done to threaten relatives by taking sublethal dose of a poison, e.g. a few sleeping pills. Youngsters resort to it when their parents disallow marriage with their loved ones.
(2) Also seen in psychologically disturbed.

D. Accidental

Accidental poisoning can occur in a number of ways

(1) Ingestion in mistake for harmless substance [acid may be ingested as it appears like water]
(2) Inhalation of vapors through accident [MIC vapors inhaled in Bhopal gas tragedy]
(3) incorrect preparation of medicines that contain a poison
(4) Excessive self medication
(5) drug addiction
(6) infected food
(7) Animals (snake and spider bite, scorpion sting).

E. Abortion

Most **irritant poisons** would cause abortion (by causing irritation of uterus, causing it to contract). **Ex**. Aconite, Arsenic, Calotropis, Cantharides, Croton, Ergot, Lead, Mercury, Oleander, Potassium permanganate, Semecarpus.

F. Aphrodisiacs

An ***aphrodisiac*** (from Aphrodite, the Greek goddess of sensuality and love) is a substance that increases sexual desire. Usual substances considered aphrodisiacs are arsenic, Cannabis, Cantharides, Cocaine, Opium and Strychnine.

G. Arrow Poisons

Arrow poisons are used at the tip of arrows for hunting and to kill enemies.

Salient features:

(1) **Ideal arrow poison** - must be
(i) Poisonous parenterally, but
(ii) Safe orally so prey killed by an ideal arrow poison would be safe for consumption.

(2) **Typical arrow poisons** are
(i) Plant based - (a) Abrus precatorius, (b) aconite, (c) calotropis, (d) croton oil, (e) curare, (f) Strophanthus (g) strychnine.
(ii) Animal based - (a) **Batrachotoxin** [BTX].

H. Cattle Poisoning

Used to kill cattle of enemies, or to obtain hides. Usual poisons are Abrus precatorius, Aconite, Arsenic, Calotropis, nitrates, Oleander, Organophosphorus, Strychnine, Zinc phosphide.

I. Stupefying

Used to stupefy travelers for robbery, or women for sexual assault. **Ex**. Cannabis, Chloral hydrate, Datura.

IV. CLASSIFICATION OF POISONS

Classification of poisons can be (A) according to symptoms produced (B) according to use (C) according to affected target organ.

A. According to Symptoms

1. Corrosives

a. Strong acids

i. Inorganic acids

Sulphuric, **nitric**, **hydrochloric acids.**

ii. Organic acids

Carbolic, **oxalic**, acetic, salicylic acids.

b. Strong alkalis

NaOH, KOH, NH_4OH.

c. Metallic salts

Bichromates, Chromates, **Copper sulphate**, Ferric chloride, Potassium cyanide, Silver nitrate, Zinc chloride.

2. Irritants

a. Inorganic

i. Non-metallic

Phosphorus, Chlorine, Bromine, Iodine.

ii. Metallic

Arsenic, **Copper**, **Lead**, **Mercury**, Thallium, Zinc.

b. Organic

i. Plant

(a) All parts poisonous

Calotropis, Cerbera thevetia, Nerium odorum.

(b) Leaves poisonous

Aloe vera, Conium maculatum (hemlock), curare, digitalis, tobacco.

(c) Fruits poisonous

Capsicum annum, Colocynth, Strychnos nux vomica.

(d) Seeds poisonous

Abrus precatorius (Ratti), Castor, Croton, Datura, Semecarpus anacardium.

(e) Bark / stem poisonous

Cinchona, Plumbago rosea.

(f) Root poisonous

Aconite, Colocynth, Plumbago.

ii. Animal

Snake and insect venom, cantharides, fireants, scorpion.

c. Mechanical

Diamond dust, hair, Powdered glass.

3. Systemic

a. CNS poisons

i. Cerebral

(a) CNS stimulants

Amphetamine, caffeine, cyclic antidepressants, methamphetamine, methylphenidate, modafinil.

(b) CNS depressants

Alcohols, anesthetics, hypnotics, opioids, sedatives.

(c) Deliriants

Belladonna, Cannabis, cocaine, Datura, Hyoscyamus.

ii. Spinal

Gelsemium, Nux vomica.

iii. Peripheral nerve poisons

Conium, curare, several agricultural poisons such as OP compounds and carbamates.

b. CVS poisons

Aconite, Oleander, Quinine, Tobacco, antiarrhythmic agents, aminoglycosides, macrolides, General anesthetics.

c. Asphyxiants

CO, CO_2, H_2S, Cl_2.

4. Miscellaneous

Botulism, Food poisoning.

B. According to Use

(1) Poisons causing abortion (2) Poisons used to stupefy (3) Cattle poisons (4) Arrow poisons (5) Aphrodisiacs (6) Others. For details, please see above under the heading "**Nature of poisoning**".

C. According to Affected Target Organ

1. Poisons affecting the hematologic system

Anticoagulants (warfarin).

2. Poisons affecting the immune system (immunotoxic agents)

Immunosuppressive drugs.

3. Hepatotoxic agents

There are 2 good mnemonics for remembering hepatotoxic drugs – one short, another longer.

Memory Aid 2: Hepatotoxic drugs

HEPATIC - **H**emolytic poisons, **E**thanol, **P**aracetamol, **A**flatoxin, **T**oxaphene, **I**ron, **C**hlorpromazine

Memory Aid 3: Hepatotoxic drugs

A SOAP IN A CAPP - **A**lcohol, **S**ulphonamides, **O**ral contraceptives, **A**rsenic, **P**aracetamol, **I**ron, **N**aphthalene, **A**minoglycosides, **C**arbon tetrachloride, **A**nabolic steroids, **P**araquat, **P**hosphorus

4. Nephrotoxic agents

(A) Poisons causing **glomerular injury**: cocaine, quinine, conjugated estrogens, gold compounds, NSAIDs, penicillamine. (B) Poisons causing **proximal convoluted tubule (PCT) necrosis**.

Memory Aid 4: Poisons causing PCT necrosis

[**Mnemonic 1**]

APPROACH MAG MOM

Aminoglycosides, **P**henol, **P**igments, **R**adiocontrast agents, **O**rganohalides [eg Chloroform, CCl_4], **A**nesthetics (Fluorinated), **C**antharides, **C**arbon tetrachloride, **C**ephalosporins (and other beta-lactam antibiotics), **C**resol, **H**alogenated alkenes [derivatives of $\mathbf{C_nH_{2n}}$], **M**etals (Ar, Cd, Cr, Hg, Pb, Fe, U, Zn), **A**ntineoplastics, **G**lycols (e.g. ethylene glycol), **M**yoglobin (including hemoglobin), **C**ycotoxins (**O**chratoxin A, Fumonisin B).

[**Mnemonic 2**]

PCT [shorter mnemonic, but useful]

Phenol, **P**igments

Cantharides, **C**ephalosporins (and other beta-lactam antibiotics), **C**orrosive sublimate [$HgCl_2$] [included in metals in 1st mnemonic], **C**resol

Tetrachloride [carbon] [included as $\mathbf{CCl_4}$ in 1st mnemonic]

(C) Poisons causing **distal convoluted tubule (DCT) necrosis**.

Memory Aid 5: Poisons causing DCT necrosis
CAG
Cisplatin, **A**mphotericin, **G**lycols

(D) Poisons causing **crystalluria**: acyclovir, ethylene glycol, sulfonamides,

(E) **General Nephrotoxic agents**

Memory Aid 6: General Nephrotoxic agents
NEP^{2}HRO
NSAIDS, **E**rgot alkaloids, **P**otassium chlorate, **P**re renal failure, **H**eavy metals, **AR**habdomyolysis causing drugs, **O**xalic acid, **C**hlorates

5. Pulmonary toxic agents

chlorine (bronchitis), titanium (hard metal disease), silica (silicosis), phosgene (pulmonary edema), cholinergic drugs (bronchoconstriction), paraquat (acute pulmonary fibrosis).

6. CNS toxic agents

See classification "According to symptoms" above.

7. CVS toxic agents

See classification "According to symptoms" above.

8. Dermatotoxic agents

Arsenic (carcinogenic), camphor (photoallergy), corrosives (chemical burns), Furocoumarins (phototoxicity), industrial solvents (allergic contact dermatitis).

9. Agents affecting the reproductive system

alkylating agents (affect gonadogenesis), Chlorophenoxy herbicides (affect spermatogenesis), Plasticizers (testicular toxicity).

10. Agents affecting the endocrine system

acrylonitrile (adrenal cortex), atenolol (adrenal medulla), Aluminum (parathyroid), calcitonin (pituitary), Thiouracil (thyroid gland).

V. TYPES OF POISONING

The following types of poisonings depend on (i) the amount of poison taken and (ii) the time period over which poison taken.

A. Hyperacute [Fulminant] Poisoning

Produced by a single massive dose. Death occurs very rapidly. There may be no time for symptoms to occur.

B. Acute Poisoning

Acute poisoning is caused by excessive single dose (usually equal to fatal dose), but less than that used in fulminant poisoning.

Types of acute drug fatalities

a. Primary drug fatalities

Victim dies primarily because of the toxic or adverse effect of drug/poison. There may be contributing influence of pre-existing, unrelated disease.

b. Secondary drug fatalities

Victim dies from secondary complications of drugs (viral hepatitis, bacterial endocarditis, air embolism, foreign body embolism). Most commonly occur due to use of contaminated needles during drug abuse. [cf. Secondary poisoning. Please see above].

c. Drug associated fatalities

Violence [accidental, homicidal, suicidal] arising directly or indirectly from activities related to obtaining and use of illicit drugs.

C. Subacute Poisoning

Subacute poisoning occurs gradually over some time; occurs more slowly than acute poisoning, but quicker than chronic poisoning. Doses taken are small and over a short period of time. A typical example is **drug automatism** [**ch 28**].

D. Chronic Poisoning

Chronic poisoning is caused by very minute doses taken over a long period of time. Typically seen in occupational poisoning, such as **Plumbism**. A murderer who has access to victim's food (e.g. spouse of the victim) is in a position to administer minute doses of poison over a long period of time. Typical poison used is arsenic. After months, the person may die of gastroenteritis.

E. Acute on Chronic Poisoning

Primarily the victim is suffering from **chronic poisoning**. Acute poisoning occurs on top of it. Mostly happens in cases of **chronic drug abuse**, when one day victim inadvertently takes more than the usual dose.

VI. DIAGNOSIS OF POISONING

A. In the Living

Diagnosis of poisoning in the living is done mainly by three points (**Table 2**).

Table 2: Diagnosis of poisoning in the living

1. Clinical history
2. Clinical signs [including toxidromes]
3. Lab investigations

1. Clinical history

Clinical history is of great importance [**Memory Aid 7**]. There is no single symptom which is characteristic of poisoning. A constellation of symptoms however (please see toxidromes below) may often point to poisoning.

Memory Aid 7: Major questions to ask in poisoning cases

In poisoning cases history really "**MATtERS**"

M"**M**edications or **M**aterials ingested or exposed to

A"**A**mount ingested

Tt"**T**ime of exposure

E"**E**mesis [did it occur? What came out?]

R"**R**eason for the ingestion or exposure. [Accidental, suicidal or homicidal]

S"**S**igns and **s**ymptoms

Suspicion should be aroused when

(1) There is **sudden appearance of symptoms. Major symptoms** associated with **poisoning** are

(i) **GIT -** (a) abdominal colic and pain [**Table 3**] (b) vomiting, diarrhea [**Table 4**] (c) Hematemesis - (I) Aminophylline, (II) corrosives, (III) iron, (IV) salicylates (d) Constipation [**Table 5**]

(ii) **Hepatic** - (a) jaundice [please see list of hepatotoxic agents above]

(iii) **CNS** - (a) paralysis (b) tremors [**Table 6**] (c) convulsions and seizures [**Table 7**] **–** cause rhabdomyolysis (d) coma (iv) **Renal** - (a) oliguria with proteinuria and hematuria] (v) **Natural diseases** - A number of poisons mimic them [**Table 8**]

(2) **there is association with food** – symptoms occur after taking food

(3) **there are multiple victims - several persons** fall ill simultaneously. **Confirmation** is by detection of poison in food, vomit and feces.

Table 3: Causes of Acute Abdomen Due to Poisoning

1. **Black widow spider bites**
2. **Drugs** – (i) Anticoagulants [due to Pancreatitis, Bowel hematoma and Retroperitoneal bleeding]; (ii) Azathioprine, (iii) Steroids, (iv) Thiazides [due to Pancreatitis]; (v) Barbiturates [due to reactions in porphyria patients]; (vi) Corticosteroids [due to rapid withdrawal Addisonian crisis]; (vii) Oral contraceptives [due to Hyperlipemia and pancreatitis]
3. **Heavy metals**-Pb, As, Hg
4. **Improperly canned food**
5. **Mushroom poisoning**.

Table 4: Poisons causing vomiting and diarrhea

1. All irritants as mentioned in classification of poisons above.
2. OP [opposite effect in anticholinergics as both are physiological antidotes].

Table 5: Drugs and Poisons causing constipation

1. Anticholinergics [eg antiparkinsonian drugs, antipsychotics, Antispasmodics, *Atropa belladonna,* Atropine, *Datura*, H1 antihistamines, *Hyoscyamus niger*, tricyclic antidepressants (TCA)][opposite effect in OP as both are physiological antidotes]
2. botulism
3. Iron
4. Lead
5. Lithium
6. Opium and derivatives [morphine, codeine, heroin].

Table 6: With Memory Aids: Poisons Causing tremors

Mnemonic – **CAPCAMP**

1. Caffeine and Theophylline
2. Antidepressants [tricyclic]
3. **P**hosphorus [when liver damage has occurred]
4. Carbon monoxide
5. **A**lcohol
6. **M**etallic Poisons [esp Mercury]
7. **P**henothiazines.

Table 7: Agents that cause convulsions and seizures. **[with Memory Aid – 8]**

Mnemonic - **OTIS CAMPBELL**

1. **O**rganophosphates, organochlorines (DDT)
2. **T**heophylline, tricyclic antidepressants
3. **I**soniazid, insulin
4. **S**alicylates**, s**trychnine, sympathomimetics
5. **C**amphor, cocaine
6. **A**mitripty-line, amphetamines, anticholinergics
7. **M**ethylxanthines (theophylline, caffeine)
8. **P**hencyclidine (PCP)
9. **B**enzodiazepine withdrawal, botanicals (water hemlock)
10. **E**thanol withdrawal
11. **L**ithium, lidocaine
12. **L**ead, lindane.

Table 8: Poisons and diseases/ailments mimicked by them

S.No	Poison	Disease/ailment mimicked
1.	Abrus precatorius	Viper bite
2.	Arsenic [acute]	Cholera
3.	Arsenic [Chronic]	Addison's disease, Measles
4.	Bromide	Acne
5.	Carbolic acid	Alkaptonuria
6.	Dinitro compounds	Thyrotoxicosis
7.	Iodide	Mumps (Iodide mumps)
8.	Organophosphorus	Asthma
9.	Phosphorus	Acute yellow atrophy
10.	Strychnine	Tetanus
11.	Thallium	Guillain–Barré syndrome
12.	Zinc, some other metals	Flu, Malaria (zinc chills)

2. Clinical signs

Important clinical signs which point to poisoning are:

a. BP

Table 9: Poisons causing hypotension. **[with Memory Aid – 9]**

CRASH – **C**lonidine, Cyanide [late], **R**eserpine [antihypertensives], **A**ntidepressants, **S**edative–hypnotics, **H**eroin.

Table 10: Poisons causing hypertension

Hypertension with tachycardia:

1. **Generalized sympathomimetic agents:** (i) Amphetamines and derivatives (ii) Cocaine (iii) Ephedrine and pseudoephedrine (iv) Epinephrine (v) Levodopa (vi) LSD (vii) Marijuana (viii) MAOI
2. **Anticholinergic agents** - (i) Antihistamines (ii) Atropine and other anticholinergics (iii) Tricyclic antidepressants
3. **Other** -(i) Ethanol and sedative-hypnotic drug withdrawal (ii) Nicotine [early stage] (iii) OP [early stage].

Hypertension with bradycardia or AV block -

1. Clonidine, tetrahydrozoline, and oxymetazoline
2. Cyanide [initially]
3. Ergot derivatives
4. Methoxamine
5. Norepinephrine
6. Phenylephrine
7. Phenylpropanolamine.

b. Heart rate

Poisons may cause bradycardia [**Table 11**], tachycardia [**Table 12**] or cardiac arrhythmias [dysrhythmias] [**Table 18**]. Intoxication is the single commonest cause of cardiac arrhythmias.

Table 11: Poisons causing bradycardia

1. Alcoholic myocardiopathy
2. β-blockers, eg propranolol [Inderal]
3. calcium channel blockers
4. cholinergics
5. clonidine
6. Cyanide [initially]
7. digitalis, Digoxin and related drugs
8. opioids
9. organophosphates
10. plants [(i) Foxglove (digitalis), (ii) Lily-of-the-valley (*Convallaria majalis*), (iii) oleander]
11. Procainamide
12. quinidine toxicity
13. **Miscellaneous** - Also seen in (i) *hypokalemia* of chlorothiazide diuretics and (ii) *hyperkalemia* of (a) spironolactone (b) triamterene [Dyrenium] and (c) uremia.

Table 12: Poisons causing tachycardia. **[with Memory Aid – 10]**

(1) Alcohol (2) Amphetamines (3) anticholinergics (4) antihistamines (5) cocaine (6) Cyanide [late] (7) phencyclidine (8) salicylates (9) sympathomimetics (10) theophylline (11) tricyclic antidepressants. **Memory Aid C-PA^{4}S^{2}T^2.**

Table 13: Poisons causing Cardiac Arrhythmias

(1) Alcohol (2) caffeine (3) digitalis (4) procainamide (5) propranolol (6) quinidine (7) tobacco (8) Diuretics cause electrolyte disturbances that may cause or contribute to cardiac arrhythmias.

c. Ocular signs

(1) Normal pupil size in adults: **2-4 mm** in dia (in bright light); **4-8 mm** in the dark. The iris allows the diameter of the pupil to change from **1 to 12 mm**, depending on **illumination** and **accommodation**; such changes are influenced by a perfect balance between the sympathetic and parasympathetic nervous systems.

(2) **Miosis** - Constriction of the pupil to <2mm diameter (i) Mechanisms – (a) Central [eg Opium"ch 40] (b) Peripheral [eg OP"ch 35]

(3) **Table 14**, **Table 15**, **Table 16**, indicate common ocular signs and their causes.

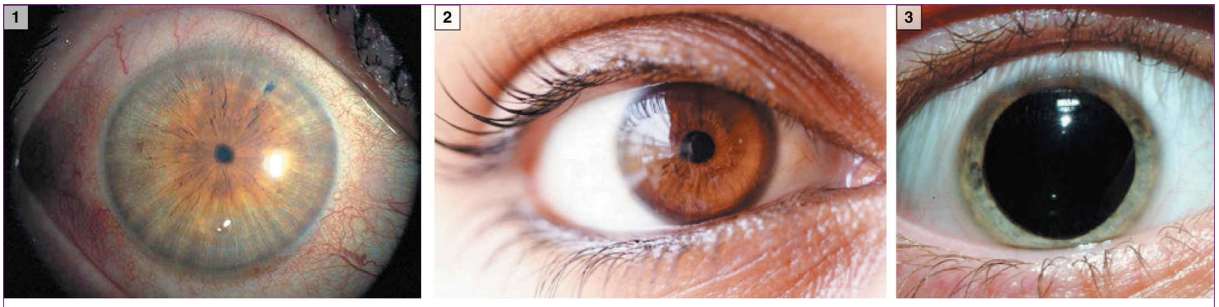


Fig. 31.1: **(1)** Constricted pupil, size 1 mm in opium poisoning **(2)** Normal pupil, size 4 mm and **(3)** Dilated pupil, size 12 mm in Datura poisoning.

Table 14: Poisons causing Miosis (Pupillary constriction)

(1) *Abrus precatorius* (2) Acetone (3) Alcoholic coma (4) Antipsychotics - (i) Haloperidol (ii) Phenothiazine, eg chlorpromazine (iii) ziprasidone (5) Barbiturates (6) Benzodiazepines (7) Bromide (8) Caffeine (9) Carbamates (10) Carbolic acid [Phenol] (11) chloral hydrate (12) Chlorophenoxy herbicides (13) Clonidine (14) Methyldopa (15) Muscarinic mushrooms (16) Nicotine (17) Opium and related narcotics [dilated in withdrawal] (18) OP insecticides (19) Parasympathomimetics (20) Phencyclidine [PCP] (may cause both miosis or mydriasis) (21) phenothiazines.

Table 15: Poisons causing Mydriasis (Pupillary dilation)

(1) Acetylsalicylic acid (2) Alcohol [but constricted in alcoholic coma] (3) Amphetamine (4) Anticholinergic [e.g. atropine. Pupils do not respond to light] (5) Antihistamines (6) Chloroform [dilate initially, followed by contraction] (7) CO [nystagmus also] (8) cocaine [pupils respond to light] (9) Curare (10) Cyanide (11) Datura (Atropine) (12) Ephedrine (13) Ergot (14) Formic acid (15) Gelsemium (16) LSD (17) Methanol (18) Opioid withdrawal (19) Organochlorines (20) Paraldehyde (21) Phencyclidine [PCP] (may cause both miosis or mydriasis) (22) Scorpion (23) Sympathomimetics (24) Tricyclic antidepressants (25) Viper bite (26) Yew [Taxus baccata].

Table 16: Poisons causing Nystagmus

S.No	Type of nystagmus	Poison
1.	Horizontal	(1) Alcohol (most common cause) (2) Barbiturates (3) carbamazepine (4) CO [mydriasis also] (5) lithium (6) MAOI (7) meprobamate (8) Phencyclidine (9) phenytoin (10) primidone (11) quinine (12) sedative-hypnotics (13) solvents
2.	Vertical	(1) Phencyclidine (2) phenytoin (3) sedative-hypnotics
3.	Rotary	(1) Phencyclidine (2) phenytoin (3) sedative-hypnotics

d. Ears

Buzzing in ears: (i) camphor (ii) ergot (iii) methyl alcohol (iv) quinine (v) Salicylates (vi) streptomycin.

e. Oral signs

(1) Burns: (i) Corrosives (ii) oxalate-containing plants
(2) Dry mouth: (i) Amphetamines (ii) anticholinergics (iii) antihistamine
(3) Dysphagia: (i) botulism (ii) Corrosives
(4) Gum lines: (i) arsenic (ii) bismuth (iii) Lead (iv) mercury
(5) Salivation: (i) corrosives (ii) OP (iii) salicylates (iv) strychnine.

f. Pulmonary

Respiratory rate
Table 17, **Table 18**.

Table 17: Poisons causing bradypnea (slow respiration, respiratory depression)

S.No	Mechanism	Poisons causing
1.	Failure of respiratory center	(1) Antidepressants [Tricyclics] (2) Antihistamines (3) Antipsychotics [Phenothiazines] (4) clonidine [and other sympatholytic agents] (5) Cyanide [late stages] (6) Ethyl alcohol [and other alcohols] (7) Gamma hydroxybutyrate (GHB) (8) narcotics [e.g. heroin, opiates] (9) sedative-hypnotics [eg barbiturates, benzodiazepines]
2.	Failure of respiratory muscles	(1) Botulinum toxin (botulism) (2) NM blocking agents (3) Nicotine (4) OP and carbamates (5) Paralytic shellfish poisoning [Saxitoxin (red tide)] (6) Snake bite [cobra] (7) Strychnine (8) Tetrodotoxin (9) Warfare nerve gases

Table 18: Poisons causing tachypnea (fast, rapid respiration)

(1) Acetylsalicylic acid (2) Amphetamines (3) barbiturates (early) (4) carbon monoxide (5) Cyanide [initial stages] (6) ethylene glycol (7) methanol (8) paracetamol (9) salicylates.
NB – Drugs causing metabolic acidosis (aspirin, paracetamol) will cause tachypnea.

g. CNS signs

(1) **Ataxia:** (i) Alcohol (ii) antidepressants (iii) barbiturates (iv) anticholinergics (v) phenytoin (vi) narcotics
(2) **Coma:** (i) barbiturates (ii) CO (iii) cyanide (iv) cyclic antidepressants (v) lead [encephalopathy] (vi) narcotics (vii) OP (viii) PCP (ix) salicylates (x) Sedatives
(3) **Muscle fasciculation:** (i) OP (ii) theophylline
(4) **Muscle rigidity:** (i) Cyclic antidepressants (ii) haloperidol (iii) PCP (iv) phenothiazines
(5) **Paresthesia:** (i) Cocaine (ii) camphor (iii) PCP (iv) Monosodium glutamate [MSG]
(6) **Peripheral neuropathy:** (i) Lead (ii) arsenic (iii) mercury (iv) organophosphates.

h. Skin [cutaneous signs]

(1) **Alopecia** – ch poisoning with (i) arsenic (ii) lead (iii) mercury (iv) Thallium
(2) **Blisters and bullae:** (i) barbiturates (ii) CO [for a complete list please see **ch 40**]
(3) **Diaphoresis:** (i) alcohol [acute] (ii) arecoline (iii) arsenic (iv) aspirin (v) cocaine (vi) Dinitrophenol (vii) fluoride (viii) insulin (ix) LSD (x) mercuric chloride (xi) muscarinic mushrooms (xii) nitrates (xiii) OP (xiv) pilocarpine (xv) salicylates
(4) **Dry hot skin:** (i) Anticholinergic agents (ii) botulism
(5) **Erythema:** (i) Anticholinergics (ii) Boric acid (iii) cyanide (iv) mercury.
(6) **Formication** – As [**ch 36**], Cocaine [**ch 41**]
(7) **Hyperkeratosis and pigmentation:** (i) ch arsenic
(8) **Injection marks and Needle tracks:** (i) Drug abuse (a) Amphetamines (b) Heroin (c) PCP (d) Any other drug taken IV or S/c (ii) snakebite [two fang marks]
(9) **Jaundice** - Hepatotoxic poisons (please see list of hepatotoxic agents above)]
(10) **Petechiae and purpuric spots** – Warfarin
(11) **Sweating** – please see diaphoresis.

i. Temperature

(1) Hypothermic patients [**Table 19**] may have a barely perceptible pulse and BP.
(2) Hypothermia may cause or aggravate hypotension, which will not reverse

until the temperature is normalized.

Table 19: Poisons causing hypothermia

(1) Alcohols* (2) Antidepressants (3) barbiturates (4) benzodiazepines (5) CNS depressants (6) Carbon monoxide (7) ethanol (8) hypnotics/sedatives (9) hypoglycemic agents (10) opioids (11) phenothiazines (12) Sedative hypnotics (13) tricyclic antidepressants

*Ethanol adversely affects thermal regulation and, depending upon the ambient temperature, may cause either hypothermia or hyperthermia.

Table 20 lists poisons causing hyperthermia.

Table 20: Poisons causing hyperthermia [hyperpyrexia]

(1) Acetylsalicylic acid (2) Amphetamine (3) Anticholinergics [Datura] (4) Arsenic (5) Cyanide (6) Dinitro-Orthocresol [DNOC] (7) dinitrophenol pesticides (8) Halothane (9) hydroxy benzonitrile herbicides (10) pentachlorophenol (11) procainamide (12) quinidine (13) Suxamethonium

j. Urine

May be colored in certain poisonings [**Table 21**].

Table 21: Some possible causes of colored urine

S.No	Colour	Possible Cause
1.	Yellow/ brown	Bilirubin, hemoglobin, myoglobin, chloroquine, cresol, quinine
2.	Red/ brown	Bilirubin, hemoglobin, myoglobin, Porphyrins, urobilin deferoxamine, levodopa (black on standing)
3.	Blue/ green	Bile, biliverdin, indican (on standing) Acriflavine (green fluorescence), **phenol**
4.	Black	Blood (on standing), **phenol**, homogentisic acid, indican (on standing)

k. Blood

i. Collection

[Collection of blood for toxicology in the dead"**ch 5]** Following precautions are observed

(1) **Equipment:** (i) Use clean syringe, needles and containers. None should have any trace of alcohol. (ii) Nowadays Vacutainers are used, which are sterile glass or plastic tubes with a closure that is evacuated to create a vacuum inside the tube facilitating automatic drawing of a predetermined volume of blood. They already contain an anticoagulant and a preservative. (iii) Container – screw-capped glass bottle of “universal” size. Not needed if Vacutainers are used.

(2) **Cleaning skin** - (i) Do not use spirit (ii) use 1:1000 $HgCl_2$ or plain soap and water

(3) Use adequate preservative - **ch 5**.

(4) **After collection:** (i) sealing - (a) The container should be tightly clamped and sealed to prevent loss of alcohol by evaporation. (b) Rubber stoppers should be avoided because they may contaminate the sample with oxidizable

substances. (c) In medicolegal cases, number of seals must be mentioned and a sample of seal must be sent along with [chain of custody]. (ii) Labeling – label with CR no, name, age, sex, date and time of collection of specimens

(5) Send for analysis immediately. If cannot be sent immediately for some reason, refrigerate, but must not be frozen. Freezing causes cells to lyze causing spurious concentrations.

(6) Analysis – should best be done within a week.

ii. Inspection

(1) Brown serum – Poisons causing rhabdomyolysis [myoglobin release]

(2) Chocolate color - methemoglo-binemia.

(3) Pink serum – hemolysis [eg sodium chlorate poisoning].

l. ECG

Useful in cardiotoxic ingestions, e.g. AlP, thallium.

m. EEG

Useful in neurotoxic ingestions.

n. Hair

Hair analysis can be done in chronic poisoning both in the living and in the dead. First toxic substances to the analyzed in hair were heavy metals by means of atomic absorption spectroscopy to document the exposure in the past. French Emperor **Napoleon Bonaparte** [1769 – 1821] is now thought to have been poisoned by arsenic, because it has been detected in his hair. In **1979**, opiates were detected in hair of heroin users. In the living it is done for

(1) intentional or unintentional chronic poisoning in criminal cases

(2) as preemployment and employment screening

(3) doping control of banned substances

(4) investigation of chronic drug consumption

(5) gestational drug exposure

(6) environmental exposure to toxins and

(7) to demonstrate even a previous single dose administration in a very low amount.

(8) Segmental [syn, sectional, sequential] hair analysis can provide information

about the time course of the substance use or exposure [presuming hair growth of 0.4mm/day – **ch 3**]. Segments of hair closest to the scalp reflect the most recent exposure, while those farthest from the scalp reflect earlier blood concentrations. As one moves away from scalp, every 0.4mm represents one earlier day, thus enabling an exact dating of toxic exposure.

o. Saliva [oral fluid]

(1) **Advantages:**

(i) Easy collection

(ii) Less invasive than taking other samples [eg urine, blood]

(iii) Carries a smaller risk of spreading infection

(iv) Collection can be directly supervised without the intrusion of privacy".es the chance of adulterating or substituting the sample. Very useful in driving under the influence of alcohol and drugs cases, when evidence of intoxication is generally needed.

(v) Finding drugs in saliva is a better indication of recent use and possible impairment than detecting them in a urine sample.

(2) **Collection devices** – A number of commercial collection devices are available. However saliva can be collected in a small test tube simply by asking the patient to chew on chewing gums, parafilm, rubber etc. They do not affect testing of common drugs and poisons.

p. Sweat

Collected by wiping the skin surface with cotton, gauze or towel.

q. Urine

(1) Urine is often the sample of choice because

(i) Drugs present in higher concentration [it has 25% more water than an equal volume of blood]

(ii) Large volumes easy to collect

(iii) No pain or discomfort

(iv) Non-invasive. Because of these advantages, it has been called "**toxicologists' gold**".

(2) **Drawbacks:**

(i) If bladder contained urine before drug ingestion began, urine secreted during

drug consumptions will be diluted with plain urine present already. Can result in faulty estimates of blood concentrations.

(ii) Time lag – of about 30 minutes exists before equilibrium between blood and urine is reached. Collection of urine before this period causes faulty values.

(iii) Passive diffusion – Most drugs would pass through the lining of bladder in either direction both in life and after death, depending on the relative concentration of alcohol in blood and urine.

(3) **Method of collection:**

(i) Analysis of 2 urine samples is preferable.

(ii) 1st sample should be taken immediately after the victim arrives, the bladder being completely emptied.

(iii) The 2nd sample should be taken 30 min later.

(iv) The conc of poison [eg alcohol] in the 2nd specimen reflects the blood poison level during the "interspecimen interval".

(v) The difference in poison concentrations in the two samples indicates whether the subject was in absorptive phase, at its peak or in the elimination phase. [**Ch 5**"method of collection in the dead].

r. X-Ray

Useful in radio-opaque ingestions [**Table 22**].

B. In the Dead

Diagnosis of poisoning in the dead is done mainly by five points (**Table 23**).

Table 22: A select list of radiopaque drugs and poisons [with **Memory Aid 11**]

1st Mnemonic – CHIPS

1. **C**hloral Hydrate
2. **C**alcium carbonate
3. **C**ocaine packets
4. **H**eavy metals [Iron is listed separately also]
5. **H**alogenated hydrocarbons [eg carbon tetrachloride, chloroform, methylchloroform, methylene chloride, tetrachloroethylene, trichloroethylene]. The radio-opacity seems to be strictly correlated to the ratio:

$$\frac{\text{Number of Chlorine atoms}}{\text{Molecular weight}}$$

6. **I**ron
7. **P**henothiazines, **P**hosphorus, **P**otassium, **P**lay-Doh [a modeling compound used by young children for art and craft projects]
8. Slow release drugs, Sodium chloride

2nd Mnemonic – COINS

C → **S**ame as above

O → **O**piate packets

I → **S**ame as above

N → **N**euroleptic agents (This is the same as Phenothiazines in the previous mnemonic)

S → **S**ame as above

3rd Mnemonic – CHIPPED

CHIPP → Same as above

E → Enteric-coated pills [same as slow release drugs]

D → Dental amalgam

Table 23: Diagnosis of poisoning in the dead

(1) Clinical history (2) PM appearances (3) Laboratory Analysis (4) Experiments on animals (5) Moral and Circumstantial evidence

1. Clinical history

Full clinical history should be taken from relatives, people present with the victim at the time of incidence, investigating officer etc. A record should be made of the drugs, medications to which the deceased had access. Visit to the scene may give important clues in the form of empty bottles, foils of drugs etc.

2. PM appearances

a. External

i. Stains

(1) Clothes, corners of mouth, or other surface of the body may show **poison stains**, **marks of vomit** or **feces**.

(2) **Color changes in the gastric mucosa** can provide a hint regarding poison.

ii. PM staining

For various colors of pm staining and associated poisons, please see **ch 9**.

iii. Smell

iv. Natural orifices

Mouth, nostrils, rectum and vagina may show the presence of poisonous material or the signs of it having been used.

v. Skin

Should be examined for

(1) Alopecia

(2) Bullae

(3) **Injection marks**

(4) **Jaundice**

(5) **hyperkeratosis** and **pigmentation**. [For interpretation of findings, please see above – Diag of poisoning in the living].

vi. Marks of violence

Abrasions, bruises, wounds may suggest violence in addition of poisoning.

b. Internal

All organs must be examined and all contents preserved.

i. Smell

(1) Usually the smell is obvious even **before opening** the body.

(2) If smell not obvious, ***skull*** should be opened first to note smell. If other cavities are opened first, smell may be masked. In some cases, opening the body merely makes the smell more obvious [as in alcohol]

(3) **Table 24** gives some common smells associated with poisons.

Table 24: Poisons that can be detected by smell [odor]

S.No	Smell	Poisons causing
1.	Acrid	Chloral hydrate, paraldehyde
2.	Bitter almonds	Cyanide
3.	Burnt rope	Marijuana (Cannabis)
4.	Carrots	Water hemlock (cicutoxin)
5.	Disinfectants (hospital odor)	Carbolic acid, creosote
6.	Garlic	Arsenic, arsine gas, dimethyl sulfoxide (DMSO), OP, phosphorus, selenium, tellurium,
7.	Glue	Toluene, other solvents
8.	Kerosene	OP [Both garlic like and kerosene smells have been described in OP]
9.	Mothballs	Camphor, Naphthalene, paradichlorobenzene
10.	Peanuts	N-3-pyridylmethyl-N-4-nitrophenylurea (VACOR rat poison)
11.	Pears	Chloral hydrate, paraldehyde
12.	Rotten eggs	Carbon disulfide, dimercaptosuccinic acid (DMSA), disulfiram, H_2S, Mercaptans, N-acetylcysteine
13.	Rotten fish (musty)	AlP, Zinc phosphide
14.	Sweet, fruity or apple like	Acetone, chloroform, ethanol, isopropanol, lacquer, nail polish remover, nitrites
15.	Shoe polish	Nitrobenzene
16.	Stale tobacco	Nicotine
17.	Vinegar	Acetic acid
18.	Wintergreen	Methyl salicylate

ii. Mouth, throat and associated structures

(1) **Mouth, throat and tongue** – Examine for any evidence of inflammation,

erosion, ulceration or staining. Causes of oral ulceration are same as those causing esophageal ulcerations [please see below]

(2) **Pharynx:**

(i) Corrosion and ulceration – causes same as esophageal ulceration below

(ii) Areas of necrosis – seen in drugs causing agranulocytosis [Amidopyrine, barbiturates, dinitrophenol, sulphonamides and thiouracil].

iii. Esophagus

(1) **Softening and desquamation of mucosa** – produced by alkalis [acids produce more destruction in the stomach]

(2) **Ulceration:** (i) Cantharidin (ii) corrosives (iii) Iodine (iv) Mercuric chloride (v) Paraquat and diquat

(3) **Perforation** – in (i) several corrosive ingestions (ii) swallowed foreign bodies [e.g. button batteries and coins].

iv. The duodenum

(1) **Strong acid reaction** – in duodenum indicates acid ingestion [has more value than a similar reaction in stomach]

(2) **Normal duodenum** – Rules out ingestion of strong corrosives

(3) **Perforation** – of duodenum usually caused by alkalis [e.g. NaOH].

v. Respiratory tract

(1) **Edema of glottis**, congestion and desquamation of the mucus membrane of trachea and bronchi – in corrosive poisoning, especially if corrosive has entered respiratory tract

(2) **Non specific congestion and edema of lungs** – in several poisonings, especially gaseous poisons.

vi. Stomach

Poisons tend to collect in the rugae and crypts of the mucosa. These may have to be scraped with the blunt end of knife and sent for chemical analysis. Following points are of importance.

(a) Drugs secreted in the stomach

Please see above under the heading “Fate of poisons in the body”.

(b) Hyperemia

(1) **Cardiac end and greater curvature involved** - Hyperemia of the mucus membrane caused by irritants is marked at cardiac end and greater curvature. Very rarely at pyloric end.

(2) **Color and extent** – Usually patchy and of a deep crimson color

(3) **Mucus membrane** – (a) covered with sticky secretion (b) shows small hemorrhagic foci

(4) **Ridges** – involved more

(5) **Other conditions in which hyperemia is seen** - (a) asphyxia (b) during digestion (c) disease. In these conditions, hyperemia would be uniformly spread over whole surface and not in patches

(6) **Putrefaction** – masks hyperemia. Histology and toxicology would be helpful. **Table 25** shows colors other than hyperemic red seen in poisoning.

(c) Softening

(1) **Softening seen in –** corrosives, especially alkaline corrosives. Areas involved are (a) mouth (b) throat (c) esophagus (d) cardiac end and (e) greater curvature of stomach

(2) **Disease** – If softening is due to disease, it would be limited to the organ affected, e.g. stomach only

(3) **Putrefaction** – Softening due to putrefaction (a) begins in most dependent parts (b) affects all coats of the stomach (c) softened patch is not surrounded by inflammation.

Table 25: Color changes in the mucus membrane of the stomach in various poisonings

S.No	Color	Poison
1.	Black, charred	H_2SO_4, HCl, Acetic acid, Oxalic acid
2.	Blue	Amytal capsule, copper sulfate
3.	Brown	Cresols
4.	Buff white	Carbolic acid
5.	Cherry red	CO
6.	Congested	Irritant poisoning, e.g. Phosphorus
7.	Green	Ferrous sulfate, fruit juice, food stains, bile
8.	Pink	Soneryl
9.	Slate gray	Mercury
10.	White particles	Arsenic
11.	Yellow	HNO_3

(d) Ulcers

(1) Liquids generally pass from esophagus to the intestine, via a small ribbon like passage along the lesser curvature of the stomach (**Fig 31.2**). This passage has been called "***Magenstrasse***" [German for "stomach street"], and it constitutes longitudinal mucosal folds along with liquids travel so easily that they may pass into the intestines within 10 minutes.

(2) Because liquids (including corrosives) preferentially take this path, ulceration is usually found along the ***lesser curvature***, and the stomach must be opened along the ***greater curvature***, so these ulcerations can be examined without cutting through them.

(3) **Ulceration beyond the pylorus** – usually due to natural disease and not due to corrosives

(4) **Table 26** shows important differences between ulcerations due to corrosives and disease.

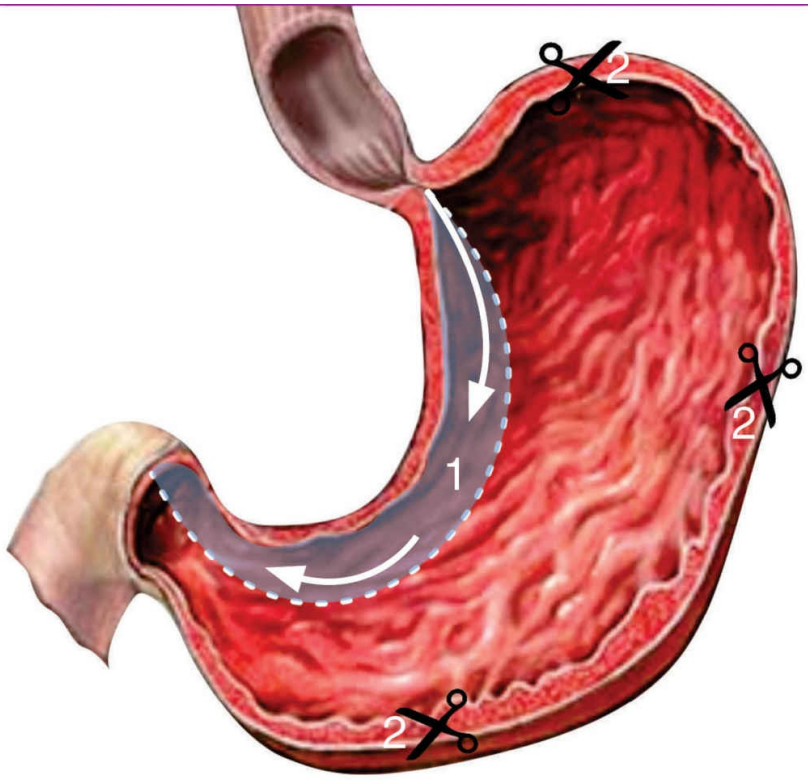


Fig. 31.2: (1) *Magenstrasse* or "stomach street". Poisons take this route to pass from esophagus to duodenum. To preserve 'pathologies' in this route, stomach must be opened along greater curvature (2).

Table 26: Differences in ulcerations due to corrosives and those due to disease

S.No.	Feature/Criteria	Ulceration due to corrosives	Ulceration due to disease [eg chronic gastric ulcer]
1.	Site	Lesser curvature	Anywhere
2.	Margins	Ill-defined, thin, friable	Well-defined, thick, indurated
3.	Surrounding mucosa	Softened, inflamed	Not so
4.	Adhesions to surrounding structures	Absent	Present

(e) Perforation

(1) Perforation is seen in corrosives [both acids and alkalis]

(2) Commonest with sulphuric acid [Please also see **ch 33** "Corrosives"]

(3) Rare with irritant poisons

(4) **Table 27** shows differences in perforation due to corrosives and due to pm autolysis.

Table 27: Differences in perforation due to corrosives and due to pm autolysis

S.No.	Feature/ Criteria	Perforation due to corrosives	Performation due to postmortem autolysis
1.	Appearance	Large, irregular, pulpy, edges rough	Oval, rounded, edges punched out
2.	Site	All along the GIT where corrosive came in contact, e.g. tongue, palate, esophagus, stomach etc	Confined to stomach. Commonly at the cardiac end
3.	Surrounding mucus membrane	Inflamed	Softened and gelatinous
4.	Peritonitis	Present	Absent

(f) Stomach wall

Examine - for fragments of poison adherent to it, e.g. (i) cantharides (ii) capsule fragments (iii) leaves or fruit fragments (iv) powdered poisons and (v) starch from tablets.

(g) Contents of the stomach

Note: (i) Color (ii) Volume (iii) Nature of contents [including food]. Contents of significance may include (a) Capsules (b) Foreign bodies [e.g. glass particles, nails and pins etc] (c) Leaves (d) other plant matter and (e) Seeds etc. Cells from plant material may be examined by a botanist [forensic botanist] to identity the plant. Plant cells retain their characteristic shape, dimensions, surface ornamentation and other characteristics in the stomach. They can also be identified from the vomit.

vii. Intestines

(1) C**olitis** – Colitis of ascending and transverse colon, similar to diphtheric colitis or enteritis of acute bacillary dysentery is seen in mercury poisoning.

(2) **Normal GIT** – Rules out poisoning by corrosives, arsenic, mercury and phenol.

viii. Liver

Hepatotoxic poisons – Please see above – "Classification of Poisons according to affected target organ".

ix. Kidneys

Nephrotoxic poisons – Please see above – "Classifi-cation of Poisons according to affected target organ".

x. Heart

Subendocardial hemorrhages – in left ventricle seen in poisoning with arsenic, antimony, phosphorus and viper bite.

xi. Genitourinary system

Criminal abortion – vagina and uterus of females must be examined for inflammation, corrosion etc. These organs must be sent for chemical analysis.

3. Laboratory analysis

a. Collection of specimens at autopsy

Please see **ch 5** – Collection of specimens.

b. Concentration of poison

(1) **In the living -** Concentration of poison is highest in the **portal blood** [because this is the vein carrying ingested substances]

(2) **In the dead** – Concentration will vary. Causes (i) Uneven destruction of poison by enzymatic and microbiological activity at different sites (ii) Postmortem diffusion [please see **ch 5**] from sites of higher concentration.

c. Precautions in the Dead

(1) Postmortem Diffusion - [**ch 5**]

(2) **Poisons which appear in the body due to putrefaction** - [**ch 9**]

d. Separation of poison from tissue

The first stage of the analytical process involves separation of the drug or compound of interest from the biological matrix in which it is contained.

4. Experiments on animals

(1) **Experiments** are performed on animals with the poison recovered from body as well as from the possession of the accused.

(2) **Not an ideal test** - Because animals may behave differently to poisons than humans.

(i) Alcohol - Atrial fibrillation occurs in humans [Holiday heart syndrome] and rabbits, but not in dogs.

(ii) Atropine - Pigs, goats, sheep, and rabbits are insusceptible to atropine [belladonna, Hyoscyamus and stramonium]. Horses and oxen are somewhat more susceptible, while cats, dogs, and birds are sensitive to belladonna.

(iii) Carbon monoxide - Canaries and mice are excessively sensitive to CO and methane [please see **ch 44** for details]

(iv) Opium -Pigeons are not affected by opium

(v) Cats and dogs are affected by poisons much in the same way as man. These are the best animals to conduct experiments on.

5. Moral and circumstantial evidence

This evidence consists of

1. Motive
2. Possible gain to accused from poisoning his victim
3. Evidence of witnesses [shopkeepers etc] about recent purchase of poison [purchase of rat poison etc]
4. Behavior of the accused before and after commission of offence
5. Recovery of poison from possession of accused
6. Relationship between accused and victim.

VII. DUTIES OF A MEDICAL PRACTITIONER IN A CASE OF

SUSPECTED POISONING

Duties of a medical practitioner in suspected cases of poisoning are: (1) Medical (2) Legal.

A. Medical

Includes treatment of the patient (see next chapter for details). If patient is serious, medical duties will take precedence over legal duties.

B. Legal

1. Information to the police

Information to the police must be given in ***ALL cases of poisoning*** whether ***suicidal***, ***homicidal*** or ***accidental***. ***This applies to both government and private doctors***

2. At the hospital

(1) **Routine body fluids** – e.g. gastric lavage, blood, saliva, sweat, urine, vomit, feces, milk etc must be preserved and handed over to the police [please see above - Diagnosis of poisoning " in the living " lab investigations. Also **ch 5** – "Medicolegal autopsy", for an exclusive table enumerating all possible body fluids to be sampled].

(2) **Clothes:**

(i) **Soiled** [with vomit, feces etc] etc should be preserved, sealed and handed over to the police to be sent for chemical examination.

(ii) **Unsoiled clothes** – especially underwear and other undergarments should also be sealed and handed over to police. They absorb sweat from which poison may be detected, especially drugs of abuse.

3. Arrangement for dying declaration

If the doctor feels that the patient may not live, he must arrange for dying declaration.

4. Information

If the case is of food poisoning, public health authorities must be notified.

32. General Principles of Management

I. INTRODUCTION

Treatment of poisoning follows following broad principles **(Table 1)**.

Table 1: Broad guidelines to follow in treatment of poisoning

1. Immediate resuscitative measures
2. Removal of unabsorbed poison from the body
3. Removal of absorbed poison from the body
4. Administration of antidotes
5. Symptomatic Treatment
6. Adequate follow-up

II. IMMEDIATE RESUSCITATIVE MEASURES

1. Remove patient from poisonous environment (removal from scene of fire in acute CO poisoning).

Memory Aid 1: ABCDE of resuscitation

Airway, **B**reathing, **C**irculation, **D**epression of CNS, **E**valuation of patient. All are best done in an **ICU. ABCDE method** was first used by **Scandinavian physicians** in **mid-1940s** in barbiturate poisoning, with dramatic success. Looking after the ABCD is now also known as the **Scandinavian method of treatment** (especially in **barbiturates**).

Additional measures:

D also stands for **decontamination [**Ocular—copious saline lavage; Skin—copious water, then soap and water; GIT—emesis, lavage**], dextrose, dialysis, disability evaluation** and **drugs.** An additional mnemonic is **DEFG** "**D**on't **E**ver **F**orget the **G**lucose.

A. Airway

(1) **Assessment by** (i) Assess protective reflexes (ii) Monitor oxygen saturation continuously.

(2) **If inadequate** (i) open up and clean airway [of secretions, vomit etc] (ii) Endotracheal intubation (iii) Tracheostomy if necessary.

B. Breathing

(1) **Assessment by** (i) noting if there is adequate tidal volume. (ii) Perform arterial blood gases (ABG) (iii) Perform capnography [monitoring of the partial pressure of CO_2 or End-Tidal CO_2 conc ($ETCO_2$)]

(2) If inadequate: Supplemental O_2 therapy [by a mask or by endotracheal intubation]. Maintain tidal vol at 10-15mL/kg.

C. Circulation

(1) **Assessment by** (i) Check for presence or absence of radial, femoral, and carotid pulses. (ii) Start continuous cardiac monitoring [12-lead ECG]

(2) **If inadequate** (i) Secure IV access (ii) IV fluid administration (iii) Antidysrrhythmic therapy (iv) Vasoactive agents (v) Consider atropine, $NaHCO_3$, Mg (vi) CPR – Chest compressions.

D. Depression of CNS

(i) Turn unconscious patient to one side [prevents tongue blocking the throat, allows fluids to gravitate out]. (ii) Depression of CNS is best evaluated according to **Glasgow Coma Scale** (GCS) **[Table 2]** or **Matthew-Lawson Scale** (MLS) **[Table 3]**. (iii) See pupillary size, reactivity.

Table 2: Glasgow coma scale

I. **Best eye response (E)**
1. No eye opening
2. Eye opening in response to pain
3. Eye opening to speech
4. Eyes opening spontaneously

II. **Best verbal response (V)**
1. No verbal response
2. Incomprehensible sounds (moaning)
3. Inappropriate words
4. Confused
5. Oriented

III. **Best motor response (M)**
1. No motor response
2. [E]xtension to pain (dec[e]rebrate rigidity). Injury is at the level of brainstem.

Memory Aid 2: Best motor response in Glasgow coma scale-1

[Extens]ion means more [Extens]ive injury.

3. Abnormal flexion to pain (decorticate rigidity). Injury is at the level of corticospinal tracts.

Memory Aid 3: Best motor response in Glasgow coma scale-2

De[cor]ticate ↔ [Cor]ticospinal. Patient points towards the [COR]e of body!

4. *Withdrawal to pain* - Pinch nail bed on finger to cause pain. [Other methods (less acceptable to some) - pinching trapezius muscle, rubbing knuckles on patient's sternum, pressing supraorbital ridges]. Patient tries to pull the finger away.
5. *Localizes to pain* - Pinch nail bed on finger to cause pain. Patient uses other hand to reach over and tries to stop ['localizing']. *Patient moves towards the location of pain.*
6. Obeys commands

Interpretation: Individual elements as well as the sum of the score are important. Hence, the score is expressed in the form **"GCS 7 = E1 V3 M3 at 10:40"**.

Poisoning is classified as:

- **Severe**, with GCS ≤8 [next integer of ½ of maximum possible score]. Typically, patients with this score from any cause are considered in coma.
- **Moderate**, GCS 9 - 12
- **Minor**, GCS ≥ 13.

Memory Aid 4: Criteria used in Glasgow coma scale

EVM [[E]lectronic [V]oting [M]achine = [E]ye, [V]erbal, [M]otor]. Number of criteria **4, 5, 6** in the same order. Maximum total score can be **15**.

Table 3: Matthew-Lawson Scale of determining severity of Coma

Grade 0:	Fully conscious, alert
Grade 1:	Drowsy but responding to verbal commands
Grade 2:	Maximum response to minimum [pinching earlobe] painful stimuli
Grade 3:	Minimum response to maximum [sternal rub] painful stimuli
Grade 4:	No response to painful stimuli. Loss of all reflexes, including Corneal, laryngeal, pharyngeal

E. Evaluation of Patient

Not an immediate resuscitative measure, yet should be done as soon as the patient is stabilized

(1) **Breath sounds - Auscultate** (and **re-auscultate**), particularly after IV fluids have been given. Helps to diagnose **pulmonary edema**, **acute lung injury** and **aspiration pneumonia**. If crackles and rhonchi are present, especially if coupled with an abnormal breath odor (such as seen in hydrocarbons), it may point to pulmonary etiology rather than cardiac etiology. This is important because administration of cardiac medications may be dangerous in case of pulmonary etiology

(2) **Heart sounds:** (a) **Murmurs** - in an injection drug user, especially when accompanied by fever indicate **bacterial endocarditis** (b) **Dysrhythmias** – suggest overdose of cardiac poisons (e.g. digoxin, β-adrenergic antagonists)

(3) **Abdominal examination:** (a) **Enlarged liver** – Hepatoxic poisons, chronic alcoholism (b) **Bowel sounds** – to diagnose or exclude cholinergic/anticholinergic toxicity. Helps to decide if purgatives will be useful, e.g. Lack of bowel sounds " anticholinergic poisoning " purgatives may not be very useful.

(4) **Examination of extremities. E** also stands for **Elimination of poison, ECG** and **Expose** [e.g. in cases of toxic gases]**.**

F. Other Measures

(1) **Additional** – Pl see memory aid below.

Memory Aid 5: Other resuscitative measures in poisoning [besides the ones given in Memory Aid 1]

Other measures are " FGHIJKLMNOP. All of them are not necessarily immediate resuscitative measures, but are important measures nevertheless.

F-Fluid management, also **Fahrenheit** (note temperature);

G-Get vital signs, Gastric lavage;

H-Head of toe examination;

I-Imaging [***radiopaque poisons*** "***ch 31***], **Interventions, Inspect back;**

J-Jaundice, attend to (for hepatotoxic poisons);

K-Kidney failure, attend to;

L-Laboratory tests;

M-Magnesium hydroxide (cathartic);

N-Narcosis, attend to;

O – Observe, Oxygen

P- Pulse.

Most patients – whether conscious or unconscious- would recover by these measures alone.

(2) **Shift patient to a proper environment:** Dark and quiet room – (a) because of photophobia – Datura, Methyl alcohol (b) because of hyperexcitability to light and sound – strychnine.

III. REMOVAL OF UNABSORBED POISON FROM THE BODY

A. Inhaled Poisons

In case of inhaled poisons [eg CO, H_2S, Cl_2 etc]

(1) **Fresh air** - Remove patient into fresh air.

(2) **Air passages** - Clear air passages of mucus by postural drainage or aspiration.

(3) **Endotracheal intubation** if necessary.

(4) **Artificial respiration** and **O_2** [6-8 L/min]

(5) If **bronchospasm** present [as in irritant gases]:

(i) **Aminophylline** 250-500 mg, slow IV

(ii) Inhaled **b**2 agonist

(iii) Oral or parenteral corticosteroids
(6) **Diuretics** – if pulmonary edema is threatening.
(7) Monitor for respiratory distress.
(8) If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis.

B. Injected Poisons

In case of injected poisons (insect and snake bite, scorpion stings, drug abuse, iatrogenic injection):

(1) **Reassurance** - Calm the patient. Reassure. Sedatives may be required.
(2) **ABCDE** - Monitoring patient's ABCs (please see above)
(3) **Shock** - Treat shock
(4) **Allergies** - Observe patient for allergic reaction. Treat anaphylactic reactions on standard lines.
(5) **Remove sting** – In case of bee, wasp, scorpion or centipede stings.
(6) **Local vasoconstriction** – adrenaline injection around injected area. Apply ice locally or immerse injected part (finger etc) in cold water [10°C]
(7) Tight ligature above injection site, wound excision, sucking out poison with mouth, local neutralization of poison with suitable chemicals are **no more recommended** now.

C. Contact Poisons

1. Eye

(1) Irrigate exposed eyes with copious amounts of room temperature water for at least 15 min
(2) Remove contact lenses if present.

2. Skin

(1) Remove contaminated clothing and wash exposed area thoroughly with soap and water [for about 30 minutes]. Same procedure if poison applied to wound, or is inserted into urinary bladder, rectum or vagina
(2) Remove jewellery if present.

D. Ingested Poisons

1. Emesis

An ***emetic*** is a substance that induces vomiting when administered orally or by injection. Emesis is virtually obsolete now. May come in handy when nothing else is available.

Salient features:

(1) **When used:**

(i) if there is difficulty in obtaining or using stomach tube

(ii) if vomiting center is still functional and responsive to emetics

(2) **Position** - During induction, the person must either be sitting or lying on his side, which the head dependent; otherwise there is danger of inhaling gastric contents

(3) **Mechanically induced** – by tickling throat [patient's own fingers, doctor's fingers, feather, spoon handle, wooden tongue depressor]. Usually ineffective.

(4) **Pharmacologically induced** – All emetics considered obsolete now by many authorities. Last to fall out of favor was **ipecac** in late **1990s**. Many others however continue to use it; for this reason emetics find mention in subsequent chapters. (i) Ipecac – (a) Source – Root of a small shrub Cephaelis ipecacuanha [syn Cephaelis acuminata]. Native to Brazil; In India grows in W. Bengal. (b) Preparations – (I) ipecacuanha powder 1-2 g (II) Syrup of ipecac – 30 ml [adults]; 15 ml [children 1-12 y]; 10 ml [9-12 m]; 5 ml [6-9 m]. Administer several glasses of water afterwards. Induces vomiting in 90-95% cases within 30 min. Dose is repeated if vomiting does not occur by this time. (c) Active principles – Cephaeline, Emetine, Psychotrine [traces] (d) Mechanism - (I) Central – stimulation of vomiting center (II) Peripheral – stimulation of sensory receptors in GIT. (e) Current status – Although very popular up to the final years of 20^{th} c, no more recommended now. **Reasons** (I) No evidence of clinical benefit (II) Poison removed is highly variable and diminishes with time. (III) Reduces effectiveness of established and useful therapies [activated charcoal, oral antidotes, Whole Bowel Irrigation]. (IV) Adverse effects [cardiotoxicity, complications of emesis] (V) May be harmful if administered to a patient who has a .ed level or impending loss of consciousness. (VI) abuse by bulimics (VII) use as an agent for Munchausen's syndrome by proxy. (ii) **Obsolete since long** (a) Apomorphine - Not effective

orally; must be given parenterally [s/c] (b) Copper sulfate (c) Hand dishwashing liquid detergent. Its use was recommended only when time was critical, and nothing else was available. (d) mustard water (e) Saturated sol^{n} of common salt – causes fatal hypernatremia (f) Tartar emetic (g) Zinc sulphate.

a. Contraindications

(1) Same as those for gastric lavage [eg hydrocarbons having a high aspiration potential, corrosives]
(2) Active or prior vomiting [eg nicotine]
(3) Ingestions that can alter mental status [coma]
(4) Exposure to a toxin with more pulmonary toxicity from inhalation than toxicity from GI absorption [e.g., hydrocarbons]
(5) Poisons inducing seizures.
(6) Tricyclic antidepressants, emesis in contraindicated [although gastric lavage may be done]
(7) If need for advanced life support within 60 min is anticipated.
(8) Debilitated, elderly patients
(9) Medical conditions that may be further compromised by induction of emesis [heart disease, pregnancy].

b. Adverse effects

(1) Aspiration
(2) Boerhaave syndrome [Esophageal rupture]
(3) Coma
(4) intractable vomiting
(5) Mallory-Weiss syndrome [bleeding from tears in the mucosa at the junction of the stomach and esophagus]
(6) Seizures.

2. Gastric lavage

a. Disadvantages

(1) May delay administration of activated charcoal, which is a more useful procedure
(2) May push tablets further into the GI tract. In general, gastric lavage is better

than emesis because of more discomfort caused to the patient in vomiting.

b. Procedure

Tubes used - are: (a) Ewald's tube [devised by **Carl Ewald** (1845 - 1915) a German gastroenterologist] **[Fig 32.1]**, (b) Boas tube [devised by **Ismar Boas** (1858 –1938) a German gastroenterologist, and quite similar to Ewald's tube], (c) Edlich tube **[Fig 32.2]** [devised by Richard Edlich (1939)]or (d) a Lavacuator **[Fig 32.3]**.

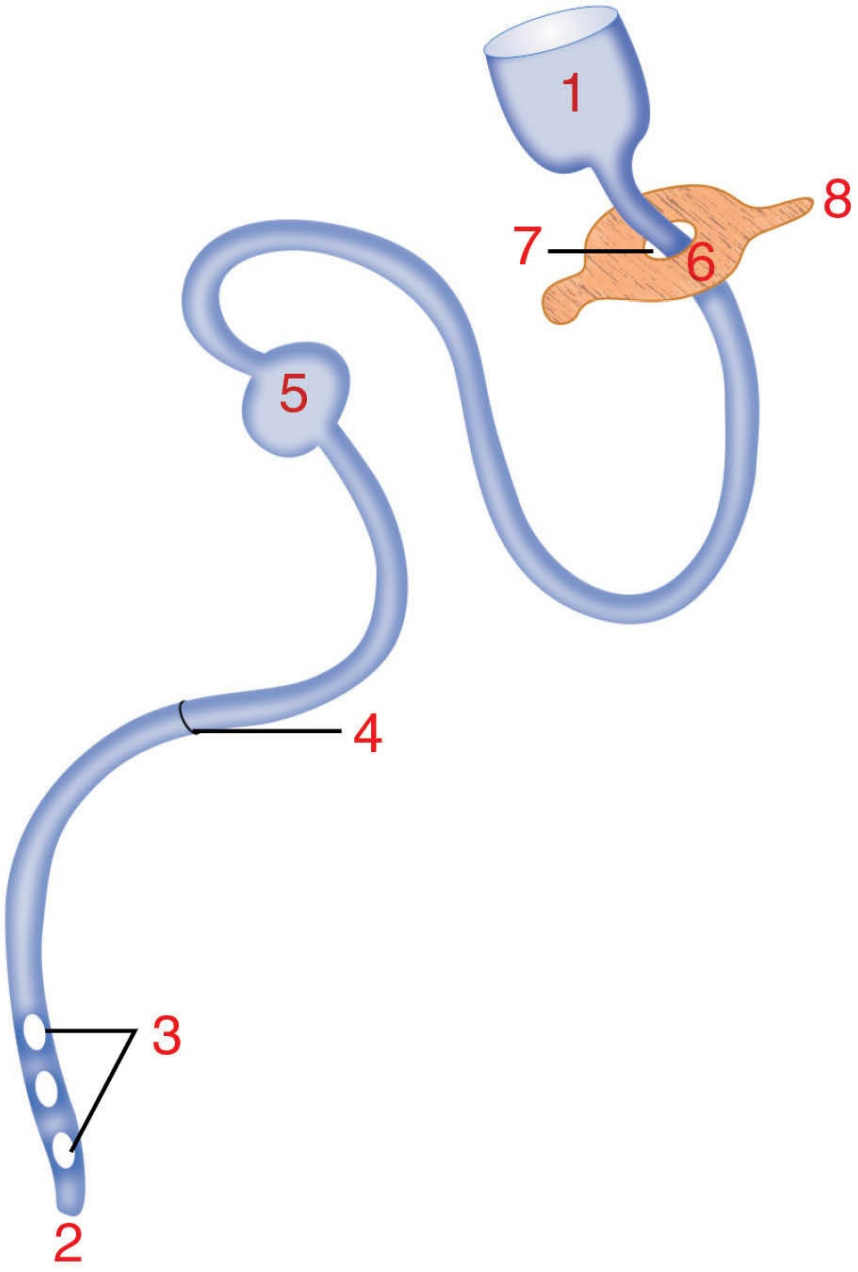


Fig. 32.1: Gastric lavage (Ewald's) tube. Please correlate nos (given in red). with text.

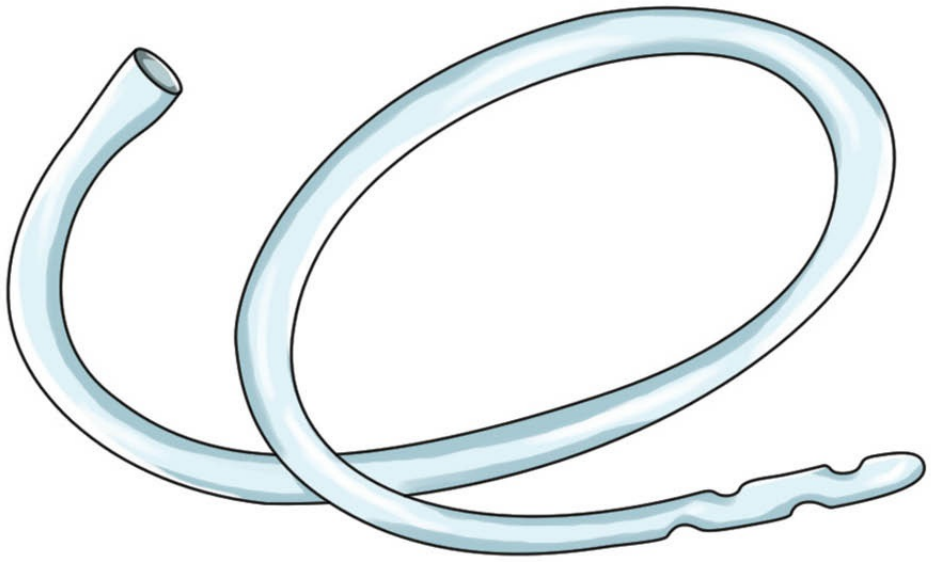


Fig. 32.2: An Edlich tube

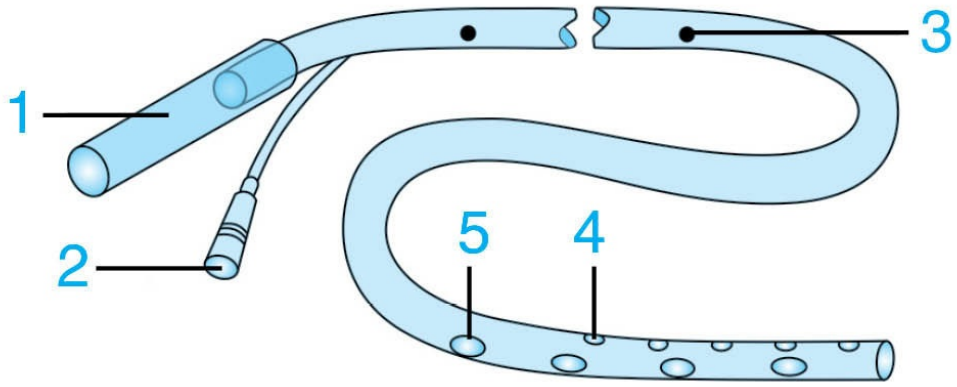


Fig. 32.3: A Lavacuator
(1) Suction connector
(2) Irrigation/vent connector
(3) Depth marks (4) Irrigation/ vent eyes (5) Suction eyes

Ewald's tube

(i) **Diameter and length** - A standard Ewald's tube is 1 cm in diameter and 150 cm in length, with a funnel (1) attached at one end. (ii) The **other end** is rounded (2) with lateral openings (3) (iii) **Mark** - There is a mark (4) 100 cm from the funnel end and 50 cm from distal end (2) indicating the level up to which the tube should be introduced. (iv) **Suction bulb** - At about the midpart of the tube, there is a suction bulb (5) which is used to pump out the stomach contents. (v) A **wooden mouth gag** (6) comes along with it. It has a hole (7) in the middle to allow for the passage of the tube through it. One end of the gag is pointed (8) so that it can forcefully be inserted by the side of the mouth in uncooperative patients (vi) **Size** – Comes in different sizes, which are measured by F scale. "F" stands for French scale, each unit on the French scale being equal to 0.3 mm. The largest orogastric lavage tube that can reasonably be passed should be used

(1) **Position of the patient** - The patient should be on his left side or prone, with head hanging over the edge of the bed, and face down. Patient is kept in

Trendelenburg position [mouth is at a lower than feet to prevent regurgitation of fluid leaking out through the sides of tube]. Face is supported by an assistant. **Trendelenburg position** was first advocated by the German surgeon **Friedrich Trendelenburg** [1844 –1924] in the **1860s**. In the classic Trendelenburg position the **feet are higher than the head** by **15-30°**.

(2) **Introduction of the tube** - (a) If dentures are present they must be removed (b) wooden mouth gag is placed in between teeth, so teeth do not bite tube (c) end of tube is lubricated with glycerine, olive or sweet oil or paraffin (d) Tongue is depressed with tongue depressor [or two fingers] (e) Lubricated tube slowly passed downwards through the pharynx and esophagus into the stomach, till the 50 cm mark is reached. (f) If there are no marks on the tube – Measure distance between bridge of nose and tip of xiphoid process. The tube should be passed for the same distance (g) Precautions – Do not use force.

(3) **Confirmation of the tube in stomach** – (a) Absence of coughing and of breath sounds in the funnel will confirm that the tube has not entered trachea (b) In case of doubt – insert free end of the tube, just below water surface. Air from stomach is usually expelled completely in 2-3 expirations. Air from lungs causes bubbling at each expiration.

(4) **Lavaging the stomach** – (a) Preserve first sample for chemical analysis:

(i) Pass about 250 cc of plain warm water through the funnel, held high above the head

(ii) When funnel is empty, pinch tube just below the funnel between finger and thumb

(iii) Lower it below the level of stomach

(iv) Release finger and thumb to relieve blockade

(v) contents will be emptied by siphon action

(vi) Preserve this sample for chemical analysis. This solution must be handed over to police in all cases of poisoning [suicidal, homicidal or accidental, and whether the doctor is govt or private] [Please also see **ch 31**, under the heading **"Information to police"**]. (b) Precautions – Stop the procedure, if there is any bleeding (c) Subsequent washings with lavage fluid - Normal saline (50 to 100 mL in young children and 150 to 200 mL in adolescents) is then introduced [or any other suitable lavage fluid - **Table 4**] and lavaged repeatedly.

(5) **Indication to stop lavage** – (a) Stop when the returning fluid is clear,

odorless, and is of the same nature as the fluid introduced. This indicates that there is no further interaction between the poison and antidote (b) In case of $KMnO_4$, the returning fluid is initially colorless, because $KMnO_4$ oxidizes poisons and in the process itself gets reduced to colorless compounds. Lavage should be stopped when the returning fluid has pink color (the color of normal $KMnO_4$).

(6) **Late lavage useful in:**

(i) Poisons causing .ed gastric motility or pyloric spasm – (a) Antihistamines (b) Barbiturates (c) morphine (d) Phenothiazines (e) Tricyclic antidepressants

(ii) Poisons which cake together forming a doughy mass [it sticks to stomach walls] (a) salicylates.

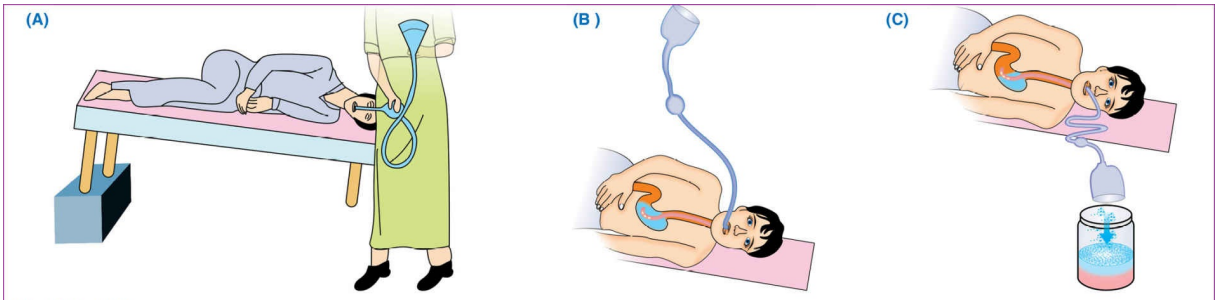


Fig. 32.4: (A) Position of patient during gastric lavage (Trendelenburg position). This can be achieved simply by putting some bricks under the lower end of the bed. **(B)** Indicates the position of lavage tube inside the stomach at start of procedure. Stomach is seen filled with lavage fluid (blue) **(C)** After introduction of lavage fluid, the tube is pinched between fingers and thumb, and funnel lowered below the level of stomach. All lavage fluid drains out by siphon action. The process may be repeated if more poison needs to be evacuated.

Table 4: Solutions used for gastric lavage against different poisons

S.No	Solution used	Poison
1.	Calcium gluconate, lactate and other calcium salts	Oxalates
2.	Castor oil with warm water	Carbolic acid (phenol)
3.	Copper sulphate [recommended earlier]	Phosphorus
4.	Desferrioxamine (2 g in 1 L of water)	Iron
5.	H_2O_2	CN
6.	Potassium iodide (1%)	General
7.	Potassium permanganate [$KMnO_4$) 1:5000	Oxidizable poisons [alkaloids, Phosphorus, salicylates]
8.	Saline	Any unknown poison
9.	Saturated lime water	General
10.	Sodium bicarbonate (5%)	General
11.	Sodium iodide (1%)	General
12.	Sodium thiosulfate (25%)	CN
13.	Starch solution	Iodine
14.	Tannic acid (4%)	General

c. Contraindications

Contraindications of gastric lavage are [A] **Absolute** (i.e. must not be done

under any circumstances) and [B] **Relative** (i.e. Should not be done normally, but may be done after taking precautions). [A] **Absolute:** (1) **Corrosives** - because of risk of perforation. Exceptions are organic acids like (i) Carbolic acid [stomach wall becomes thick and leathery] (ii) Oxalic acid and (iii) Acetic acid etc [if patient is seen early and corrosion of stomach wall is not great]. (2) **Nontoxic** ingestions (3) **Sharp and pointed material ingestions** – pins, needles, glass – risk of perforation[B] **Relative:** (1) Cardiac arrhythmias (2) Children (below toddler age group) " relative contraindication, because stomach wash tube is too thick. Can be done with Ryle's tube, or a number 10 to 12 French catheter (3) **Coma** " because airway protective reflexes are suppressed; may cause aspiration. Can be done with a cuffed endotracheal tube in place (4) **Convulsant poisonings** (e.g. strychnine) " because it can exacerbate convulsions. Convulsions need be controlled first (5) **Esophageal varices** – Look for accidental rupture (6) **Hemorrhagic diathesis** – may cause bleeding (7) **Hypothermia** " body temperature would need careful attention (8) **Kerosene poisoning** " aspiration of kerosene in the respiratory tract setting up fulminant chemical pneumonitis. Can be done with a cuffed endotracheal tube (9) **Late arrival of patient** – Generally after 3 hours, the recovery of poison is none or very little. Poisons in which late gastric lavage is useful (a) Poisons which cause pyloric spasm or reduce GIT motility: (i) Anticholinergics (ii) Morphine, (iii) Tricyclic antidepressants (b) Poisons which cake together forming a thick mass - (i) salicylates (10) ***Poison*** having an effective antidote (11) **Pregnancy** [advanced] "- intraabdominal pressure may be harmful for child (12) **Surgery** [recent] " Risk of opening up of sutures (internal or external) (13) **Volatile poisons** " eg gasoline, lighter fluid. Can cause aspiration pneuomonitis. Use cuffed endotracheal tube to prevent aspiration (14) **Vomiting** [severe] " Poison already expelled.

d. Complications

(1) Aspiration pneumonia [most dangerous. If it occurs, administer (a) hydrocortisone 3-4 mg/kg IV (b) O_2 (c) Broad spectrum antibiotics] (2) Bradycardia (3) Cardiac arrhythmias and cardiac asystole - Due to vagal stimulation and hypoxia during gastric lavage. (4) ECG changes – ST elevation (5) Epistaxis (6) Hypochloremia (7) Hyponatremia (8) Hypoxia and hypercapnia (9) Laryngospasm (10) Mallory-Weiss syndrome [esophageal tear] (11)

Mechanical injury to the throat, esophagus, and stomach (12) Metabolic alkalosis [**ch 31**] (13) Perforation (14) Water intoxication [**ch 45**]. Combative patients may be at greater risk of complications.

3. Cathartics

According to current thought, cathartics should never be considered part of routine management of poisoning. Following principles must be kept in mind

(1) **Poison adsorbable by AC** - If poison is adsorbable by activated charcoal (AC), a cathartic given with AC is as efficacious as AC alone

(2) **Poison *not* adsorbable by AC** - If poison is not adsorbable by AC, whole bowel irrigation (WBI) [please see below] is better than cathartics

(3) **When cathartics may be given** – When facilities and expertise for WBI are not available. In such cases, use if

(i) Sustained release preparations have been ingested

(ii) Poison not adsorbable to AC

(iii) large amounts of poison has been ingested

(iv) desorption from AC is a possibility (as with aspirin).

(4) **Cathartics used:**

(i) M**agnesium citrate** 6% sol^{n} 150-300 mL orally

(ii) **Sorbitol**. Given with the first dose of AC.

(5) **Cathartics not to be used:** (i) $MgSO_4$. May produce CNS depression in cases of renal failure.

4. Whole bowel irrigation (WBI)

Whole bowel irrigation (WBI) flushes out the entire GIT in 4-6 hours. It was originally developed to cleanse the large bowel before surgery or colonoscopy. Initially a solution of NaCl, KCl, and $NaHCO_3$ was used but it was absorbed by the body, leading to complications. This gave rise to specialized irrigation fluid (**polyethylene glycol**) that is not absorbed. Induces a liquid stool.

a. Procedure

WBI involves rapid administration of large volumes of **PolyEthylene Glycol-Electrolyte Lavage Solution** (PEG-ELS) **[GoLYTELY, CoLyte, MoviPrep]** via a nasogastric tube, while the patients sits on a toilet seat. The solution is administered at a rate of 500 mL/h in children and 2000 mL/h in adolescents and

adults. The procedure continues **until the rectal effluent is clear**. If patient vomits, infusion must be slowed down or an antiemetic given.

b. Indications

Should not be used routinely in poisoning situations. Usually reserved for patients

(1) who have ingested toxic doses of medications not adsorbed by activated charcoal (such as lithium, heavy metals)

(2) toxic ingestions of sustained-release or enteric-coated drugs or

(3) body packing/stuffing (cocaine body packer syndrome).

c. Contraindications

(1) Bowel perforation (2) Bowel obstruction (3) Clinically significant gastrointestinal hemorrhage (4) Ileus (5) Unprotected compromised airway (6) Hemodynamic instability (7) Uncontrollable intractable vomiting.

IV. REMOVAL OF ABSORBED POISON FROM THE BODY

A. Diaphoretics

(1) ***Diaphoretics*** are drugs that - sweating.

(2) It is controversial if they - poison elimination through sweat.

(3) Common diaphoretic agents are: (i) Alcohol (ii) antipyretics (iii) Blankets (iv) Hot beverages (tea, coffee, milk, lemonade) (v) Hot water bottles (vi) Pilocarpine nitrate (5 mg s/c) (vii) Salicylates.

B. Enhanced Renal Excretion

Large amounts of fluid are given (**forced diuresis**). Method is much like flowing buckets of water from top of stairs to clean them.

Dangers and pitfalls

Can cause **pulmonary** and **cerebral edema**.

C. Urine Alkalinization

[A] ***Urine alkalinization*** is a treatment regimen that - es poison elimination by

the administration of IV sodium bicarbonate to produce urine with a pH ≥ 7.5. [B] **Principle:** (1) ***Uncharged ions*** can freely pass through biological membranes, while ***charged ions*** cannot (**Fig 32.5**). (2) If a poison molecule is in uncharged form, whatever molecules are filtered by glomeruli, would diffuse back in the system. Poison molecules in charged form would be retained within the tubules. (3) Normally a drug in the urine is both in ionized and unionized forms. As pH of urine changes, the ratio between the two species changes. At its ***pKa***, the drug is 50% ionized (**Fig 32.6**).

(4) An acidic drug would be ionized more in an alkaline urine and vice-versa. (5) If we want an acidic drug not to cross the tubule (in order to - elimination), we must alkalinize the urine; reverse (acidification) should be done in case of alkaline drugs. [C] **Drugs/poisons in which urine alkalinization is useful**: (1) Barbiturates, eg. Phenobarbitone [**ch 40**] (2) Salicylates [**ch 45**] (3) 2-4 Dichlorophenoxyacetic acid [2-4D]- A herbicide [widely used by national Govts to destroy illegal poppy plants] (4) Mecoprop (MCPP) – A selective hormone type phenoxyherbicide [Please see more on this in chapter on agricultural poisons] (5) Chlorpropamide (6) Diflunisal (7) Fluoride (8) Methotrexate (9) Formate. (10) **During BAL therapy** – because BAL-metal complex dissociates in acidic urine (11) **In poisons causing rhabdomyolysis** [eg **nicotine**]-helps minimize risk of myoglobin-induced kidney damage.

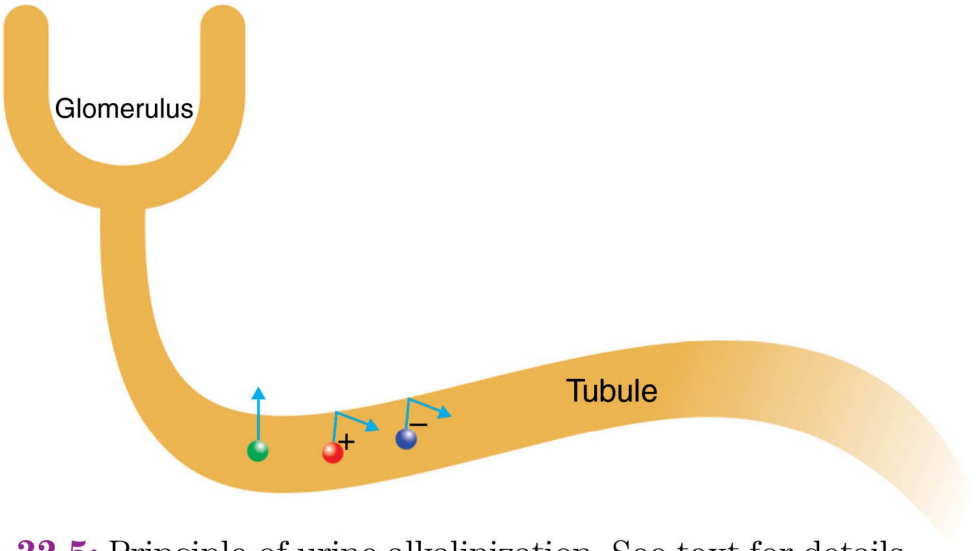


Fig. 32.5: Principle of urine alkalinization. See text for details.

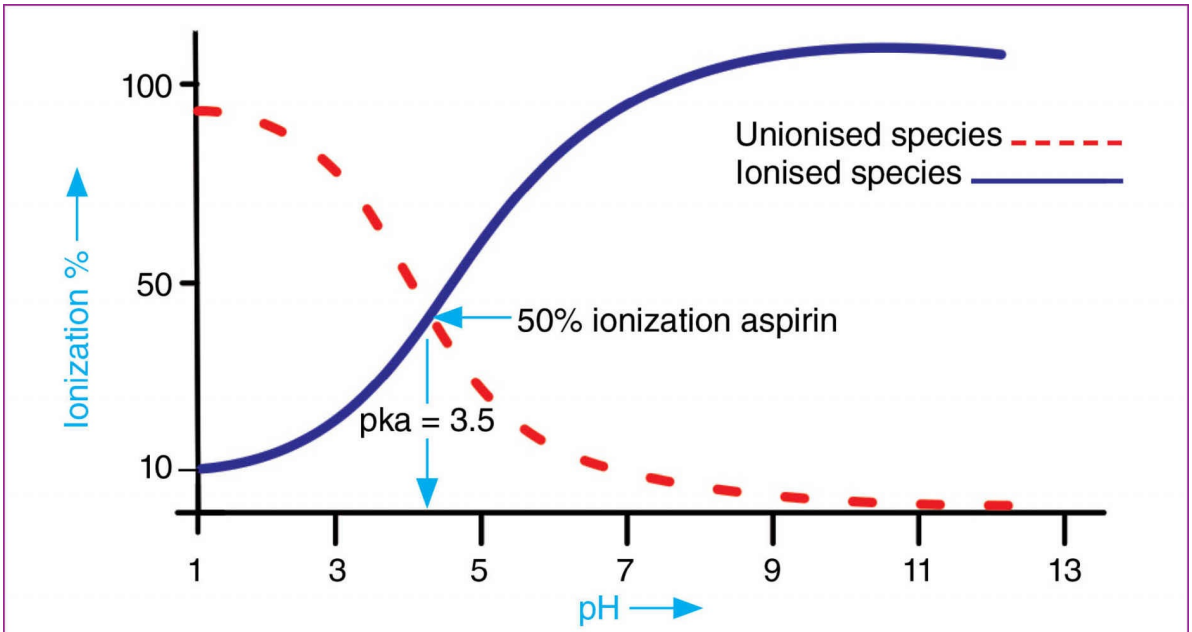


Fig. 32.6: At its pKa, 50% of the drug is ionized and 50% unionized

Memory Aid 6: Urine alkalinization is useful in

(A) Most imp

Bed **S**ide **D**ialysis in an **M C D** hospital

(1) **Barbiturates** (2) **Salicylates** (3) **2-4D** (4) **Mecoprop** (5) **Chlorpropamide** (6) **Diflunisal**

Even among these 6, top 3 are most imp for student.

(B) Others

Forensic **M**edicine **F**or **B**est **R**esident

(7) Fluoride (8) Methotrexate (9) Formate (10) BAL therapy (11) Rhabdomyolysis

[D] **Goal** – Normal pH of urine = 6.0. Goal in **urine alkalinization** is to achieve urine pH of ≥ 7.5. Goal in **urine acidification** is to achieve urine pH of = 5.0. [E] **How to achieve alkalinization** : (i) By giving $NaHCO_3$ 1-2 mEq/kg every 3-4 h. [F] **Urine output** – Normal urine output in a 60 kg man is 1 ml/kg/hr. In urine alkalinization it - s to 3-6 ml/kg/hr. [G] **Contraindications to urine alkalinization**: (1) Congestive heart failure (2) renal failure (3) cerebral edema.

D. Urine Acidification

Also known as **forced acidic diuresis**. Principle same as above.

Memory Aid 7: Urine acidification is useful in

(A) Most imp - MAP

(1) **M**ethadone

(2) **A**mphetamine

(3) **P**hencyclidine (PCP)

(B) Others – Comfortable **qu**estions are **simp**ly **n**ever a**s**ked

(4) **Co**caine

(5) **qu** inine

(6) **qu**inidine

(7) **symp**athomimetics

(8) **N**icotine [theoretically beneficial, but almost **never** carried out because of risks – **ch 43**]

(9) **S**trychnine

How to achieve acidification

By giving **IV ascorbic acid** or **ammonium chloride 1-2 g** every **4-6 h**.

E. Hemodialysis (HD)

Hemodialysis is a method for removing metabolic wastes as well as free water from the blood when the kidneys are in renal failure. Hemodialysis was first used in **1913** for the removal of salicylic acid in experimental poisoning, but was not tried clinically until **1950** when it was used for the treatment of **aspirin poisoning**. Since then it has been used on a considerable scale.

Salient features:

(1) Hemodialysis is one of three major renal replacement therapies, the other two being renal transplant and peritoneal dialysis.

(2) **Hemodialysis is useful** in:

Memory Aid 8: Hemodialysis is useful in

(1) **Mnemonic 1 – BLAST** [**B**:Barbiturates, Boric acid, Bromides; **L:**Lithium; **A**:Alcohols (methyl, ethyl, isopropyl etc); **S**:Salicylates, strychnine; **T**:Theophylline, thiocyanates]

(2) **Mnemonic 2 - I STUMBLE** [**I**sopropyl alcohol; **S**alicylates; **T**heophylline; **U**remia; **M**ethanol; **B**arbiturates; **L**ithium; **E**thylene glycol/Ethanol].

Memory Aid 9: Hemodialysis is not useful in

ABCD GOMP

A: Anticholinergics, antidepressants, antihistaminics

B: Belladona alkaloids, benzodiazepines

C: Chloroquine

D: Digitalis

G: Glutethimide

O: Opiates (morphine, heroin etc)

M: Methaqualone

P: Paracetamol, phenothiazines (antipsychotics)

F. Peritoneal Dialysis (PD)

Peritoneal dialysis (PD) is a procedure in which patient's peritoneum is used as a membrane across which toxins in the blood are exchanged.

Salient features:

(1) **Procedure** – The patient's peritoneal membrane (A) serves as the semipermeable barrier between the blood (in the peritoneal capillaries) and a balanced dialysate solution (B). The dialysate is introduced into the patient's peritoneal cavity from a solution bag (C) through a catheter (D) and allowed to dwell for a period of **hours.** A 3-way valve (E) allows the dialysate to go into the peritoneum and then drain out into the drainage bag (F). The osmotic gradient pulls **toxins** from the **intravascular space** into the **dialysate**.

(2) **Dialysate used** – For adults – 2L. For children <5 y – 200 ml

(3) **Effectiveness** – about 25% of that of hemodialysis

(4) **Poisons effectively removed by PD:** (i) Alcohols (ii) Barbiturates [long acting] (iii) Bromides (iv) Chloral hydrate (v) Lithium (vi) Mercury [inorganic] (vii) Quinidine (viii) Salicylates (ix) Sodium chlorate (x) Theophylline.

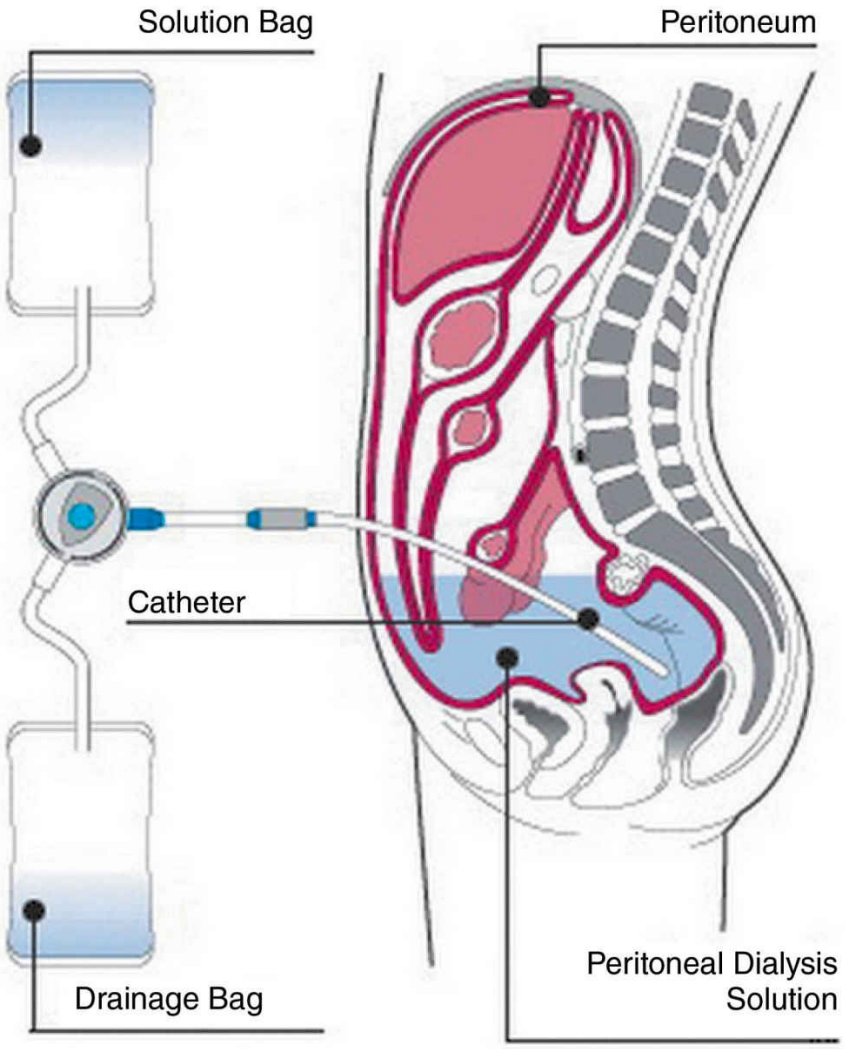


Fig. 32.7: The principle of peritoneal dialysis

G. Hemoperfusion (HP)

Hemoperfusion is a procedure in which large volumes of patient's blood are passed over an ***adsorbent*** substance in order to remove toxic substances.

Salient features:

(1) It is an extracorporeal form of treatment because the blood is pumped through a device outside the patient's body.

(2) **Sorbents most commonly used - resins** and **activated charcoal** [charcoal hemoperfusion]**.**

(3) Hemoperfusion is useful, even with highly protein bound substances, that

have a high volume of distribution (V_d) and are lipid soluble (acetaminophen, barbiturates, carbamazepine, digitalis, ethchlorvynol, glutethimide, meprobamate, methaqualone, methotrexate, paraquat and theophylline).

V. ADMINISTRATION OF ANTIDOTES

According to the International Programme on Chemical Safety definition, an ***antidote*** (Gk antididonai, "given against") is a therapeutic substance used to counteract the toxic actions of a specified xenobiotic.

A. Classification of Antidotes

1. Classification based on site of action

Antidotes may counter poisons

(1) when they are within the GIT

(2) when they are in the blood

(3) when they are acting on the target cells (**Table 5**, **Fig 32.8**).

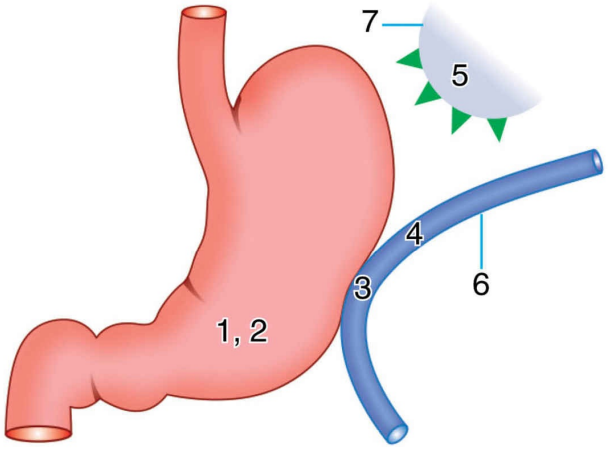


Fig. 32.8: Classification of antidotes (1 – 5) according to their site of action. (1) Physical antidotes (2) Chemical antidotes (3) Chelating agents (4) serological antidotes (5)Physiological antidotes (6) Circulatory system (7) Target cell

Table 5: Classification of antidotes based on the site of action

1. **Mechanical or physical antidotes**
2. **Chemical antidotes**
3. **Chelating agents**
4. **Serological antidotes**
5. **Physiological or pharmacological antidotes**

a. Mechanical or physical antidotes

Mechanical or ***physical antidotes*** work ***mechanically*** on poisons within the GIT. They are classified as

(1) **Adsorbents** – which adsorb poisons on to their surface [eg activated charcoal]

(2) **Demulcents** - prevent their absorption by making a protective coating on stomach wall [milk, ghee, oil, butter]

(3) **Diluents** – Dilute the poison rendering them ineffective [eg plain water, bulky food]. Some antidotes may work in more than one way, eg. milk may act as a demulcent as well as a diluent.

i. Adsorbents

Adsorbents are antidotes which adsorb poisons onto their surfaces [**ex** – Activated charcoal, Bentonite [particularly calcium bentonite, which is also a major constituent of fuller's earth (Multani Mitti)]. Fuller's earth [Fuller's earth was once used by fullers (workers who cleanse clothes -particularly wool- to eliminate oils, dirt, and other impurities. The process is known as "fulling". In clinical toxicology it is mainly used for ***paraquat poisoning*** (***ch 35***)]. Most important adsorbent is ***activated charcoal*** [syn Activated carbon, Activated coal, AC, Medicinal Carbon, Medicinal Charcoal]. It is a form of carbon that has been processed to make it extremely porous and thus to have a very large surface area available for adsorption of poisons.

Salient features:

(1) AC is typically given after gastric lavage.

(2) It is considered to be the single most effective antidote available today.
(3) **Dosage** - The usual adult dose 50-100 g mixed in about 250 ml of water. The pediatric dose is 1 g/kg.
(4) Drugs not well adsorbed (as well as some contraindications to AC) are given in **Table 6** and **Table 7**.

Table 6: (with **Memory Aid 10**) Drugs and Poisons NOT adsorbed by activated charcoal

Remember the mnemonic CLIME [also see Table 6]
Corrosives (acids and alkalis), **C**yanide
Lithium
Iron
Methanol, **M**etals [but AC is useful in inorganic Hg]
Ethanol, **E**thylene Glycol
[In all the above poisons, AC is **not recommended**, except in CN and methanol. (i) Binding of AC to **cyanide** is <4%, yet AC is a very useful antidote in cyanide poisoning because its toxic dose is very small. 50 g of AC binds more than 10 lethal doses of KCN] (ii) In **methanol**, activated charcoal is given because it prevents absorption of co-ingested toxins.

(5) **Mechanism of action:** (i) Adsorbs toxic substances, thus inhibiting GI absorption (ii) For selected drugs - es clearance by interfering with enterohepatic recycling (iii) By lowering intraluminal concentration of poison in the GIT, AC creates a concentration gradient between the blood and bowel fluid. This - es the flow of poison from the blood into GIT, a process known as **gastrointestinal dialysis** or **gut dialysis**. (iv) the addition of sorbitol results in hyperosmotic laxative action causing catharsis
(6) **Palatability** – Black and gritty nature of AC makes it unacceptable to patients, especially children. This problem is overcome by giving it with flavoring agents e.g. chocolate and cherry syrups, ice creams and sherbets.

(7) **Adverse effects:** (i) Gastrointestinal: (a) Black tongue and stools (b) Constipation (c) intestinal obstruction (d) Vomiting (e) diarrhea (ii) Ocular: (a) Corneal abrasions (iii) Respiratory: (a) Aspiration pneumonitis

(8) **Contraindications:** (i) Intestinal obstruction (ii) Perforation (iii) Patients at risk of hemorrhage or GI perforation [please also see **Table 7**]

(9) **Multiple Dose Activated Charcoal (MDAC)** – ***MDAC*** is administration of 50 g of AC every 6 hours for 3 days.

Table 7 (with Memory aid 11): Activated charcoal not helpful/contraindicated in

Another mnemonic is PHIALS [Also see Table 6]

P esticides, petroleum distillates, unprotected airway

H ydrocarbons, heavy metals

I ron, **I** leus, intestinal obstruction

A cids, **A** lkali, **A** lcohols, **A** ltered level of consciousness, **A** spiration risk

L ithium, **L** ack of gag reflex

S olvents, **S** eizures

ii. Demulcents

Demulcents (Latin demulcere, "caress") are substances that form a protective film over gastric mucosa, preventing entry of poison to the systemic circulation.

Salient features:

(1) **Ex**. aluminum hydroxide gel, egg white, fats, milk, milk of magnesia, oils, starch.

(2) **Precautions** – Fats and oils dissolve fat soluble poisons and may - their absorption. They should thus not be used in fat soluble poisons such as acetone, aniline, CCl_4, DDT, kerosene, OP, phenol, phosphorus and turpentine.

iii. Diluents

Dilute the poison and reduce its effectiveness. **Ex**. Boiled mashed potato, boiled

rice, boiled vegetables, halwa. Bulky food is particularly useful in ingestion of powdered glass.

b. Chemical antidotes

Chemical antidotes neutralize the poisons ***chemically*** within the GIT. **Ex**

(1) **Acids** - neutralize alkalis. Weak solutions should be given eg Canned fruit juice, Lemon juice, Vinegar. Neutralization of acids with strong alkalis and vice-versa must be avoided, because the antidote may itself be injurious and strong exothermic reactions cause further injury. Sodium carbonate and bicarbonate to neutralize acids are avoided because of excessive release of CO_2, which may cause perforation of the weakened stomach walls.

(2) **Albumen** - Found in egg white. Can be given in mercuric chloride poisoning [forms insoluble mercury albuminate] and Cu poisoning [forms insoluble copper albuminate]

(3) **Alkalis** – Neutralize acids. Weak solutions should be given. **Ex:** Alkaline hydroxides, e.g. (i) magnesium hydroxide (ii) calcium hydroxide [$Ca(OH)_2$; **slaked lime**] is a white powder obtained when calcium oxide [CaO; lime or quicklime] is mixed, or "slaked" with water. Also called **hydrated lime**, builders lime, slack lime, cal, or pickling lime. For medicinal use slaked lime is sweetened [**saccharated lime**] for ease of ingestion. It may be given against many acids.

(4) **Common salt** - May be given for silver nitrate poisoning. Forms insoluble silver chloride.

(5) **Copper sulfate** - Was once given for phosphorus poisoning. Forms insoluble cupric phosphide. Not used now.

(6) **Dialysed iron** – was once given for arsenic poisoning because it forms insoluble **ferric arsenite** with arsenic salts. Not used now.

(7) **Dicobalt edetate** – chemically combines with cyanides

(8) **Freshly prepared hydrated ferric oxide [Fe_2O_3]-** was once given in arsenic poisoning because it forms insoluble **ferric arsenite** with arsenic salts.

(9) **Iodine** - A sol^n of tincture iodine or Lugol's iodine 15 drops added to half a glass of warm water precipitates most alkaloids [eg quinine, strychnine etc], lead, mercury and silver

(10) **$KMnO_4$** :

(i) Potassium permanganate in 1:5000 conc [Condy's lotion] is a strong oxidizing solution.

(ii) Useful in (a) Atropine (b) Barbiturates (c) Cyanides (d) Hydrocyanic acid (e) Opium and its derivatives (f) Phosphorus and (g) Strychnine.

(iii) It oxidizes poisons [rendering them inactive], and in the process gets reduced itself [loses its pink color]

(iv) Thus the stomach wash must be continued till the solution coming out is of the same pink color as that of the solution introduced

(11) **Tannic acid** - 4% sol^{n} or tannin [strong tea] precipitates aconite, apomorphine, cinchona, cocaine, metals [aluminum, cobalt, copper, lead, mercury, nickel, silver, zinc], nicotine, pilocarpine and strychnine.

c. Chelating agents

Chelating agents (chelants) [Latin chela, claw; because they grip poison as if in a claw] are chemicals that form soluble, complex molecules (chelates) with metal ions, inactivating them so that they cannot react with other elements (may act within GIT too).

Salient features:

Characteristics of an ideal chelating agent: (i) Ability to compete with natural chelators, eg strong intracellular binding of cadmium to **metallothionein** prevents most chelating agents from removing it from this natural chelate. (ii) Complexes formed should be non-toxic (iii) Distribution - Same as that of metal (iv) High affinity for toxic metals but low for essential metals (v) High water solubility (vi) lipid solubility. (vii) minimal toxicity (viii) good absorbability from the GIT (ix) Rapid elimination of the toxic metal.

i. BAL

BAL [British anti-Lewisite, Dimercaprol], is a compound developed by the British at Oxford University during World War II.

Salient features:

(1) BAL has 2 unsaturated –SH groups, which combine with arsenic, rendering it ineffective.

(2) **Dose** – BAL comes as a yellow, viscous, oily liquid with a disagreeable sulphurous odor. Available as 3-mL ampules containing 100 mg/mL of BAL, 200 mg/mL of benzyl benzoate and 700 mg/mL of peanut (arachis) oil. Since

it is oily it must be injected deep IM (gluteal region). IV infusion causes fat embolism. Aqueous solutions are easily oxidized and therefore unstable. Peanut oil stabilizes BAL. Benzyl benzoate makes BAL miscible with oil. The dose is 3mg/kg every 4 hours for 48 hours and then twice daily for 10 days.

(3) **Indications** – Pl see below.

Memory Aid 12: Indications of BAL

Mnemonic 1 - BAL is primarily used in **MAL** - **M**ercury, **A**rsenic, **L**ead

Mnemonic 2 - A B C Gets **Th**umping **M**ajority

BAL is also in several other heavy metals eg **A**ntimony, **B**ismuth, **C**opper, **G**old and **Th** alliu**M**

(4) **Contraindications:** (i) **Cadmium** - Cadmium is primarily a nephrotoxic poison and BAL - es the uptake of cadmium in kidneys, thus - ing its nephrotoxicity. (ii) **Iron poisoning** – must be avoided because BAL-iron complex causes severe vomiting and - s metal chelation by other chelators. In other metal poisonings, iron supplements must not be given for the same reason. (iii) **Organic Hg** [eg **Methylmercury]** because causes redistribution of organic Hg to brain, - ing its toxicity (iv) Hepatic dysfunction (v) peanut allergy (because BAL is formulated in peanut oil)

(5) **Precautions:** (i) **Alkalinize the urine** – Must be done because BAL-metal chelate dissociates in acidic urine (ii) Use with caution in **G6PD deficiency** – causes hemolysis (iii) May cause **deficiency of essential metals** like zinc.

ii. BAL analogues

(a) DMSA

DMSA (2,3-**D**i**M**ercapto**S**uccinic **A**cid, Succimer, Chemet) was synthesized in 1949 in England.

Salient features:

(1) Both DMSA and DMPS are water soluble analogs of BAL.

(2) Used primarily for **MAL** (**M**ercury, **A**rsenic, **L**ead).

(3) In combination with EDTA, it is very effective.

(4) Its advantages over BAL and $CaNa_2EDTA$ are: (i) Can be **orally** administered (ii) does not . levels of essential trace metals such as Zn (iii) toxicity is less (iv) Fe can be co-administered (v) can be used in **G6PD deficiency** (vi) Superior to $CaNa_2EDTA$ in **Pb poisoning** as it does not redistribute Pb to the brain (vii) can be used in **methylmercury**.

(5) **Dose** – Succimer is available as 100-mg bead filled capsules. If capsule can not be swallowed, it is separated and beads sprinkled over juice or ice-cream and ingested. Dose is 10mg/kg tds # 5 days, followed by same dose bd # 14 days.

(b) DMPS

(1) **DMPS** (2,3-**D**i**M**ercapto-1-**P**ropane **S**ulfonic **A**cid, **Dimaval, Unithiol**) has same indications as DMSA.

(2) Dosing regimen is same as DMSA, except that 12.5mg/kg is used.

iii. Calcium Disodium Edetate [$CaNa_2EDTA$]

Calcium Disodium Edetate [syn. Edetic acid, EDTA, Ethylenediaminetetraacetic acid, Versene] is capable of chelating many metals [cadmium, cobalt, copper, iron, lead, mercury, and nickel], but is currently used almost exclusively for ***lead poisoning***.

Salient features:

(1) **Mechanism** - $CaNa_2EDTA$ combines with lead, to form a stable, soluble, non-ionized compound which is subsequently excreted in the urine. $CaNa_2EDTA$ - s urinary lead excretion 20-50 fold.

(2) **Availability** – Available in 5-mL ampules containing 200 mg of $CaNa_2EDTA$ per mL (1 g per ampule).

(3) **Administration** - (a) **Route** - IM administration causes pain. So given by IV infusion in a conc of 0.5% over 24 hours [in 5% dextrose or 0.9% NaCl]. (b) **Dose** - 1500 $mg/m^2/d$ in adults; 1000 $mg/m^2/d$ in children. (c) **Concentrations** > 0.5% may lead to thrombophlebitis and should be avoided. (d) $CaNa_2EDTA$ is incompatible with other solutions.

(4) **Contraindications** - Anuria, hepatitis, renal disease.

(5) **Precautions:** (i) Lead encephalopathy and cerebral edema – patients may experience a lethal - in intracranial pressure following IV infusion; the IM route is preferred for these patients. Co-administration of BAL is useful. In cases where the IV route is necessary, avoid rapid infusion. The dosage schedule should be followed and at no time should the recommended daily dose be exceeded.

iv. D-Penicillamine (DPA)

D-Penicillamine (Cuprimine, Depen) is a metabolite of penicillin, having no antibiotic properties.

Salient features

(1) **L-isomer - L-penicillamine** is toxic (it inhibits the action of pyridoxine; may produce optic neuritis), and is thus never used.

Memory Aid 13: Indications of D-Penicillamine

D-Peni **c**i **l** la**m** ine is useful against

1. **c**opper, 2. **l**ead and 3. **m**ercury.

2 more indications are remembered by

Pencil is **used** as **Z**ip **G**u**N** in **Ind**ia

It can be decoded as:

Pencil lamine is **used** in

4. **Z**inc

5. **G**old

6. in chronic exogenous Cu poisoning, such as **Ind**ian childhood cirrhosis.

7. some **N**on toxic conditions, eg **c**ystinuria, **r**heumatoid arthritis, **s**cleroderma, Wilson's disease (due to disorder of copper metabolism). **Mnemonic** – **Wilson's disease** is a **C** U**R S**E.

(i) in chronic exogenous copper poisoning, such as **Indian childhood cirrhosis**. (ii) some non toxic conditions [**Wilson's disease** (due to disorder of copper metabolism) **cystinuria, rheumatoid arthritis** and **scleroderma**].

(2) **Dose** – 30mg/kg body wt up to a total of 2 g/day in 4 divided does **orally** for 7 days.

(3) **N-Acetyl-D-Penicillamine (NAPA):** (i) It is another penicillin metabolite – Useful in Hg. (ii) Dose – 1-2 g/day orally.

v. Deferoxamine

Deferoxamine [syn, ***desferrioxamine*** B or DFO] is a bacterial siderophore produced by the actinobacter Streptomyces pilosus.

Salient features:

(1) The **mesylate salt** is commercially available.

(2) **Useful** – in acute iron poisoning.

(3) **Availability** – Deferoxamine mesylate (**Desferal**) is available in two vial sizes containing either 500 mg or 2 g sterile, lyophilized powder. Adding 5 or 20 mL of sterile water for injection to either the 500-mg or the 2-g vial,

respectively, results in a 100 mg/mL solution.

(4) **Dose** - (a) Oral – 5-10 g/day (b) IM - 1 g initially, followed by 0.5 g 4 and 8 hours later and then every 4-12 hours as necessary. Total up to 6 g in 24 hours. (c) IV – Given only for patients in shock. 1 g at a rate of 15 mg/kg/h, followed by two 0.5 g doses every 4 hours. Total up to 6 g in 24 hours. These recommendations for total dosages are not scientifically developed and are based on arbitrary assumptions.

(5) **Effectiveness** – 1 mole of DFO binds 1 mole of Fe^{3+}; therefore 100mg DFO binds 8.5mg Fe^{3+}.

d. Serological antidotes [immunotherapy]

(1) Antisnake venom,

(2) Antispider venom

(3) Fab therapy for digitalis [**ch 43**] and PCP [**ch 46**]

(4) Immunotherapy for As [**ch 36**].

e. Physiological or pharmacological antidotes

Physiological or pharmacological antidotes work at the target cell by producing pharmacological effects exactly opposite to those produced by poison. Also known as dynamical antidotes.

Salient features:

(1) Act on **principle of antagonism**

(2) Action is mostly partial

(3) Their use is limited and somewhat dangerous

(4) Used after poison has been absorbed in circulation

(5) **Ex** - (i) **Analeptics** against barbiturates - Analeptics (Gk analeptikos, restorative, strengthening) are arousal agents which include proconvulsants (picrotoxin, strychnine, pentylenetetrazol, camphor), sympathomimetics (amphetamines, methylphenidate), Xanthines (caffeine, ethamivan), and nonspecific stimulants (nikethamide, bemegride, prethcamide, amiphenazole). They were once recommended against barbiturates, but are no more recommended. (ii) **Amyl nitrite** against cyanide (iii) **Barbiturates** against amphetamine, picrotoxin and strychnine (iv) **Physostigmine** against atropine **Table 8** gives some common antidotes and poisons against which they are used.

Table 8: Some common antidotes and poisons against which they are used

S.No	Antidotes	Poisons against which used
1.	Antivenin (specific)	Snake venom
2.	Atropine	Anticholinesterases (e.g., organophosphate pesticides, Carbamates)
3.	Botulin antitoxin	Botulism
4.	Calcium chloride	Calcium channel antagonists, Fluoride
5.	Chelating agents	Heavy metals
6.	Dextrose (glucose)	Oral Hypoglycemics
7.	Digoxin-specific Fab antibodies	Digitalis, Other similar glycosides
8.	Ethanol (ethyl alcohol)	Methanol, Ethylene glycol
9.	Flumazenil	Benzodiazepines
10.	Folic Acid	Methotrexate, Methyl alcohol
11.	Fomepizole (4-methylpyrazole)	Methanol, Ethylene glycol
12.	Glucagon	β-adrenergic antagonists, Calcium channel antagonists
13.	Hydroxocobalamin	Cyanide
14.	Intravenous lipid emulsion (ILE)	Local anesthetic (lidocaine) toxicity and cardiovascular collapse due to selected drugs (bupropion, cyclic antidepressants, calcium channel antagonists, β-adrenergic antagonists)
15.	N-Acetylcysteine	Acetaminophen
16.	Nalmefene	Opioids
17.	Naloxone	Opioids
18.	Methylene blue	Oxidizing chemicals (e.g., nitrites, benzocaine, sulfonamides)
19.	Oximes (e.g. Pralidoxime)	Anticholinesterases (e.g., organophosphate pesticides). Contraindicated in carbamates
20.	Oxygen (hyperbaric)	Carbon monoxide
21.	Physostigmine	Anticholinergics (e.g., atropine, Datura, Hyoscyamus)
22.	Pralidoxime	Cholinergic agents
23.	Protamine	Heparin
24.	Pyridoxine (Vitamin B_6)	Isoniazid
25.	Silibinin (Legalon SIL)	Amatoxin (Amanita phalloides)
26.	Sodium bicarbonate	Sodium channel blockers , Tricyclic antidepressants, For urinary alkalinization
27.	Sodium nitrite	Cyanide
28.	Sodium thiosulfate	Cyanide
29.	Thiamine	Wernicke syndrome, Wet beriberi
30.	Vitamin K_1	Anticoagulant rodenticides

VI. ADJUNCTIVE THERAPIES AND SYMPTOMATIC TREATMENT

Done on general well-accepted lines. Treat (1) Anxiety and Agitation (2)

Allergic Reactions (3) Cardiac arrhythmias (4) Cerebral edema and -ICP (5) CNS Depression [Coma] – Give **coma cocktail.**

Memory Aid 14: Composition of coma cocktail

DNB

1. **D**extrose – 100 mL of 50% sol^{n}
2. **N**aloxone – 2mg
3. Thiamine [Vitamin **B_1**] – 100 mg

All given IV

(6) Convulsions (7) Delirium (8) ECG anomalies (9) Electrolyte disturbances (10) Heart Block (11) Hypertension (12) Hypocalcemia (13) Hypoglycemia (14) Hyperpyrexia [hyperthermia] (15) Hypoprothrombinemia (16) Hypotension (17) Hypothermia (18) Parenteral nutrition (19) Respiratory insufficiency (20) Rhabdomyolysis (21) Seizures (22) Sinus tachycardia and (23) Vomiting.

VII. ADEQUATE FOLLOW-UP

(1) Necessary to treat complications
(2) Psychiatric consultation – in suicidal cases.

33. Corrosives

I. INTRODUCTION

Corrosives [syn caustics] fix, erode and destroy living tissues with which they come in contact with.

Salient features:

(1) Corrosives include both acids and bases and convert hemoglobin into hematin.

(2) **Dangerousness** - (i) ***A l kalis are more dangerous***. They produce **l iquefaction necrosis [Memory Aid 1]**, resulting in deeper penetration [details below]. (ii) A**c**ids are less dangerous, as they produce **coagulative necrosis [Memory Aid 2]**. It results in **hard eschar**, which prevents deeper

penetration. Hydrofluoric acid is the only acid that produces liquefactive necrosis. It is thus more dangerous than other acids.

II. MINERAL ACIDS

An **acid** (L acidus; sour) is a substance that tastes sour, reacts with metals and carbonates, turns blue litmus paper red [litmus test for acids], and has a pH <7.0. A ***mineral acid*** (or ***inorganic acid***) is an acid derived from one or more inorganic compounds. It ***does not contain a carbon atom*** [unlike its organic counterpart, which does].

Salient features:

(1) All mineral acids release hydrogen ions when dissolved in water.

(2) **Mechanism of action:** (i) Acids cause protein coagulation (**coagulative necrosis**)"Form a hard eschar"Limits further penetration of acids"Penetration by acids is thus self-limiting. (ii) **Concentrated forms** – React with tissue water to generate significant heat"superimposed thermal injury.

A. Hydrochloric Acid

Hydrochloric acid [acidum salis (salt acid), muriatic acid (L. muria, brine) spirit of salts] is a colorless, fuming, pungent liquid. Iranian chemist **Geber** [721-815] first made HCl in **800 AD** by mixing with common salt and sulfuric acid.

Salient features:

(1) It is often yellow in color due to impurities [traces of iron].

(2) It is a natural constituent of stomach secretions.

(3) **Uses** - (i) Bleaching agent [<10% HCl] (ii) Descaler in boilers (iii) Drain and metal cleaner (iv) Dyeing industry (v) Flux for soldering (vi) Laboratory reagent (vii) Metal refinery.

1. Signs and symptoms

a. Acute poisoning

i. Dermal contact and ingestion

(1) **Corrosive action** – Less than that of H_2SO_4.

(2) **Skin** – is not usually corroded or damaged

(3) **Mucus membranes [MM]**– readily corroded and destroyed. They are first grey or grey-white"Later becomes brown and then black due to the production of acid hematin.

ii. Inhalation

Same as in inhalation of nitric acid.

iii. Contact with eyes

Same as in nitric acid.

b. Chronic poisoning

Occurs due to chronic exposure to fumes.

(1) **Eyes** – (i) Conjunctivitis (ii) Corneal ulcers

(2) **Nose** - Coryza [inflammation of nasal mucous membranes]

(3) **Oral cavity** - (i) Inflammation of gums (ii) Loosening of teeth

(4) **GIT** – Pharyngitis

(5) **Resp system** - Bronchitis.

2. Diagnosis

Please see below under sulphuric acid.

3. Fatal dose

15-20 mL.

4. Fatal period

12-24 hours.

5. Management

Same as that for H_2SO_4.

6. PM appearances

(1) Same as that in H_2SO_4, but corrosion is less severe.

(2) **Stomach** – (i) Mucosal folds - discolored brownish (ii) contains brownish fluid (iii) Perforation – rare

(3) **Respiratory passages and lungs** – (i) acute inflammation (ii) edema.

7. Tests

(1) **Ammonia test** – place an open bottle of ammonia near stomach contents, vomitus or suspected poison"Thick white fumes of NH_4Cl would result. Normal HCl of stomach is too dilute [0.2-0.5%] to produce similar fumes [cf diagnosis of ammonia].

(2) **Litmus test** – Same as for H_2SO_4.

(3) **Silver nitrate test** – suspected sol^n + $AgNO_3$"AgCl [thick white ppt].

8. ML importance

(1) **Suicide** -

(i) Ingested as such

(ii) Used in detergent suicides by producing H_2S [**ch 44**].

(2) **Accident** -

(i) Iatrogenic - May be confused with antiseptics by negligent medical staff. In **Nov 2011**, in a hospital in West Bengal, a negligent paramedical staff applied HCl on the private parts of **Sikha Bibi**, 32 after she had delivered a male baby. As soon as it was applied, the victim felt a burning sensation and lost consciousness. Her private parts and lower abdomen turned black. The baby died later due to unrelated causes.

(ii) Accidental exposures in chemistry class [eg dropping a bottle, constant inhalation by chemistry staff].

(3) **Homicidal** – Very rare due to its corrosive nature

(4) **Abortifacient** – rarely introduced in the vagina to produce abortion.

B. Nitric Acid

Nitric acid [syn. azotic acid; aqua fortis; Engraver's acid; red spirit of nitre] is a clear, colorless, fuming, heavy liquid having a peculiar choking odor.

Salient features:

Xanthoproteic reaction [Gk xanthos, yellow]- Conc HNO_3 combines with tyrosine or tyrosine-containing proteins [as in human skin, and mucosa] to form a yellow color which is intensified to orange-yellow by the addition of alkali. The yellow color is due to formation of xanthoproteic or picric acid.

1 Signs and symptoms

a. Dermal contact and ingestion

(1) Same as that of H_2SO_4.

(2) More eructation, greater abdominal distention owing to gas formation

(3) **Yellow discoloration** - of tissues with which it comes in contact [crowns of teeth, gums, lips, tongue, esophagus, stomach wall]. Even clothing on which acid falls turns yellow.

b. Inhalation

(1) **Resp system** -

(i) Coughing and dyspnea

(ii) Sneezing

(iii) Intense irritation of throat and lungs

(iv) Suffocation [a feeling of asphyxiation]

(2) **General** - cyanosis.

c. Contact with eyes

Eyes - (i) Lachrymation (ii) Photophobia.

2. Diagnosis

Please see above under sulphuric acid.

3. Fatal Dose

10-15 mL.

4. Fatal Period

12-24 h.

5. Management

Same as for H_2SO_4.

6. PM appearances

(1) **Orange-yellow to brown stains** on

(i) Skin of mouth and where contact with the acid has occurred

(ii) Hands and neck if any of the acid has been dropped on those parts [due to xanthoproteic reaction]

(2) **Esophagus** - Mucous membrane softened, and is yellow [Xanthoproteic reaction] or brown in color [due to acid hematin]

(3) S**tomach and duodenum**-

(i) Mucous membrane - (a) yellow-brown, or occasionally green [through the action of HNO_3 on the coloring matter of the bile] (b) Soft, friable, ulcerated and easily detached.

(ii) Walls - may rarely be perforated

(4) **Other changes** - similar that in H_2SO_4.

(5) **Inhalation of fumes** –

(i) Larynx, trachea and bronchi - congested

(ii) Lungs - congested and edematous.

7. Tests

(1) **Litmus test** – Same as for H_2SO_4.

(2) Take stomach contents [or vomit] in a test tube"Add strong ferrous sulphate sol^n and H_2SO_4 gently from the sides"Brown ring forms at the junction of fluids if HNO_3 is present

(3) Take stomach contents [or vomit] in a test tube"Drop a small piece of copper"heat"pungent dark brown fumes of **nitrogen dioxide** emanate if HNO_3 is present.

8. ML importance

(1) **Suicide** – most cases of poisoning are suicidal

(2) **Accident** – rare

(3) **Homicide** – extremely rare.

C. Sulphuric Acid

1. Signs and symptoms

a. Ingestion

(1) **Immediate** – (a) Burning pain in the mouth, (b) dysphagia, (c) epigastric

pain [which soon spreads all over thorax and abdomen] (d) odynophagia [painful swallowing](e) **pharyngeal pain** [most common presenting symptom] (f) salivation, (g) stridor

(2) **Intense thirst, eructations, nausea, vomiting** – (a) Vomit is (i) brownish black [due to acid hematin; coffee grounds vomit] (ii) mucoid (iii) strongly acidic and (iv) contains shreds of charred wall of the stomach. (b) intense thirst [attempts to drink water cause more vomiting]

(3) **Findings in face** - (a) Eyes – sunken (b) Pupils – dilated (c) Lips – Swollen, excoriated (d) Angles of mouth – Brown or black streaks found extending from angles of mouth to the sides of the chin, and sometimes to the front of neck. (e) Mucus membranes of mouth, throat and esophagus – corroded (f) Teeth – chalky white (g) Tongue – black, sodden, swollen

(4) **Voice** – hoarse and husky [**dysphonia**]. Due to coincidental damage to larynx during swallowing or during vomiting

(5) **Abdomen** – (a) distended and tender, (b) tenesmus

(6) **Constipation** – severe

(7) Features of **generalized shock**

(8) **Metabolic acidosis** – due to (a) absorption of acid (b) severe tissue burns (c) shock

(9) **Leucocytosis**

(10) **Chemical peritonitis** – if perforation occurs [of all acids, perforation is commonest in sulfuric acid. Please also see ch 31 “General Principles” under the heading “Perforation”]

(11) **Mind** - remains clear till death

(12) **If victim recovers, late symptoms, signs and sequelae are** – (a) Permanent scars on skin (b) Strictures of esophagus and stomach [hour glass deformity] (c) pyloric stenosis (d) -propensity for carcinomas.

b. Contact with eyes

Can occur accidentally or with an intention to injure [vitriolage – please see MLI below]. (1) Conjunctivitis (2) Corneal edema and ulceration (3) iridocyclitis (4) Necrotizing keratitis (5) Periorbital edema.

c. Contact with skin

Can occur accidentally or with an intention to injure [vitriolage – please see MLI

below]. (1) Intense burning pain, (2) Immediate corrosion, (3) destruction of skin.

2. Diagnosis

a. For all corrosives

(1) **Saliva** – pH tested by litmus paper

(2) Stains on clothing, scene etc – Add few drops of Na_2CO_3 or $NaHCO_3$"Bubbles indicate acid.

(3) - prothrombin time (PT) and -partial thromboplastin time [PTT]

(4) Arterial pH < 7.22, indicate acid ingestion.

(5) **Chest and abdominal radiographs** – may show signs of esophageal or gastric perforation [pneumomediastinum, pneumoperitoneum]. Pleural effusion may also be seen. CT is much more sensitive than X-rays for detecting viscus perforation.

(6) **Contrast esophagram** - For assessing the extent of esophageal injury. In late ingestions, can detect strictures.

(7) **Endoscopy** - should be performed **within 12 h** [safest during this period]. Not later than 24 h, because risk of perforation -es after this period. Early endoscopy permits early grading of injuries and helps to determine treatment plan and nutritional support.

b. Specific for H_2SO_4

Vomitus"Add 10% $BaCl_2$. Heavy white ppt [$BaSO_4$].

3. Fatal dose

10-15 mL.

4. Fatal period

12-24 hours.

5. Cause of death

a. Immediate

(1) Circulatory collapse [due to trauma from corrosive injury] (2) Spasm or

edema of glottis (3) Perforation of stomach.

b. Delayed

(1) Hypostatic pneumonia (2) Renal failure (3) Secondary infection (4) Starvation [due to esophageal strictures]

6. Complications

a. Immediate

(1) Atelectasis (2) GI hemorrhage (3) Obstructive lung injury (4) Perforation [esophageal and gastric] (5) Sepsis (6) Tracheobronchial necrosis (7) Upper airway injury.

b. Late

(1) Esophageal strictures (2) Pyloric stenosis (3) Upper airway obstruction (4) Vocal cord paralysis (5) carcinoma.

7. Management

a. Systemic ingestion

Management depends on the extent of injury.

i. Grade 1 esophageal injury

(1) Diet as tolerated

(2) No further therapy needed, supportive care.

ii. Grade 2A esophageal injury

(1) If unable to tolerate PO, provide nutritional support via nasogastric, orogastric, or percutaneous feeding tube; or total parenteral nutrition

(2) Admission recommended, supportive care.

iii. Grade 2B and 3 esophageal injuries

(1) Admission into intensive care unit

(2) Initiate early percutaneous feeding tube; or total parenteral nutrition.

(3) **Prevention of stricture formation** –

(i) Antacids

(ii) Antibiotics

(iii) Corticosteroids. Optimal management remains controversial. Some authorities do not recommend steroids and antibiotics.

(4) **If strictures have developed** -

(i) Nasogastric tubes

(ii) Total parenteral nutrition [TPN]. Gastrostomy may be required.

(iii) Traditional treatment for focal esophageal stricture - Esophageal dilatation with esophagoscopy.

(5) Requires additional imaging or surgical exploration for gastric injuries.

b. Contact with skin and eyes

(1) **Wash** - the affected part with

(i) Plenty of water and soap or

(ii) Sodium or potassium carbonate. In case of eyes, wash with very dilute sol^{n} of sodium bicarbonate [since much weaker than sodium carbonate, it does not injure eyes]; then instill a few drops of castor oil or olive oil.

(2) **Neutralize** – After washing apply a thick paste of magnesium oxide or carbonate.

8. Progress of injury

Perforation occurs immediately. If perforation does not occur corrosive injury proceeds as follows:

(1) immediate to 4th day – inflammation

(2) 4th day – 3 wks – Neovascularization and fibroblast proliferation take place, laying down new collagen and replacing the damaged tissue with granulation tissue.

(3) 8 wks onwards – Remodeling occurs. Progressive narrowing of the esophageal lumen. The dense scar formation presents clinically as a stricture, leading to dysphagia and significant nutritional deficits.

9. PM appearances

Depend on (1) **strength** of acid (2) **quantity** of acid and (3) **survival time** of patient.

a. External

(1) **Clothings** – acid burns, stains

(2) **Linear burns** – coursing down the angles of the mouth.
(3) **Burns** – (a) of lip, chin, front of chest, hands (b) if acid is taken with a spoon these areas will escape injury
(4) **Swelling** – of lips and mouth [due to inflammation]
(5) **Color of burnt areas** – initially grayish white, but soon becomes brown or black and leathery. May simulate abrasions.

Fig. 33.1: Corrosion of lips and gums in corrosive ingestion

b. Internal

(1) **Esophagus** – perforation is rare [Please see above under "acids and bases – differences in action"]
(2) **Stomach** – (a) Corrosion and perforation common (b) If corrosion is absent, there will be (i) inflammation, (ii) swelling due to edema and (iii) severe interstitial hemorrhages (c) Consistency – Stomach turns into a soft, spongy, black mass, which readily disintegrates when touched (d) Lesser curvature –

May be affected more because acid may travel along magenstrasse [please see ch 31 for details] (e) Pyloric region – Because of pyloric spasm, acid cannot pass through and collects here. Antral pooling occurs. Causes more injury to pyloric region (f) Mucosal ridges – being raised, may show more injury than intervening furrows, which lie deeper (g) Color of mucosa – Black. Charred appearance.

Fig. 33.2: Sulphuric acid ingestion. Pl note soft, spongy, blackish stomach

10. Preservation of samples

(1) **Organs** - Vitreous humor and lungs have to be preserved [please see **ch 5** for more details].

(2) **Clothings** – may reveal acid, if accidentally spilled over them [**ch 5**].

11. Tests

(1) **Litmus test** - Suspected sol^{n} would turn blue litmus red.

(2) Suspect material + BaCl or $BaNO_3$"white ppt of $BaSO_4$.

(3) Pour suspect material over organic matter (eg cotton)"Charring.

12. ML importance

(1) Vitriolage - ***Vitriolage*** or ***vitriol throwing*** is throwing of a corrosive substance over the face of an adversary due to jealousy or revenge.

Salient features:

(1) Corrosive used - Usually conc. H_2SO_4 is used, but sometimes an alkali, a corrosive salt [e.g. corrosive sublimate or $HgCl_2$] or irritant juice of a plant [e.g. marking nut, calotropis] may be used. (ii) Aim - is not to kill but disfigure. (iii) Used by – mainly by jilted lovers on their girl friends; but anyone can use. Some common motivations are enmity, hatred, jealousy, revenge, rioting and rivalry. (iv) *Method* - (a) corrosive is filled inside an empty light bulb [or some other *easily breakable* container] and thrown over the victim (b) Filled inside a *pichkari* [spray gun used in *Holi*] and sprayed over victim. (v) Management – please see above [contact with skin and eyes] (vi) Outcome - (a) Destruction of garments (b) Disfiguration of face, if not treated promptly. (c) Sometimes blindness. (d) Contractures – around joints may cause restriction of movements (e) Death may also occur. (vii) Medicolegal investigation - (a) Clothes - must be collected and sent for chemical examination (b) plain water swabs - from affected areas – sent for chemical examination (c) Scene of crime - Spilt traces of corrosives may be found – on ground, walls, nearby vehicles etc. If acid was contained in glass bulb, pieces of broken glass may be found. Chemical examination of fluid on glass pieces would reveal same corrosive as that found on the body of victim (d) Identification of perpetrator – is rarely possible, if during the act of throwing acid, few drops sprinkle on his own body. (viii) *MLI* [of vitriolage] – (a) It is a grievous hurt, punishable **u/s 326** IPC [**ch 11**]. (b) All hospitals, whether **Govt or private** must provide immediate **first-aid** or **medical treatment free of cost** to any victim of **vitriolage** [**S.357C, CrPC**]. If such treatment is not provided, punishment is **1 y** or **fine** or **both** [**S.166B, IPC]** (**ch 11)** (c) If a police officer fails to record FIR **u/s154(1) CrPC** of a victim of **vitriolage**, he shall be fined and given rigorous imprisonment for a minimum of **6 months** and **up to 2 years [S.166A, IPC (ch 25)]**.

(2) **Accidental poisoning** – results due to H2SO4 being mistaken for glycerine,

castor oil or linseed oil

(3) **Suicide** – May be taken for suicide. Generally taken orally, but rectal administration has been described.

(4) **Homicide** – Can not be used for homicide, because of its corrosive nature. Food gets charred when mixed with it

(5) **Disposal of dead bodies** – May be used for criminal disposal of dead bodies after murder.

(6) **For blinding an enemy or to extort confessions** – Acids have been used for blinding an enemy. The police use acids in order to extort a confession.

(7) **Occupational hazard** - Inhalation of vapor may occur inadvertently in chemical factories. May require compensation from factory owner

(8) **Abortifacient** – injected in vagina for criminal abortion

(9) **Battery acid** – is 30-35%)H_2SO_4. Used in the **illicit manufacture of several narcotic drug derivatives** [e.g. cocaine sulfate] - (please also see chapter on deliriant poisons – ML imp of Cocaine).

(10) **Self defense** - Acids have been used by women for defending themselves against sexual assault.

III. ORGANIC ACIDS

A. Carbolic Acid (Phenol, C_6H_5OH)

1. Properties

(1) **Appearance** -

(i) Pure acid consists of short, colorless, prismatic, needle like crystals

(ii) Commercial phenol is a brownish liquid containing impurities like cresol.

(2) **On exposure to air** – crystals turn pink and liquefy

(3) **Smell** – Characteristic “carbolic” or phenolic [one that emanates from hospitals, OTs]

(4) **Taste** - burning sweetish.

(5) **Solubility** –

(i) Sparingly soluble in water [6.6% at 17°C]

(ii) Freely soluble in alcohol, benzene, ether and glycerine.

(iii) Miscible with camphor

(6) **Not a true acid** – Does not turn litmus paper red
(7) **Use** – antiseptic, disinfectant.

2. Toxicokinetics

a. Absorption

Readily absorbed from all routes including GIT, Rectum, Respiratory tract, Serous cavities, skin, Vagina and wounds.

b. Excretion

(1) Phenol is converted to **pyrocatechol** and **hydroquinone** in the liver before being excreted in the urine partly free and partly in unstable combination with sulphuric and glucuronic acid. Their further oxidation in the urine gives rise to green colored urine. Initially the urine is colorless or slightly green, but later it becomes dark green, which darkens further to almost black on standing. This symptom is known as **carboluria**.
(2) Traces – excreted in lungs, salivary glands, stomach and skin.
(3) Time required for complete excretion – 36 hours.

3. Acute poisoning

a. Signs and symptoms

Poisoning by CA is known as **carbolism**.

i. Local

(1) **Skin -**
(i) Numbness [due to damage to nv endings]
(ii) Burns – (a) CA precipitates proteins and coagulates cell contents (b) When comes in contact with skin"produces a painless white opaque eschar"falls off in a few days"Leaves a brown stain. (c) Longer contact produces deep burns"black (d) If necrosis and gangrene of tissue sets in"corroded area becomes greenish-white or brownish-white. Dead tissue sloughs readily (e) Lysol burns produce a brownish purple color
(2) **Digestive tract** –
(i) Nausea, vomiting – in about 20% cases.

(ii) Hot burning pain – Extends from mouth to stomach"followed by tingling"anesthesia
(iii) Lips, mouth, tongue – corroded. Soon harden and become white
(iv) Deglutition and speech – become difficult.

ii. Systemic

(1) **General** -
(i) Odor - Strong odor of phenol in breath
(ii) Temp.
(iii) Pupils – variable. Usually contracted. But may sometimes be dilated.
(iv) Breathing – stertorous
(v) Pulse – Feeble, irregular, Rapid
(vi) Face – covered with cold sweat, Dusky cyanosis
(2) **CNS** -
(i) Phenol initially causes stimulation of CNS, but later acts as a depressant to CNS, especially respiratory center
(ii) Stimulatory symptoms - (a) agitation (b) Convulsions, seizures, lockjaw
(iii) Depressant symptoms - (a) Headache, giddiness, confusion (b) unconsciousness, coma towards the end
(3) **CVS** - Hypotension, arrhythmias.
(4) **Resp system** -
(i) Breathing – slow, labored, short and infrequent gasps"progresses to resp failure and arrest.
(ii) Laryngeal and Pulmonary edema [due to irritation]
(iii) Bronchitis, bronchopneumonia [due to aspiration of vomit]
(5) **Liver** – damaged. Signs of hepatotoxicity.
(6) **Blood** -
(i) Hemolysis
(ii) Methemoglobinemia [in severe cases]
(7) **Acid-base balance** - (i) Metabolic acidosis (ii) Respiratory alkalosis
(8) **Urine** -
(i) Scanty, may be completely suppressed
(ii) Contains albumin and free Hb
(iii) Color – green [please see above under “excretion” for details].

(9) **Rare symptoms** – Fine, rapid, rhythmic contractions of the perioral musculature [rate about 5 Hz] resembling the chewing movements of a rabbit [rabbit syndrome]. Usually an extrapyramidal side effect of neuroleptic drugs. Phenol causes tremors directly by inducing -ed Ach release both in the peripheral nervous system at motor nerve endings and within the CNS.

b. Fatal dose

10-15 g.

c. Fatal period

3-4 h.

d. Management

i. Contact

(1) If phenol falls on clothing – remove clothing immediately. Clean skin as below

(2) Mechanism of injury and treatment of phenol differs from that of standard corrosive exposures. In contrast to other corrosives, phenols cause injury by their ability to accept an electron pair to form a covalent bond. This difference alters management of dermal burns in that irrigation should be done with **polyethylene glycol** solution because water can actually worsen the injury. If water is all that is available for irrigation, it should be mixed with soap.

(3) **Washing** - with (i) Ethyl alcohol [10%] (ii) Methylated spirit and (iii) Olive oil is also helpful.

ii. Ingestion

(1) **Emetics** – would generally fail due to anesthetic effect

(2) **Gastric lavage:**

(i) Wash carefully with lukewarm water mixed with any of the following - (a) castor oil (b) olive oil (c) glycerine [10%] (d) magnesium or sodium sulphate (e) saccharated lime or (f) soap soln. Phenol combines with them to form harmless products

(ii) Continue washing - till washings are clear, colorless and odorless

(iii) After completion of lavage – leave (a) medicinal liquid paraffin [about 250

cc] or (b) $MgSO_4$ [30 g] in the stomach

(3) Demulcents

(4) **Normal saline with $NaHCO_3$ [7g/L]** IV – helps in 3 ways

(i) Combats circulatory collapse

(ii) Dilutes carbolic acid [CA] content of blood

(iii) Promotes diuresis "-ed excretion of CA

(5) **Hemodialysis** – if there is renal failure

(6) **To correct methemoglobinemia** -

(i) Methylene blue IV

(ii) Exchange transfusion – if methemoglobinemia is >70%.

e. Cause of death

(1) **Asphyxia** -

(i) Failure of respiration

(ii) Edema of glottis

(iii) Complications [eg bronchopneumonia]

(2) **Syncope**.

f. PM appearances

i. External

(1) Smell – of phenol from body, especially from mouth

(2) **Corrosion of skin** – around mouth; Especially as trickling marks from angles of mouth. Color brownish or grayish

(3) **Tongue** – White, swollen, hardened.

(4) **Lips, mouth, throat** – mucus membrane is (i) coagulated (ii) corrugated (iii) detached [partially] (iv) opaque (v) sodden and (vi) swollen (vii) color - whitened, brown or ash-gray. (viii) Shows numerous small submucous hemorrhages.

ii. Internal

(1) **Esophagus** –

(i) Mucosa same as that of mouth and throat.

(ii) In addition arranged in longitudinal folds

(2) **Stomach** –

(i) Contents – reddish fluid mixed with mucus and shreds of epithelium
(ii) Smell – of phenol
(iii) Mucosa - same as that of mouth and throat.
(iv) In addition, it is thick and leathery – because of longer duration of contact
(v) Necrotic mucosa – may show partial separation, with severe congestion of underlying tissue
(vi) Furrows - show more damage. Intervening areas show less damage, are dark red and are not opaque
(3) **Duodenum, jejunum, upper part of ileum** – show similar changes, but to a lesser degree
(4) **Resp tract** – Changes are seen especially if inhalation of acid or vomitus has occurred
(i) Coagulation necrosis of mucosa
(ii) Severe congestion of submucous layers
(iii) Laryngeal and pulmonary edema
(5) **Liver, spleen** – show a whitish, hardened patch where the stomach is in contact with them, because of transudation of phenol
(6) **Kidney** – hemorrhagic nephritis, if death is delayed
(7) **Brain** – congested, edematous
(8) **Blood** – dark, semifluid, partially coagulated.

g. Tests

1 ml of solution to be tested (urine)+few drops of 10% ferric chloride solution"Bluish color will develop if phenol is present. Salicylates also give +ve results.

4. Chronic poisoning (phenol marasmus)

Occurs due to chronic exposure to phenol.

Signs and symptoms

(1) **General** - (i) Anorexia (ii) Headache (iii) Vertigo (iv) Wt loss
(2) **Urine** – dark
(3) **Pigmentation** –
(i) Yellowish (ocher-like) discoloration of cartilage, sclera and skin [**ochronosis**] seen on microscopic examination.

(ii) However, macroscopically the affected tissues appear bluish grey because of a light scattering phenomenon.

(iii) D/d – (a) Alkaptonuria – an inborn error of metabolism because of deficiency of homogentisic acid oxidase. Homogentisic acid gets deposited in cartilages, fibrous tissues and ligaments.

Memory Aid 3: Ochronosis

O chron osis occurs in **chron** ic phenol poisoning

5. ML importance

(1) Used for **suicide**

(2) **Accidental poisoning** -

(i) due to carelessness in storage

(ii) misguided lay medical treatment, like application on raw wounds etc

(3) **Criminal abortion** – introduced into vagina and uterus. May cause accidental poisoning

(4) **Homicidal poisoning** – rare because of odor and taste.

B. Oxalic Acid

1. Description

(1) **Two forms** - Oxalic acid (acid of sugar; salt of sorrel; IUPAC name- Ethanedioic acid) occurs in two forms – anhydrous (without associated water molecules) and dihydrate (with two water molecules). Typically it occurs as the dihydrate.

(2) Colorless, transparent prismatic crystals

(3) Resembles in appearance to the crystals of magnesium sulfate (Epsom salt) and zinc sulfate, but can be easily differentiated [**Table 1**]. Failure to differentiate may result in accidental poisoning, especially as both are used as medicines. Epsom salt is used as a laxative – a common household remedy, and zinc sulphate is used in acne, ivy poisoning, lupus erythematosus, and impetigo. Two patients at a mental hospital in Scotland died in 1956 after receiving doses of oxalic acid administered in mistake for Epsom salts.

(4) Efflorescent. Solubility – 1 in 12 in H_2O.

(5) Produced as a result of ethylene glycol metabolism, and is a major reason of its toxicity.

Table 1: Differences between oxalic acid, $MgSO_4$ and $ZnSO_4$

S.No	Feature	Oxalic acid	$MgSO_4$/$ZnSO_4$
1.	Taste	Sour	Bitter
2.	Reaction	Strongly acidic. PH<7.0	Neutral. PH=7.0
3.	On heating with sodium	Sublimates	Not so
4.	On heating with bicarbonate	Effervesces	Not so
5.	Application on ink stains or iron moulds	Stains disappear	Do not disappear

2. Occurrence in nature

Oxalic acid (along with oxalates) are present in many plants and occur naturally in animals.

(1) **Plants containing oxalic acid** (i) Cabbage (ii) Carambola fruit [unripe] (iii) Jack-in-the-Pulpit [Arisaema triphyllum] (iv) monstera fruit [unripe] (v) Rhubarb. (vi) spinach (vii) wood-sorrel. Daily excretion in urine is 20 mg.

(2) **Kidney stones** – most common component - Calcium oxalate.

(3) **Bacteria** - produce oxalates from oxidation of carbohydrates.

3. Uses

(1) **Commercial** - (i) Book binding [for cleaning leather used as jackets] (ii) Calico printing (iii) Cleaning copper and brass articles, wooden and leather surfaces (iv) Photography and (v) Straw hats [manufacture]

(2) **Domestic** - (i) bleach (ii) ink remover (iii) paint, rust, stain and varnish remover

(3) **Illegal** - (i) removing writing and signature from papers [eg wills, contracts,

bonds] (ii) removing election stain marks on fingers [to enable voting again].

4. Mechanism of action

(1) **Locally** – acts as a corrosive

(2) **Systemically** – Reacts with Calcium in plasma "Forms calcium oxalates"(i) Hypocalcemia (ii) Precipitation of calcium oxalate crystals in liver, kidneys, heart and lungs (iii) Excretion of envelop shaped crystals [calcium oxalate] in urine.

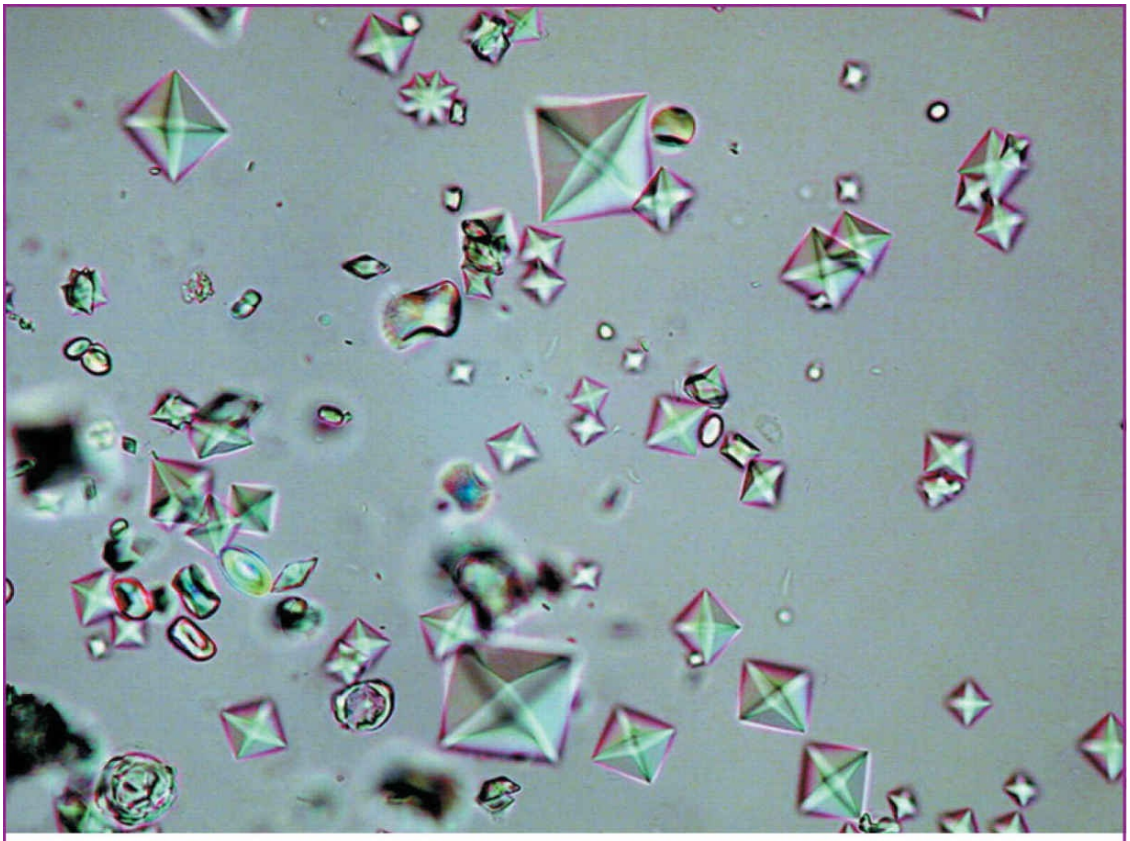


Fig. 33.3: Typical envelop shaped calcium oxalate crystals in urine of a patient with oxalic acid poisoning

5. Signs and symptoms

a. Contact

(1) Skin - is rarely damaged. May just be discolored

(2) **Mucosa** – of eye, mouth etc may be greatly damaged and may give rise to a

"scalded" appearance. Sometimes production of acid hematin may give rise to black color.

b. Ingestion

i. Immediate

(1) Burning, sour or bitter taste in the mouth which goes up to the stomach
(2) Sense of constriction around the throat
(3) Intense thirst
(4) Mouth – may appear "scalded" or sometimes black
(5) Severe pain – Begins in the epigastrium, but soon radiates all over the abdomen
(6) Abdomen is tender
(7) Persistent vomiting, eructations and diarrhea. Vomitus contains altered blood ["coffee-ground" appearance] and mucus.
(8) Signs and symptoms due to hypocalcemia -
(i) Tetany
(ii) Numbness and tingling of fingertips and legs
(iii) Chvostek sign +ve.

ii. Delayed

If patient survives initial poisoning episode, delayed symptoms may be due to **renal failure** [calcium oxalate crystals in kidneys] **Urine** – Scanty or suppressed. Contains traces of blood, albumin and calcium oxalate crystals.

6. Management

(1) **Gastric lavage** – If patient seen early, perform gastric lavage carefully with calcium salts [chloride, gluconate, lactate, chalk powder (1.5g of chalk neutralizes 1g of acid), lime water, milk]. Converts acid into insoluble calcium oxalate.
(2) **Antidote** – Calcium preparations orally [same as those used for gastric lavage].
(3) **Calcium gluconate IV** – 10 ml of 10% at frequent intervals
(4) **Parathyroid extract** – 100 units IM in severe cases. Mobilizes Ca^{+} from bones.

(5) **Dialysis or exchange transfusion** – for renal failure

(6) **Miscellaneous** - (i) Demulcents (ii) Evacuation of bowels – by castor oil or enema (iii) Symptomatic.

7. Fatal dose

600 mg/kg. For a 60 kg human 36 g.

8. Fatal period

1-2 hours.

9. PM appearances

(1) **Mucus membrane of tongue, mouth, pharynx, esophagus** -

(i) In case of concentrated solution - (a) **Whitened** as if "bleached". Similar to a "scalded" appearance (b) sometimes **brown** or **black** due to formation of acid hematin

(ii) In case of weaker solution - **Reddened** because of irritation

(2) **Esophagus** – Mucosa corrugated. Shows longitudinal erosions

(3) **Stomach** – (a) Mucosa - (i) soft and reddened (ii) Shows punctate erosions (iii) May be black [acid hematin] (iv) numerous dark brown or black streaks are seen running longitudinally along the length of the stomach, often with intercommunicating branches (v) Often entire mucosa is corroded (b) Contents – Gelatinous and brownish [acid hematin] (c) Perforation is rare

(4) **Intestines** – Upper part of duodenum shows corrosion. Rest of the intestines usually escape

(5) **Liver** - hepatic centrilobular necrosis

(6) **Kidneys** - (a) Congested and swollen by edema (b) Histologically - (i) Glomeruli - Swelling and retraction (ii) Renal tubules – full of oxalate crystals. Necrosed, especially PCT

(7) **Urinary bladder** – shows urine with calcium oxalate crystals. Under the microscope look like envelops.

(8) **All internal organs** – show congestion

(9) **Crystals of calcium oxalate** – in renal cortex and vessels and capillaries of liver, lung, and heart.

10. Test

Suspected sol^{n} + $BaNO_3$"White ppt of barium oxalate, which is soluble in HCl or HNO_3.

11. ML importance

(1) **Accidental poisoning** – Due to mistaken identity with Epsom salt or $ZnSO_4$. May lead to charges of negligence against doctors and nursing staff.
(2) **Homicide** – rare due to acrid sour taste
(3) **Suicide** – rare because of severe pain and burning
(4) **Abortifacient** – by vaginal injection.

IV. CAUSTIC ALKALIS - GENERAL

A. Ammonia

1. General

(1) At room temperature, ammonia (NH_3) is a highly water-soluble, colorless, irritant gas with a unique pungent choking odor.
(2) Ammonia has a boiling point of -33°C and an ignition temperature of 650°C.
(3) **Ammonium hydroxide** – also known as ammonia liquor, ammonia solution, ammonia water, ammoniacal liquor, aqua ammonia or aqueous ammonia is a solution of ammonia in water. It contains about 30% ammonia.

2. Signs and symptoms

Inhalation - (a) Head, ears, eyes, nose, throat (HEENT) - Facial and oral burns and ulcerations (b) Respiratory system – cough, decreased air entry, oxygen desaturation, rhonchi, salivation, stridor, Tachypnea, wheezing (c) CNS - Loss of consciousness [if exposure is massive].

3. Diagnosis

HCl test - place an open bottle of Conc HCl near stomach contents, vomitus or suspected poison"Thick white fumes of NH_4Cl would result [cf diagnosis of HCl].

4. Management

(1) Largely supportive. Directed at treating hypoxia, bronchospasm, acute lung injury (ALI), hypovolemia, and burns of the skin and eyes.
(2) Antibiotics and corticosteroids are controversial.

5. Fatal dose
15-20 mL.

B. Button Battery Ingestion (Disc Battery, Miniature Battery)
(1) **Signs and symptoms** - Irritability, pain, dysphagia, vomiting, refusal to eat. May cause Hg poisoning [**ch 36**]
(2) **Diagnosis** - X-ray
(3) **Management** - Endoscopic or surgical removal is necessary because of the risk of (i) burns (ii) perforation and (iii) metal poisoning.

C. Cement Burns

1. Mechanism of action
(1) Cement contains lime [calcium oxide], which penetrates clothing and reacts with sweat causing an exothermic reaction.
(2) Even when not exposed to moisture, the dry powder is very hygroscopic"causes a desiccation injury.
(3) Hydrated calcium oxide becomes calcium hydroxide that causes skin damage primarily due to hydroxyl ion.
(4) If cement is not removed from the skin, it continues to corrode and painlessly causes necrosis under clothing.

2. Signs and symptoms
Cement burns have an insidious onset. Most patients comment that they notice only mild irritation initially. Rarely burns may be serious

3. Management
(1) remove soaked clothing
(2) abundant washing of the wounds
(3) Neutralization with **buffered phosphate solution**. However the practice is debatable, as the heat produced by the exothermic chemical reaction of

neutralization could worsen the burns.

D. Liquid Lye (NaOH, Caustic soda)

[A] Sodium hydroxide is found in many industrial solvents and cleaners, [flooring stripping products, brick cleaners, cements], and many household products (Aquarium products, Clinitest tablets, Drain cleaners, Hair straighteners, Metal polishes and Oven cleaners) [B] **Signs and symptoms** – Burns, Irritation, Necrosis of the skin and underlying tissues [C] **Management** – As for general alkalis [D] **MLI** – May be seen in vehicular accidents as air bag ruptures [**ch 18**].

V. OTHER CORROSIVES

A. Potassium Permanganate [$KMnO_4$]

1. General

$KMnO_4$ [syn, Condy's crystals, permanganate of potash] occurs as dark purple slender crystals, having a sweet astringent taste. it is a strong oxidizing agent and is used as a disinfectant due to its strong oxidizing action on microbes. It dissolves in water to give intensely purple solutions, the evaporation of which leaves prismatic purplish-black glistening crystals. Its name **Condy's crystals** comes from UK chemist **Henry Bollmann Condy** [1826-1907], who first made **Condy's Crystals** and **Condy's Fluid** from **$KMnO_4$** in **1857**.

2. Mechanism of action

(1) **Before absorption, in solid state or strong solution** - acts as a corrosive or strong irritant. Causes coagulation necrosis.

(2) **After absorption** - Cardiac stoppage.

3. Signs and symptoms

a. Ingestion

(1) Intense thirst, nausea, vomiting and diarrhea. Vomitus is purple brown in color. Stools are black due to manganese sulfide. Excessive loss of fluids may lead to shock and collapse.

(2) **Burning pain** - from mouth to stomach
(3) Dysarthria, dysphagia
(4) **RS**- Produces severe inflammatory edema resulting in dyspnea, stridor, persistent spasmodic cough
(5) **Purple brown discoloration** - of skin and mucus membranes, with which it comes in contact. Lips, gums, teeth, tongue, tonsils and pharynx. In a few minutes the color changes to brown, dark brown and finally coal black due to conversion to manganese dioxide.
(6) **Systemic** - Methemoglobinemia, because of oxidizing nature of $KMnO_4$.

b. Local application

Locally applied as an abortifacient.
(1) Vaginal and cervical burns, erosions and ulcerations, resulting in severe scarring.
(2) Extensive bleeding and shock.

4. Diagnosis

(1) Place either a drop of H_2O_2 or a sol^n of oxalic acid and traces of H_2SO_4 over stains. They should disappear
(2) -Serum and Urine Manganese levels.

5. Fatal dose

5-10 g.

6. Fatal period

Few hours.

7. Management

(1) **Immediate dilution** - with water or milk
(2) Activated charcoal
(3) Demulcents
(4) **Gastric lavage** -
(i) May have to be done carefully, since $KMnO_4$ is a corrosive.
(ii) Fluids used - (a) dilute H_2O_2 (b) 20% sodium thiosulphate.

(iii) End point - when returning fluid is colorless

(5) **Methemoglobinemia** - Treated with methylene blue 1-2 mg/kg IV over 5 min every 4 h.

(6) **Chelation** - Done if manganese toxicity is suspected. Administer EDTA and sodium para-aminosalicylic acid.

(7) Supportive and symptomatic.

8. PM appearances

(1) **Mucus membranes of GIT** extending from lips up to intestines - Corrosion, necrosis, hemorrhage.

(2) **Liver and kidneys** - show degenerative changes.

9. MLI

(1) **Manner of poisoning** - (i) Suicidal - mostly. (ii)Accidental - (a) may occur in children, who may eat crystals mistaking them for candies. (b) When ingested or injected as an abortifacient (iii) Homicidal - very rare due to its color and peculiar taste

(2) **Production of fictitious injuries** - By applying a tablet to the skin for 10-20 min, lesions similar to those of tertiary syphilis may be produced.

VI. CHEMICAL BURNS

(1) In chemical burns, amount of tissue damaged depends upon the agent, its duration of contact, extent of penetration, quantity, strength and concentration.

(2) Chemicals continue to act on tissue until - (i) Inactivated by tissue reaction (ii) Neutralized by another agent.

(3) Hydrocarbon [eg gasoline] – Prolonged contact can cause burns due to their irritant effect and high lipid solubility.

(4) Phosphorus – also causes burns.

34. Non-Metallic Irritants and Mechanical Irritants

I. NON-METALLIC IRRITANTS

A. Phosphorus

1. Allotropes

Four allotropes of phosphorus are known:

(1) **White** [with impurities, it becomes **yellow**],

(2) **Red** [formed by heating white phosphorus to 250°C in vacuum],

(3) **Violet** [formed by dissolving white phosphorus in molten lead at 500°C, and then cooling it slowly] and

(4) **Black** [formed by heating white phosphorus at 12,000 atm]. White phosphorus is a **tetramer (P_4)**. **Gaseous phosphorus** is also known, which exists as **diphosphorus (P_2)**.

Memory Aid 1: Allotropes of Phosphorus

Vio**let** is formed by dissolving in **lead**.

Differences between the two most common allotropes - white and red are shown in **Table 1**.

Table 1: Differences between two most common allotropes of phosphorus

S.No.	Trait	White phosphorus	Red Phosphorus
1.	Color	White. Yellow (when impure)	Reddish brown
2.	Appearance	Translucent, waxy paste	Amorphous, solid mass
3.	Smell and taste	Garlic like	Odorless and tasteless
4.	Luminosity	Luminesces in the dark (poisoning results in **luminescent vomit** and **stools**)	Non-luminous
5.	Exposure to air	**pyrophoric** (self-igniting). Ignites spontaneously at 34°C. Must be kept under water.	Does not ignite spontaneously.
6.	Use	Rat baits, fireworks, fertilizers	On the side of modern matchboxes (along with powdered glass)
7.	Toxicity	Highly toxic	Non-toxic

2. Mechanism of action

(1) **Uncoupling of oxidative phosphorylation** in the hepatocyte.

(2) **Decreases ATP levels** in the hepatocyte.

(3) **Decreases transformation of triglycerides into b-lipoproteins**. Rapid **rise in hepatic triglycerides**. Massive ***hepatic steatosis*** is a hallmark of white

phosphorus toxicity.

(4) **Hepatic necrosis in zone 1** is prominent (located around the portal tracts where oxygenation is good). Significantly, other hepatotoxins, such as acetaminophen and CCl_4 produce zone 3 necrosis (located around central veins, where oxygenation is poor).

3. Symptoms and signs

a. Local application

P is an oxidizing agent, highly toxic to skin. Causes both **thermal** and **chemical** burns. 2nd and 3rd degree (Dupuytren) burns are causes most often. Since P continues to oxidize and injure tissue, it is extremely important to neutralize P immediately.

b. Ingestion

i. Acute fulminant poisoning

Occurs when the victim takes more than **1 g** (more than 15 times the average fatal dose). Vomiting, diarrhea, **peripheral vascular collapse**. Death occurs within **12 hours**.

ii. Subacute poisoning

Occurs when the victim takes fatal or near fatal dose. **Three stages** are seen.

(a) First stage [2 days]

(1) **Severe burning pain** in the mouth. Abdominal pain, **vomiting**, **diarrhea** [symptoms of GIT irritation]

(2) **Garlicky odor** (**Ch 31**).

(3) **Luminous vomit** and **stools** because of presence of phosphorus**.** Feces expelled on the bed can burn a hole through the bed sheet.

(4) Fumes emanate from the stools, as phosphorus in stools combines with atmospheric oxygen to form phosphorus pentoxide (**Smoking** or **smoky stool syndrome**).

(b) Second stage [4 days]

Patient is **symptom free**. Wants to go home. Should not be discharged.

Something like "**lucid interval**" seen in head injury [cf **ch 36**"iron toxicity].

(c) Third stage [2 days]

(1) **Hepatic toxicity** develops. Major signs are

(i) **Mousy odor** of the breath [***foetor hepaticus***]

(ii) **Flapping tremor** of hands [**asterixis**]

(iii) Hepatomegaly, jaundice, **bleeding tendencies** [prothrombin deficiency], **pruritis**.

(iv) Finally **hepatic encephalopathy** and death.

(2) **Mortality -** ranges from **20%-50%**. Prolongation of QT_C interval, ST-T wave abnormalities, more than ten times increase in **alanine aminotransferase** (ALT) and severe coagulopathy indicate **poor prognosis**.

(3) If death does not occur in eight days, patient goes on to recover.

iii. Chronic poisoning

(1) **Signs and symptoms** - Workers working with white phosphorus develop an unusual **mandibular osteonecrosis** (**phossy jaw, Lucifer's jaw** or **glass jaw**). First described by **Bristowe** in **1862**. Cachexia, anemia and fatigue also occurs.

(2) **Preventive measures** - Since mandibular necrosis starts with **toothache**, preventive measures include **regular dental checkup** of workers employed in phosphorus factories.

4. Fatal dose

1 mg/kg [**60 mg** for a 60 kg person].

5. Fatal period

½ day to 8 days. No fatalities have been reported after 8 days.

6. Management

a. Protection of healthcare personnel

Doctors, nurses and other healthcare personnel should **wear gloves** and other **protective equipment** to avoid direct contact with the patient. Phosphorus can get absorbed through skin. Also it may be present in patient's clothings and may ignite spontaneously.

b. Supportive care

(1) Continuous cardiac monitoring.

(2) Fluid resuscitation.

(3) **Vitamin K** by IV drip [to treat hypoprothrombinemia]

(4) **Prothrombin time** (PT), **Partial thromboplastin time** (PTT), hepatic enzymes, serum electrolytes, serum phosphate and serum calcium should be measured.

(5) **Whole blood/fresh frozen plasma** [to correct coagulation defects]

(6) Evaluation of **renal function** and **renal output**.

(7) **Ophthalmic irrigation** if phosphorus has entered eye.

c. Skin decontamination

Continuous **irrigation with water** or **saline**.

(1) Application of **silver nitrate** on skin (prevents ignition by depositing a thin film of silver over phosphorus particles).

(2) Application of **$CuSO_4$**.

(i) Helps in identifying occult phosphorus particles by causing a characteristic black color change

(ii) Neutralizes P at the same time [formation of cupric phosphide]

(3) Identification of phosphorus particles by **Wood's lamp** [phosphorus particles are seen to fluoresce blue], and their removal. If removal not possible, thorough debridement of skin.

d. GIT decontamination

(1) **Gastric lavage** – with **1:5000 $KMnO_4$**. Converts white phosphorus to less harmful oxide. Rarely as phosphorus comes out of the gastric lavage tube, **it can catch fire**, leading to **explosions**. It is therefore recommended that external end of gastric lavage tube be connected to a **syringe filled with water**.

(2) **Activated charcoal**

(3) **Whole bowel irrigation** with **polyethylene glycol** (decreases the absorption of phosphorus).

e. Other measures

(1) **N-Acetylcysteine** – Though primarily an antidote for paracetamol, has been tried in phosphorus poisoning with some benefit.
(2) **Ubiquinone, cysteine** and **sulfate** decrease liver damage.
(3) **Corticosteroids** are of no use in preventing hepatotoxicity.
(4) **Exchange transfusion**.
(5) Liver transplantation.

7. PM appearances

Macroscopic

(1) **Bleeding points** in the skin [bleeding diathesis produced by phosphorus]
(2) Stomach contents - **garlicky** in odor.
(3) **Luminous material** in **stomach** and **intestine** can be demonstrated (using **Wood's lamp**, as phosphorus fluoresces easily).
(4) **Erosions** and **hemorrhages** in esophagus and stomach. Rarely perforation of the stomach.
(5) **Heart** - subendocardial petechial hemorrhages
(6) **Liver** - fatty, enlarged, yellow.
(7) **Petechial hemorrhages** - in all internal organs.
(8) Individuals who have survived for a week or so would show **acute yellow atrophy.**

8. Viscera analysis

(1) **Stomach and intestines** - should be tied at both ends before submitting for toxicologic study. These viscera must be opened under nitrogen just before analysis.
(2) Other viscera - preserve in **saturated saline**. Preservation in rectified spirit may cause loss of phosphorus luminosity.
(3) Collect feces from rectum and submit for toxicology

9. Medicolegal importance

(1) **Long retention in organs after death** - Phosphorus continues to remain unchanged in the organs in fatal cases. It has been found in bodies up to one month after death. Can be detected even in exhumed bodies.
(2) **Homicide** - Because of its luminescence and strong garlicky odor, it is **very**

rarely used for homicide. In some cases it was mixed with coffee, soup, jam or rum, and given to the unsuspecting victim. In earlier days when match heads contained yellow phosphorus, match heads were **mixed in chewing tobacco** and given to the victim. Match heads were also **inserted into rectum** for homicide.

(3) **Suicide** – Deliberate ingestion of rat paste.

(4) **Accident** – Inadvertent ingestion of rat paste.

II. MECHANICAL IRRITANTS

Mechanical irritants are sharp or blunt ingested foreign bodies, which cause mechanical irritation upon ingestion.

Salient features:

(1) These should not be considered poisons in the true sense, as they do not cause death on a biochemical basis.

(2) However since they do cause death on ingestion, they have been described as poisons since long.

(3) These poisons act as **local irritants** and may cause **perforation**.

(4) Many of these substances are accidentally ingested by children.

(5) Important examples are: (i) bones [fish, chicken] (ii) button-type batteries [**ch 33**] (iii) chopped animal and vegetable hair (iv) coins (v) diamond powder (vi) jewellery (vii) magnets (viii) nails (ix) pins and needles (x) powdered glass and (xi) toys. (xii) Harmful substances that have been **reported in food** (especially packaged food) from time to time are **bone pieces**, **metal**, **cleats** (staple like wires used to hold together sections of conveyor belt in food industry), **fruit pits**, **herbaceous stems**, **rocks**, **insects** and small **animals**.

35. Agricultural Poisons

Agricultural chemicals or ***agrochemicals*** are chemicals used in agriculture. When ingested in toxic doses they may cause serious poisoning or death. Agricultural poisons include fertilizers, pesticides, grain preservatives etc.

I. CLASSIFICATION

A. Classification of Agricultural Poisons

1. Classification [According to toxicity]

Pesticides (Chemicals used to kill pests, a subgroup of agricultural poisons) may further be classified according to their toxicity.

(1) Least toxic [generally not fatal]

(i) Phenoxyacetic acid hormones [MCPA or 2-**M**ethyl-4-**C**hloro**P**henoxyacetic **A**cid (Agritox); DCPA or 2,6-**D**i**C**hloro**P**henoxyacetic **A**cid; TCPA or **T**ri**C**hloro**P**henoxyacetic **A**cid. They are inhibitors of growth induced by auxin. Used for weed control]

(ii) Cuprous oxide (Cu_2O, fungicide)

(iii) Lime sulphur washes (orchard fungicides)

(iv) Tar oil emulsion (orchard ovicides)

(v) Petroleum washes (orchard insecticides)

(vi) Weak solutions of sulfuric acid, nitric acid, pelargonic acid or acetic acid [Acids work by lowering pH level on target weeds just enough to kill them. The residues left behind by these natural weed killers are either harmless or useful to plants as fertilizer. Sulfuric acid, for example, oxidizes to form sulfates]

(vii) Natural fungicides (a) Tea tree oil (b) Cinnamaldehyde (c) Cinnamon essential oil (d) Jojoba oil (e) Neem oil (f) Rosemary oil.

(2) Mildly toxic [fatal dose >10 g]

(i) Chlorinated hydrocarbon insecticides [used as agricultural insecticides] (a) DDT (b) Gammexane (c) Methoxychlor (d) Chlordane, aldrin, dieldrin

(ii) Sodium chlorate [used as a mass herbicide].

(3) Highly toxic [fatal dose <10 g]

(i) Arsenical compounds [used as weed-killers and orchard insecticides] (a) Sodium arsenite (b) Lead and calcium arsenate (c) Copper Acetoarsenite (Paris Green)

(ii) Nicotine, sulphates, tannates [used as horticultural insecticides]

(iii) HCN, KCN, NaCN [as disinfectants and raticides]

(iv) Dinitro compounds [dinitrophenol (DNP), DNOC (dinitro-orthocresol). Selective weedkillers, ovicides and insecticides]

(v) Organophosphorus compounds [agricultural insecticides] (a) Tetraethylpyrophosphate (TEPP) (b) Hexaethyltetraphosphate (HETP) (c) Octamethylpyrophosphoramide (OMPA) (d) Parathion.

B. Classification of Insecticides

Insecticides (Chemicals used to kill insects, a subgroup of pesticides) may further be classified according to their origin.

(1) Insecticides of vegetable origin

(i) Nicotine (ii) Pyrethrins (iii) Rotenone

(2) Chemical insecticides

(i) Inorganic (a) Compounds of arsenic, barium, mercury etc. (ii) Organic (a) Organophosphorus compounds [OP] (b) Carbamates (c) Organochlorines [OC] (i) Indane derivatives (chlordane, aldrin, dieldrin)

(ii) Chlorobenzene derivatives (DDT)

(iii) Benzene hexachloride (lindane, gammexane)

(iv) Chlorinated camphenes (Toxaphene, camphechlor, polychlorcamphene, alltox, toxyphen, strobane-T).

C. Classification of OP

OP insecticides are divided into two main groups

(1) **Alkyl phosphates**

(2) **Aryl phosphates** depending on whether they have a **l**iner side chain [a**l**kyl] or **r**inged side chain [a**r**yl]. [1] **Alkyl phosphates** – Examples are (i) Demeton (ii) Dimefox (iii) **H**exa**E**thyl **T**etra**P**hosphate [HETP] (iv) Isopestox (v) Malathion (Killbug, Bugsoline) (vi) **O**cta**M**ethyl **P**yro**P**hosphoramide [OMPA] (vii) Sulfotepp (viii) **T**etra**E**thyl **P**yro**P**hosphate [TEPP] (Tetron) (ix) Trichlorfon. [2] **Aryl phosphates** – Examples are (i) Chlorthion (ii) Diazinon (Tik 20) (iii) Methyl parathion (Metacide) (iv) Paraoxon (v) Parathion [nitrostigmine] (Follidol, Killphos, Ekato).

D. Absorption

OPs are well absorbed from the (1) skin, (2) lung, (3) GIT (4) Mucus membranes

[eg conjunctiva] and (5) direct injection. Plants absorb OP compounds through leaves and stems.

E. Distribution

(1) widely distributed in the body
(2) Readily cross placenta
(3) Lipophilic"Thus cross BBB. Form deposits in fat and skin depending upon lipophilicity of individual OP.

F. Metabolism

(1) **Main sites** – of metabolism are liver and blood. Minor sites are CNS, GIT, Kidney, Lung and Skin.
(2) **Enzymes** responsible are
(i) A-esterases
(ii) Carboxylesterases [including B-esterases]
(iii) Fluorohydrolases and
(iv) Microsomal oxygenases [of these, cytochrome P450 (CYP enzymes) are most imp.]
(3) Metabolism can both activate and deactivate OP. Aryl OP require liver metabolism to become toxic.

G. Elimination

Prolonged over a week.

H. Mechanism of Action

OP are powerful inhibitors of carboxylic esterase enzymes, including acetylcholinesterase (true cholinesterase) and pseudocholinesterase. Mechanism may be understood in following steps:

(1) **Normal transmission of impulse by Ach** - With nerve impulse Ach is released in neuromuscular cleft where it attaches to Ach receptors and transmits impulse. It is then immediately deactivated by acetylcholinesterase situated much deeper in the cleft.
(2) **Normal deactivation of Ach** - Normally Ach binds to cholinesterase at 2 sites -

(i) The quaternary **n**itrogen of choline attaches at the a**n**ionic site [

n

egatively charged] **[Memory aid 1]**

(ii) Carbonyl group attaches to a serine residue at esteratic site.

Memory Aid 2: Esteratic site

Serine residue is at e **s** t **er**atic site. Phosphate group attaches here to deactivate **ChEase**.

(3) **Reaction** catalyzed by AChE is: Ach + H_2O"choline + acetate.

(4) **OP deactivate cholinesterase [ChE]:**

(i) by attaching a phosphate group to the hydroxyl group of a serine residue at the enzyme's active [esteratic] site.

(ii) The bond formed is strong and cannot be displaced even by high concentrations of acetylcholine.

(iii) Recovery from such inhibition generally takes 10–14 days.

(iv) Deactivation of ChE results in accumulation of Ach molecules at (a) ganglionic, (b) parasympathetic and (c) neuromuscular synapses, causing overstimulation.

(v) OP inhibit all carboxylic esterase enzymes of which ChE is just one. Another enzyme inhibited is Neuropathy target esterase [NTE], which is an integral membrane protein present in all neurons. This results in OPIDP [please see below].

(5) **Aging:**

(i) If cholinesterase reactivators [oximes] are not given in time, ChE undergoes a process known as "aging". It involves breaking away of one organic group [R1] from the OP-ChE conjugate (dealkylation). This makes the OP-ChE conjugate extremely stable and even oximes are unable to reactivate ChE after "aging".

(ii) Early administration - Thus oximes must be given within first few hours of poisoning.

(iii) The rate of aging - varies with the particular OP compound [10 min with soman; 48 hours with VX.].

I. Acute Poisoning

1. Clinical features

a. General considerations

(1) Ocular exposure causes persistent miosis

(2) Onset of symptoms most rapid following inhalation; least rapid following dermal absorption

(3) First affected are secretory glands and involuntary muscles; then voluntary muscles; and finally vital brain centers.

b. Signs and symptoms

i. Muscarinic manifestations

(1) **General** - Cyanosis

(2) Eyes -

(i) Pupils – (a) **Constricted** [mostly]. (b) ***Mechanism*** – peripheral. Local cholinergic action [cf, morphine"**ch40**]. (c) Occasionally unequal. (d) May be dilated rarely, if nicotinic effects prevail

(ii) Ciliary body – Blurred vision

(3) **Glands** – All secretions are -ed

(i) Sweat glands - -sweating

(ii) Lacrimal glands - (a) -lacrimation. (b) Chromodacryorrhea [shedding of **bloody tears**] due to disturbance of porphyrin metabolism [porphyrinemia]. Porphyrin accumulates in the lacrimal glands.

(iii) Salivary glands - -Salivation

(4) **Bronchial tree** -

(i) Bronchoconstriction

(ii) -ed bronchial secretions [bronchorrhea]. These two are referred to as the “killer B’s” of OP, as they are the leading cause of death.

(iii) Dyspnea

(5) **Lungs** – Pulmonary edema

(6) **CVS** -

(i) Bradycardia

(ii) Hypotension [rarely - opposite effects. It happens if nicotinic effects prevail]

(iii) ECG changes - (a) small voltage complexes (b) ST–T changes (c) idioventricular rhythms, (d) ventricular extrasystoles, (e) prolonged PR interval (f) polymorphic ventricular complexes.

(7) **GIT** - (i) Anorexia (ii) Nausea, vomiting (iii) Cramps (iv) Diarrhea (v) Fecal incontinence (vi) Pancreatitis. Pseudopancreatic cyst forms rarely. (vii) Tenesmus

(8) **Poisoning during pregnancy** -

(i) may mimic acute complications in pregnancy, eg eclampsia, seizures.

(ii) Spontaneous abortion.

(9) **Bladder** – Urinary incontinence.

ii. Nicotinic manifestations

(1) **Striated muscle** -

(i) Initially contraction [cramps]; later paralysis due to persistent depolarization [muscular weakness, areflexia]

(ii) fasciculations

(2) **Sympathetic ganglia** -

(i) Hypertension

(ii) Tachycardia

(iii) Pallor

(iv) Mydriasis [These effects are exactly opposite to muscarinic effects. Generally muscarinic effects are prevalent. Thus patient generally exhibits bradycardia, hypotension and miosis].

iii. CNS manifestations

(1) **Initially** - (i) Ataxia (ii) Confusion (iii) Emotional lability (iv) Generalized weakness (v) Headache (vi) Restlessness (vii) Slurred speech (viii) Tremor

(2) **Later** - (i) Depression of centers [resp and CVS] (ii) Drowsiness (iii) hypothermia (iv) Convulsions (v) Coma.

Memory Aid 3: Symptoms of OP Poisoning

There are 2 mnemonics to remember muscarinic symptoms of OP Poisoning – 2 for muscarinic; 1 for nicotinic and 1 for CNS manifestations.

[A] For muscarinic symptoms

1. **SLUDGEM**

Salivation

Lacrimation

Urination

Defecation

Gastrointestinal upset

Emesis

Miosis

2. **DUMBBELS**

Diarrhea

Urination

Miosis

Bronchorrhea

Bradycardia

Emesis

Lacrimation

Salivation, sweating

2. Lab diagnosis

a. Depression of cholinesterase activity

[A] **Two types of cholinesterases** - are found in the human body. (1) True cholinesterase [Acetylcholinesterase, AChE, type I ChE].

Salient features:

(i) Found in (a) motor endplates (b) RBC and (c) gray matter (ii) At motor endplates its function is to terminate nerve impulse transmission. It has a very high catalytic activity — each molecule of AChE degrades about 25,000 molecules of Ach/s. (iii) RBC cholinesterase – a very similar enzyme - is found in RBC membrane. Its function is unknown. Its levels are used to reflect neuronal cholinesterase levels because (a) it is biochemically and structurally quite similar to the neuronal AChE. (b) There is less inter- and intra-individual variability.

(2) Pseudocholinesterase – [Butyrylcholinesterase, BChE, type II ChE].

Salient features:

(i) Synthesized in liver (ii) Found in (a) plasma, (b) white matter and (c) visceral organs such as liver, heart and pancreas (d) GIT smooth muscle (e) Skin. (iii) Activity - True cholinesterase hydrolyses acetylcholine more quickly;

Pseudocholinesterase hydrolyses butyrylcholine more quickly. (iv) Physiological function - unknown. Postulated functions (a) role in the transmission of slow nerve impulses, (b) in lipid metabolism (c) a regulatory role in choline homeostasis (d) role in permeability of membranes (e) in the protection of fetus from toxic compounds (f) a role in the degradation of acetylcholine and (g) in tumorigenesis. (v) It hydrolyzes other esters on which **AChE** has no effect [eg suxamethonium (succinylcholine), mivacurium, ester local anaesthetics, diamorphine and aspirin]. Since it inactivates suxamethonium, it is of interest to anesthesiologists. People with congenital deficiency of butyrylcholinesterase can have hours of prolonged apnea after a normal dose of succinylcholine or mivacurium. [B] **Normal levels** – (1) Normal values of cholinesterase activity as measured by Michael's method [in **Δ**pH/hour/0.02 ml red cells or plasma at 25°C] are given in **Table 1**.

Table 1: Normal RBC and Plasma Cholinesterase activity in men and women

S.No.	Men	Women
RBC ChE	0.76	0.75
Plasma ChE	0.95	0.81

[C] **Levels of both RBC and plasma cholinesterase** - .ed in OP poisoning.

Salient features:

(1) RBC cholinesterase is a better indicator of OP poisoning.

(2) Interpretation of levels - (i) 25% or greater depression"indicator of poisoning (ii) 50% or greater depression"Clinical manifestations occur (iii) 90% or greater depression"Death occurs.

(3) Regeneration:

(i) Rate of RBC cholinesterase regeneration"**1%/day**

(ii) Rate of pseudocholinesterase regeneration"**3%/day**. Thus RBC cholinesterase falls slowly and regenerates slowly; pseudocholinesterase falls early and regenerates early. In severe poisoning [>90% depression],

RBC cholinesterase would regenerate to normal levels in 90 days; pseudocholinesterase would regenerate to normal levels in 30 days [**Fig 35.1**].

Table 2: Cholinesterase levels co-related with severity of poisoning

S.No	Severity of poisoning	%Cholinesterase depressed
1.	Latent poisoning	10-50%
2.	Mild poisoning	50-80%
3.	Moderate poisoning	80-90%
4.	Severe poisoning	>90%

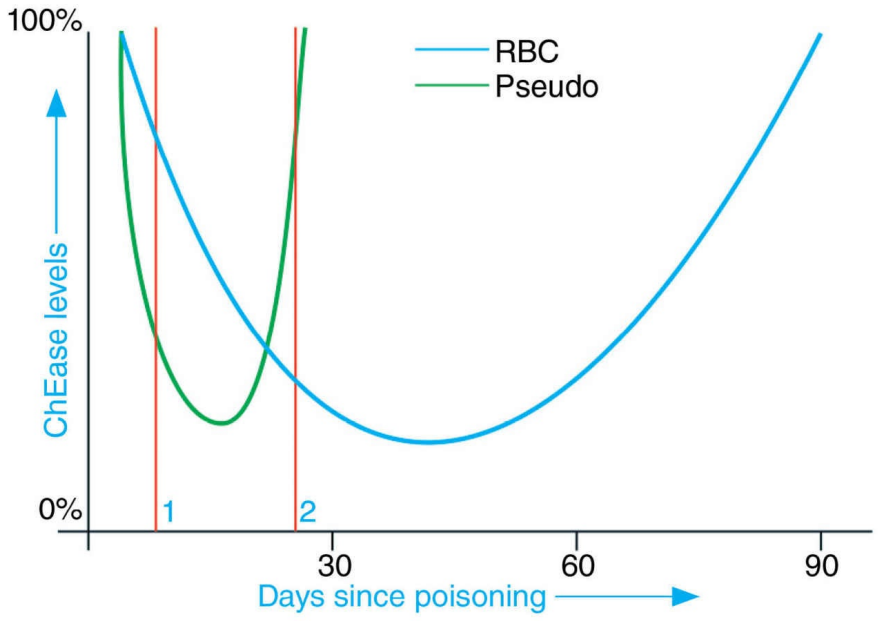


Fig. 35.1: Interpretation of **RBC** and **Pseudocholinesterase levels**. (1) Early poisoning (i) RBC: Normal (ii) Pseudo – ↓ (2) Late poisoning (i) RBC↓ (ii) Pseudo – normal.

b. Blood

(1) RBC cholinesterase activity as above

(2) Hyperglycemia [hyperactivity of sympathetic ganglia"release of catecholamines

(3) Hyperamylasemia.

c. Urine

(1) **P-Nitrophenol test** – P-Nitrophenol is a metabolite of several OP compounds and is excreted in urine. **Steam distillate of urine + NaOH"Yellow color** indicates presence of P-Nitrophenol.

(2) Glucosuria.

d. Thin layer chromatography

Ch 31.

e. Miscellaneous

Serum levels of OP – Not a prefereable method because (i) OP are difficult to measure, because they are active at very low levels (ii) the levels are different in different OP compounds (iii) Because of genetic differences, some people are poisoned by lower levels of OP than others.

3. Management

(1) **Prevention of further absorption** –

(i) Remove patient from site of exposure

(ii) Remove contaminated clothing

(iii) **Wash exposed areas** with soap and water, followed by ethanol in water

(iv) Topical creams and lotions.

(v) In case of **eye contamination** irrigate eyes thoroughly in running water for 15 minutes

(2) **Maintain airway** – **Most important step** as copious secretions may be blocking airways [cause of death in OP is respiratory failure]. (i) Clean airway of secretions. (ii) Endotracheal intubation, (iii) Administration of Oxygen. (iv) Positive pressure ventilation (v) **Tracheostomy** if necessary.

(3) **Removal of unabsorbed poison - Stomach wash** - with 1:5000 $KMnO_4$

(4) **Antidotes** -

(i) Mechanical – **Activated charcoal** 1g/kg

(ii) Physiological – (a) **Atropine sulphate** – (I) Dose - 2-5 mg IV every 5-10 min until complete atropinization (dry flushed skin, Pulse 140/min, pupillary dilatation) [pediatric dose 0.05 mg/kg]. A mild degree of atropinization should be maintained for at least 48 hours. (II) Must be given after cyanosis has been overcome. It is contraindicated in a cyanotic patient because of the possibility of inducing ventricular fibrillation. (III) Reverses the muscarinic and CNS actions of OP. No effect on nicotinic actions.

(5) **Oximes** – [A] **Mechanism of action** – (1) Main - Reactivate acetylcholinesterase by attaching to the phosphorus atom and forming an ***oxime-phosphonate*** which then splits away from the acetylcholinesterase molecule. (2) Prevent formation of phosphorylated enzyme (3) Potentiate actions of atropine; thus .the amount of atropine required (4) Directly detoxify OP. [B] **General points** - (1) Should be started as early as possible, otherwise aging occurs [please see above]. (2) Given until the patient is clinically well and not requiring atropine. [C] **Main oximes** - (I) Pralidoxime – (i) Most commonly used oxime (ii) Availability – Most commonly as (a) pralidoxime chloride [2-PAM, Protopam. Used worldwide]. Other forms (b) mesylate [P2S, used in the UK]. (c) Pralidoxime Iodide [2-**P**yridine **A**ldoxime **M**eth**I**odide, 2-PAMI] (d) methylsulfate [2-PAM methylsulfate] (iii) Dose - (a) 1-2 g in 100 mL of 0.9% NaCl sol^{n} given IV over 15-30 minutes. (b) Repeated in 1 h if muscle weakness and fasciculations are not relieved. Thereafter, additional doses may be needed every 3-8 h as long as signs of poisoning recur. (c) Maximum dose should not exceed 12 g in a 24 h period. (iv) Its action is marked at nicotinic sites, often improving muscle strength within 10-40 m. (v) Adverse effects – Rapid IV inj produces (a) temporary worsening of cholinergic manifestations [because oximes are themselves mildly anticholinergic. They bind to cholinesterase prior to regenerating its activity]. (II) Diacetylmonoxime [DAM] (III) **Hagedorn oximes** – [or H-Oximes]. These are newer oximes synthesized by Hagedorn and coworkers, because traditional oximes [eg pralidoxime, obidoxime] are ineffective against OP nerve gases [eg soman]. These are effective against all known OPs. **Ex** (i) **LüH-6** [syn by **Lü**ttringhaus and **H**agedorn] (ii) **HI-6** – or Asoxime chloride (iii) **HLö-7** [syn by **H**agedorn and **Lö**ffler]. (V) Methoxime [MMB-4] –

Particularly useful against nerve agents, eg sarin. Also used is 4-Pyridine aldoxime (4-PA), metabolite of MMB-4. (VI) **M**ono**i**so**n**itroso**a**cetone [MINA] (VII)Obidoxime Chloride – more potent, but more toxic also. Dose 250 mg IV or IM. (VIII) **Sugar oximes** - Traditional oximes [eg Pralidoxime and even HI-6] do not effectively cross BBB [penetration of 2-PAM is <10%]. Therefore, they do not provide protection against the neurological effects of OP-exposure [seizures, convulsions, behavioral and psychological changes]. Now new novel **sugar–oximes** have been developed, which cross the BBB because they contain a sugar moiety. (IX) Trimedoxime bromide [syn dipyroxime, TMB-4]

(6) **Control of convulsions** - (A) **Benzodiazepines** –

(i) Actions - (a) Anticonvulsant (b) .es synaptic release of ACh. Most useful agent is **diazepam**.

(ii) Dose [WHO recommendation]- (a) Adults - 5–10mg IV in absence of convulsions and 10–20mg IV in cases with convulsions. (b) Children - 0.2–0.3 mg/kg IV [maximum 5mg in children <5 y, and 10mg in children >5 y]. Given slowly over 3min. Repeated as required. May be repeated in adults every 10–15 min [max 30 mg]

(iii) Combination of atropine and diazepam is more effective in .ing mortality than atropine or oxime alone.

(iv) Beneficial effects - (a) .es anxiety, apprehension, agitation and restlessness (b) .es muscle fasciculation (c) arrests seizures, convulsions (d) Counteracts some effects of CNS toxicity which are not affected by atropine (e) .es morbidity and mortality when used in conjunction with atropine and an oxime. If convulsions persist, use (B) Phenobarbital – 10-20 mg/kg IV or (C) Phenytoin – 20 mg/kg IV. If status epilepticus develops give (D) General anesthesia

(7) **Plasma alkalinization** - with $NaHCO_3$ has been tried, but results are not encouraging.

(8) **Adjunct and alternative therapies** -

(i) Bioscavenger therapy - involves the use of enzymes such as cholinesterases (ChEs) in order to sequester OP compounds before they reach their physiological targets. Just as OPs are anticholinesterases, cholinesterases may be viewed as “anti-OP agents”. Main agents used are (a) cholinesterases and related enzymes [Purified human BChE, beta-

esterases]- They bind and neutralize OP. (b) OP hydrolases and anhydrases – These are bacterial enzymes, which have recently shown promising results in animal models. They catalytically hydrolyse and inactivate OP compounds;

(ii) Antibiotics – to prevent pulmonary infections.

4. Fatal dose

(1) **TEPP** [**T**etra**E**thyl **P**yro**P**hosphate] (most toxic) – 100 mg orally [50 mg IM]

(2) **OMPA [O**cta**M**ethyl-**P**yrophosphor**A**mide**]**, **Parathion** – 175 mg orally [80 mg IM]

(3) **HETP**[**H**exa**E**thyl **T**etra**P**hosphate] –350 mg orally [60 mg IM]

(4) **Malathion** and **Diazinon** – 1 g orally.

5. Fatal period

(1) Untreated cases – 24 hours

(2) Treatment given, but turned out unsuccessful – 10 days.

6. Cause of death

Respiratory failure – Due to

(1) Weakness of the muscles of respiration and

(2) Accumulation of excessive secretions in the respiratory tract.

7. PM appearances

a. External

(1) **Signs of asphyxia** - (i) congestion of face (ii) cyanosis of lips, nose, fingers and extremities. (iii) Blood stained froth at nose and mouth

(2) **Pupils** - constricted

(3) **Kerosene like odor** - from mouth, gastric contents and body [because OP are dissolved in Aromax, a petroleum derivative]

(4) **Insects and flies** - die immediately after they alight on an opened cadaver at autopsy.

b. Internal

(1) **Gastric mucosa** – (i) Congested (ii) Sometimes hemorrhagic (iii) Sometimes

found to contain an oily greenish scum [due to indigocarmine. Please see above].

(2) **Respiratory passages** - (i) Congested (ii) Contain frothy hemorrhagic exudate.

(3) **Heart** – soft and flabby. May show epicardial hgs especially in survival of more than a day.

(4) **Lungs** - (i) Gross congestion (ii) Sub pleural petechiae (iii) Hemorrhagic pulmonary edema.

(5) **Brain** - (i) Congested and edematous (ii) **Meninges** congested

(6) **Viscera** - (i) Congested (ii) Show petechial hgs [asphyxia]

J. Intermediate Syndrome

(1) Sometimes delayed muscle weakness without fascicu-lations or cholinergic features occurs **1-4 days** after acute OP exposure.

(2) **Signs and symptoms** - (i) Upper body weakness. The most commonly affected muscles are the facial, extraocular, palatal, respiratory, and proximal limb muscles. The muscle weakness can progress to respiratory distress and paralysis (ii) Cranial nerve palsies, (iii) Areflexia (iv) Fasciculations occur rarely (v) **Level of consciousness** is rarely affected.

(3) **Treatment** -

(i) Mainly **supportive** with airway protection and ventilatory assistance.

(ii) Pralidoxime or atropine are ineffective

(4) **Resolution** - Syndrome may automatically resolve within 5–18 days when the body burden of OP metabolites diminish and cholinesterase levels normalize.

K. OPIDP [Delayed Sequelae]

Organo**P**hosphorus **I**nduced **D**elayed **P**olyneuropathy [OPIDP] [sometimes also called **O**rgano**P**hosphorus **I**nduced **D**elayed **N**europathy (OPIDN)] is a delayed sequelae of acute exposure. It is due to inhibition of NTE [please see above under “mech of action”]. Most typically ***acute poisoning*** occurs within 1-4 hours; ***intermediate syndrome*** within 1-4 days; and ***OPIDP*** within 1-4 weeks after exposure. **Signs and symptoms** – Distal limb muscles are involved [*in contrast to proximal limb muscles in intermediate syndrome*].

Memory Aid 4: Signs and symptoms of OPIDP

In **D**elayed polyneuropathy, **D**istal muscles are involved

L. Chronic Poisoning

Chronic poisoning with OP is different than OPIDP. Chronic poisoning develops months or years after continued exposure. OPIDP on the other hand is merely a delayed sequelae of acute exposure.

(1) **Persons at risk:**

(i) Usually occurs as an occupational hazard in agricultural workers [eg those engaged in pesticide spraying of crops]

(ii) Persons engaged in manufacture and packaging of OP compounds

(iii) Research workers

(iv) Cholinergic ophthalmic preparations

(v) Family members of all workers exposed to OP

(2) **Absorption** - occurs through inhalation or skin contamination. Symptoms may appear after several months or years of low grade exposure.

1. Signs and symptoms [of chronic OP poisoning]

(1) **Neuropsychiatric symptoms** – Most characteristic and typical. (i) Personality change (ii) Mood destabilization (iii) Suicidal thinking (iv) Cognitive impairment (v) Language disorder - Word-finding and expressive disorders (vi) Handwriting deterioration - Significant decrease in legibility worsening with length of text (vii) anxiety (viii) confusion (ix) drowsiness (x) gait disorders [Parkinson disease like symptoms] (xi) irritability (xii) muscle cramps (xiii) paresthesias (xiv) weakness

(2) **Alcohol intolerance** - Marked increase in the inebriating effect of alcohol; may also involve severe hangover and quasi-allergic effects

(3) **Heightened sense of smell** - Typically to perfumes and scents; may be so severe as to make patients feel ill

(4) **Decreased exercise tolerance** - Normal initial muscle power, but inability to maintain it. This is not the same as generalized weakness.

(5) **Dipper's flu** - One or more episodes of severe flu-like symptoms lasting more than three days following exposure. May be accompanied by hypersalivation, abdominal cramps and diarrhoea. Symptoms may be triggered by extremely low concentrations of organophosphates.

2. Prevention

Workers in contact with OP compounds should have baseline **cholinesterase testing** for comparison and monitoring, especially **RBC cholinesterase**.

M. Medicolegal Importance [of OP]

(1) **Suicide** - very common. Alone or mixed with other poisons.

(2) **Homicide** – rare because of strong smell and bad taste

(3) **Organophosphates resist putrefaction** - Can be detected in the viscera for quite sometime after death.

(4) **Accident** – Quite common

(5) **Restricted Use Pesticide** (RUP) - A Restricted Use Pesticide is one that is available for purchase and use only by certified pesticide applicators or persons under their direct supervision. This designation is assigned to a pesticide product because of its relatively high degree of potential human and environmental hazard even when used according to label directions. Restricted use OP compounds in India are (i) Diazinon (ii) Fenitrothion, (iii) Fenthion, (iv) Methyl Parathion and (v) Monocrotophos.

II. CARBAMATES

Carbamates are organic compounds derived from carbamic acid (NH_2COOH).

Salient features:

(1) Marketed in the form of dusts or solutions eg Aldicarb [Temik], Aminocarb [Matacil], Carbaryl [Sevin], Carbofuran [Furadan], Fenobucarb [Merlino], Propoxur [Aprocarb, Baygon]

(2) **Table 3** shows some carbamates according to toxicity.

Table 3: Some carbamates according to their toxicity

Highly toxic	Moderately toxic
Aminocarb, Bendiocarb, Benfuracarb, Carbaryl, Carbofuran, Dimetan, Dimetilan, Dioxacarb, Formetanate, Methiocarb, Methomyl [Lannate], Oxamyl, Propoxur	Aldicarb, Bufencarb, Carbendazim, Isoprocarb, MPMC, Metolcarb [MTMC], Pirimicarb, Triallate [Avadex BW]

A. Toxicokinetics

(1) **Absorption** - Carbamates are rapidly absorbed from the small intestine or on inhalation. Peak concentrations occur rapidly.

(2) **Distribution** - Usually large V_d.

(3) **Elimination** - Metabolized in the liver. Excreted in the urine within several days. Aldicarb undergoes extensive enterohepatic recirculation.

B. Signs and Symptoms

Same as those of OP - It is helpful to visualize carbamates as "younger brothers of OP", in the sense that they produce similar symptoms, but much less pronounced. This is because (i) unlike OP, they hydrolyze spontaneously by cholinesterase within 24-48 h, whereas, OP does not (ii) They do not penetrate BBB; thus CNS toxicity is minimal.

C. Management

(1) **Atropine** – Drug of 1st choice

(2) **Oximes** – contraindicated [because it produces a carbamylated oxime, which is a more potent cholinesterase inhibitor than carbamate itself]. The only situations when oximes my be given in carbamates are

(i) Unknown pesticide ingestion – signs and symptoms similar to OP

(ii) Mixed [OP+carbamate] ingestion

(iii) Continued excessive requirements of atropine

(iv) Symptoms are life threatening [severe muscle weakness, paralysis, .ed respiration]

(3) **Adjunct therapies** – Same as in OP.

III. ORGANOCHLORINES

Organochlorines [OC] are organic compounds containing at least one chlorine atom, which are used as insecticides.

Salient features:

(1) OC are **nonselective insecticides**

(2) They are cyclic in nature, have molecular weights between 300 and 550 D

(3) They are CNS stimulants

(4) Poorly soluble in water but readily soluble in organic solvents, lipids and fats, which is why they accumulate in the human body [this is also why fat must be preserved in cases of organochlorine poisoning; please see **ch 5** – preservation of viscera].

(5) Very stable, both in the environment and in the body tissues, and can be demonstrated in the bodies of most people born since 1940.

A. Toxicokinetics

1. Absorption

(1) General - Absorption by any route depends on (i) vehicle and (ii) the physical state (solid or liquid) of the compound.

(2) Ingestion and inhalation - All OCs are well-absorbed by these routes.

(3) Transdermal absorption -

(i) Variable. Factors which enhance dermal absorption are (a) Nature of the compound (b) anatomic site

(ii) DDT, its analogues and toxaphene are very poorly absorbed.

2. Metabolism

OC are metabolized by the **hepatic microsomal enzyme systems** by dechlorination, oxidation, with subsequent conjugation.

(1) **OCs rapidly metabolized** (i) Endrin (ii) Lindane. Have less persistence in

body tissues

(2) **OCs slowly metabolized** - (i) DDT (ii) Dieldrin. Have significant adipose tissue storage especially in chronically exposed populations.

3. Excretion

(1) Primary route of OC excretion is **bile**.

(2) Most OCs have significant enterohepatic or enteroenteric recirculation [eg chlordecone, mirex]

(3) Excreted in maternal milk [because lipophilic]

(4) **Half-lives** - (i) lindane - 21 hours. (ii) Chlordecone, DDT – several months [because of fat solubility and poor metabolism].

B. Mechanism of Action

(1) Selectively bind to open (activated) **Na^+ channels** and slow their closing (inactivation), thus allowing the **influx of sodium ions**.

(2) The mechanism is quite similar to that of pyrethrins [please see below], despite their different chemical structures.

(3) The continuous inward ion current causes depolarization and repetitive firing of the nerve membrane.

(4) This is clinically seen as prominent tremors.

(5) DDT-induced tremors can be prevented by pretreatment with ***phenytoin***, a Na^+ channel blocker.

C. Acute Poisoning

1. Signs and symptoms

(1) **Prodromal signs or symptoms** – (i) agitation (ii) confusion (iii) dizziness (iv) headache (v) hyperesthesia and paresthesias of the mouth, tongue face and extremities (vi) leg weakness (vii) Nausea and vomiting. With lindane, the cyclodienes, and toxaphene, there are no prodromal signs or symptoms. Often the first manifestation of toxicity is a generalized seizure.

(2) **CNS stimulation** – (i) Myoclonus (ii) Tremors - May be the only initial manifestation after DDT exposure (iii) Seizures - (a) occur only after very high exposures (b) Occur within 1-2 hours of ingestion on an empty stomach; in 5-6 hours on a full stomach. (c) Dermal application – of 1% lindane for

treatment of ectoparasitic diseases may cause seizures. The time from application to seizure onset can vary from hours to days. (d) Status epilepticus - Seizures are mostly self-limited, but may recur or result in status epilepticus. (e) EEG abnormalities – occur before, during, and following seizures. (f) Recovery - If the seizures are brief and hypoxia has not occurred, recovery is usually complete.

(3) **Hyperthermia** – Occurs due to (a) -ed muscle activity (b) aspiration pneumonitis.

(4) **Pupils** - dilated

(5) **Synergy** - Ingestion of combinations results in significantly -ed toxicity [eg DDT and lindane]

(6) **Respiratory failure**, and death.

2. Fatal dose

(1) Aldrin, Dieldrin, Endrin – 2-6 g

(2) DDT, Lindane [also known as Benzene hexachloride (BHC), Gamma-hexachlorocyclohexane, (γ-HCH), Gammallin and Gammaxene] – 15-30 g. When lindane is ingested therapeutically as an anthelmintic at 40 mg/d for up to 2 wks, no symptoms are produced.

3. Fatal period

One to several hours.

D. Management

(1) **Measures for rescue personnel and bystanders** – should wear protective gear (apron, gloves etc), because OC may be absorbed through skin [being oil soluble].

(2) **Immediate resuscitative measures** – ABCDE of resuscitation [**ch 32]** (i) Move patient from toxic environment to fresh air. Monitor for respiratory distress etc. If present"start measures for respiratory insufficiency.

(3) **Clothing, jewellery etc** – remove immediately. Leather absorbs pesticides; discard all contaminated leather items.

(4) **Decontamination** -

(i) Contact – wash [skin, hair, nails, eyes, other exposed areas] copiously with water

(ii) Ingestion - (a) Emetics (b) Gastric lavage (c) Cathartics. Oil based cathartics are contraindicated [please see below].

(5) Activated charcoal

(6) Fats, oils etc not to be given by mouth.

(7) **Specific antidote** - None

(8) **Cholestyramine** -

(i) It is a non-absorbable steroid binding ion exchange resin. Binds bile acids, digitalis [**ch 43**]

(ii) -es fecal excretion of OC [especially chlordecone]

(iii) Dose – 4 g qid [16g/day] orally [mixed in fruit juice] for 2 weeks

(iv) Precautions – Interferes with absorption of other drugs, which must be administered either 1 h before or 4 h after administration of cholestyramine.

(9) **Symptomatic treatment** – Manage (i) Hyperthermia (ii) seizures – [**ch 32**]

(10) Calcium gluconate

(11) Contraindicated drugs -

(i) Adrenergic amines [eg adrenaline]. -myocardial irritability, produce refractory ventricular arrhythmias

(ii) Atropine

(iii) Oil based drugs [including cathartics].

(12) Hemodialysis, hemoperfusion – not effective.

E. PM Appearances

(1) **Mouth and stomach contents** – smell of kerosene

(2) Signs of asphyxia

(3) **Liver** - Centrilobular hepatic necrosis.

F. Chronic Poisoning

Many OCs, especially Chlordecone (Kepone) persist in the body and produce chronic toxicity. Symptoms are (1) ataxia (2) exaggerated startle response (3) intracranial hypertension (4) mental status changes (5) oligospermia (6) opsoclonus [rapid, irregular, dysrhythmic ocular movements] (7) rash (8) Tremor of head, hands and even entire body [**Kepone Shakes**] (9) weakness, (10) weight loss, (11) -ed liver enzymes (12) .ed sperm motility.

G. Specific Organochlorines

1. DDT

DDT is an organochlorine insecticide. It is a colorless, crystalline solid with a weak, chemical odor.

Salient features:

(1) It is nearly insoluble in water but has a good solubility in most organic solvents, fats, and oils.

(2) It is a restricted use OC in India.

2. Endrin

Endrin, a cyclodiene, was once used chiefly against insect pests of cotton (about 80% of total use), paddy, sugarcane, tobacco and in a limited way on grain crops.

Salient features:

(1) It is a colorless odorless solid, although commercial samples are often off-white.

(2) Active against a wide variety of insect pests (as well as rodents); hence is commonly known as **plant penicillin**.

(3) It has been banned in India and several other countries.

H. Medicolegal Importance [of Organochlorines]

(1) **Suicide** - common

(2) **Homicide** – rare because of strong smell and bad taste

(3) **Accident** – Quite common

(4) **Occupational exposure** – leads to suits for compensation

(5) **Endrin resists putrefaction**. Can be detected in the viscera for quite some time after death.

(6) **Banned OCs in India** - Following OCs are banned in India for manufacture, import and use (i) Aldrin (ii) Chlordane (iii) Dieldrin (iv) Endrin (v) Heptachlor (vi) Lindane (vii) Toxaphene

(7) **Restricted use OCs in India** are - (i) DDT (ii) Endosulfan (iii) Lindane [for def of restricted use insecticides, please see under MLI of OP].

IV. ALUMINUM PHOSPHIDE

Aluminum phosphide (AlP) is a lethal solid fumigant pesticide, insecticide and rodenticide.

Salient features:

(1) **Common outdoor and indoor pesticide** - in developing countries like India as it is (i) cheap (ii) effective (iii) free from toxic residue and (iv) does not affect seed viability. Considered an ideal grain and tobacco preservative in India.

(2) **Availability** -

(i) Available as dark brown or grayish green tablets of **3 g** each. Also available as 0.6 g pellets.

(ii) Size – diameter - 20 mm; thickness - 5 mm

(iii) Container - sealed in tens and twenties in airtight aluminum containers

(iv) Composition - Tablets are composed of pure AlP [the active ingredient, which releases phosphine gas; **56%**] and ammonium carbamate/carbonate/urea [the inert ingredient, which release CO_2 and NH_3 which prevents self ignition of phosphine gas; **44%**].

(v) Tablets of AlP are sometimes referred to as "Rice Tablets".

(3) **Phosphine** – On contact with moisture each **3 g** tablet liberates **1 g** of phosphine [kills insects and rodents in all stages of development] along with CO_2 and NH_3 [from inert ingredients].

(4) **Trade names** – Available in India under following trade names - **Alphos**, Bidphos, **Celphos**, Chemfume, Degesch, Delicia, Fumigram, Phosphotek, Phosphume, Phostoxin, **Quickphos,** Synfume and Talunex.

(5) Other pesticides similar to AlP in action are zinc phosphide [Zn_3P_2, please see below] and calcium phosphide [Ca_3P_2].

A. Toxicokinetics [Absorption and Excretion]

(1) **Absorption** –

(i) GIT - Rapidly absorbed from GIT by simple diffusion

(ii) Lungs – Phosphine liberated from AlP can be readily absorbed through lungs

(2) **Metabolism** –

(i) AlP – Some AlP is transported to liver, where it is slowly metabolized to phosphine, accounting for prolongation of symptoms.

(ii) PH_3 - Oxidized to oxyacids. Excreted in the urine as hypophosphite and also through the lung in the unchanged form.

B. Mechanism of Action

(1) **Formation of phosphine** - On coming to contact with moisture present in the air [or with HCl and water in the stomach], AlP liberates phosphine leaving behind aluminum hydroxide [or chloride], which is a harmless and non toxic **grayish white** residue:

(i) **$AlP+3H_2O$"$Al(OH)_3+PH_3$**- [in air or stomach]**.**

(ii) **$AlP+3HCl$"$AlCl_3+PH_3$**- [in stomach].

(2) **Mechanisms of toxicity of liberated phosphine:**

(i) **Esophagus** - Causes esophageal strictures by more than one mechanism (a) AlP – is thought to be **corrosive**. However it is controversial, since both unexposed and exposed tablets produce strictures. (b) Tablets of AlP are usually large"Get stuck in the oesophageal lumen causing local ulcerations due to some preservative or adulterant present within the tablet.

(ii) **Lungs** - Direct injury to alveolar capillary membrane.

(iii) **Liver** - Disturbance of hepatic fat metabolism.

(iv) **Heart** - Exact underlying mechanism of cardiotoxicity and acute circulatory failure not well understood.

(v) **Cellular level** - (a) Action on cytochromes - Phosphine reacts with cytochrome c and cytochrome c oxidase, inhibiting mitochondrial oxygen uptake [action similar to that of CN and H_2S (**ch 44**)]. (b) Blockage of oxidative phosphorylation (c) .catalase; -superoxide dismutase. (d) Formation of highly reactive hydroxyl radicals. Leads to (I) Lipid peroxidation [oxidative degradation of lipids; a process in which free radicals “steal” electrons from the lipids in cell membranes, resulting in cell damage]. (II) Oxidative stress"acute lung injury. .glutathione, .malonyldialdehyde indicate oxidative stress.

(vi) **Heme** - Phosphine reacts with the heme moiety of hemoglobin in the presence of oxygen.

C. Signs and Symptoms

Human toxicity occurs either due to the ingestion of AlP (commonest mode),

inhalation (uncommon) or even after absorption through the skin (rare).

1. Ingestion

Symptoms begin within half an hour. They are more pronounced if an unexposed tablet is consumed [liberates more PH_3]

(1) **GIT** – These are the initial and presenting symptoms

(i) Metallic taste

(ii) Excessive thirst

(iii) odor - Garlicky or of decaying fish [PH_3 is colorless and odorless in the pure form but due to the presence of substituted phosphines and diphosphines, the patient has this characteristic odor].

(iv) Burning epigastric pain

(v) Vomiting and Diarrhoea

(vi) **Esophageal strictures** – AlP produces esophageal strictures much like caustic-induced strictures [because of its corrosive action], in about $1/3^{rd}$ of patients. Onset of dysphagia is within 2 to 8 weeks of ingestion.

(vii) **Tracheoesophageal fistula** – for the same reason.

(2) **Hepatic:** (i) Hepatitis (ii) Hepatomegaly (iii) Jaundice

(3) **Pancreas** – pancreatitis

(4) **CNS** -(i) Restlessness (ii) Headache (iii) Dizziness (iv) Altered mental state (v) acute hypoxic encephalopathy (vi) convulsions (vii) coma. Patients may remain conscious till the late stage.

(5) **Hematological** – (i) intravascular hemolysis (ii) methemoglobinemia (iii) microangiopathic hemolytic anemia

(6) **CVS** -

(i) ECG abnormalities – May be attributed to .Mg levels [this is the basis of $MgSO_4$ therapy](a) rhythm disturbances, (b) ST-T changes and (c) conduction defects.

(ii) Arrhythmias

(iii) Myocarditis

(iv) Profound and refractory hypotension [in 75%-100% cases] - due to myocardial damage, peripheral vasodilatation and fluid loss.

(v) Subendocardial infarction

(vi) Shock

(7) **Respiratory** - (i) Cough (ii) Tachypnea and dyspnea (iii) Cyanosis (iv) Pulmonary edema (v) Respiratory failure.
(8) **Renal** - Renal failure
(9) **Metabolic** - Metabolic acidosis [due to the accumulation of lactic acid caused by blockage of oxidative phosphorylation and poor tissue perfusion]
(10) **Miscellaneous** – (i) Acute adrenocortical insufficiency (ii) Bleeding diathesis [due to widespread capillary damage] (iii) DIC (iv) hypo- or hyperglycemia (v) Muscle wasting (vi) Spontaneous ignition at nose or mouth – Due to liberated PH_3. (vii) Tenderness. All these are rarely seen.

2. Inhalation [of phosphine]

a. Acute toxicity

Circumstances of poisoning:

(1) **Opening a long closed fumigated godown** - Inhalation [of PH_3] may occur if a person opens a long closed godown of grains preserved with AlP.
(2) **During play** - Children have died playing on fumigated wheat for just 1 h.
(3) **During travel** - Deaths have been reported when people were travelling in fumigated boxcars containing grains fumigated with aluminum phosphide. In one report, the victims had been in the car for approx 16 h and had even periodically opened the hatch for fresh air as needed.
(4) **House near a storage facility** - Death can occur even if victim's house is a little away from a storage facility where grains are preserved with AlP.

i. Mild toxicity

(1) Nausea, vomiting, diarrhea, headache (2) Acute respiratory distress (3) Dizziness (4) Fatigue (5) Irritation of mucus membranes (6) Tightness in the chest.

ii. Moderate toxicity

(1) Ataxia (2) Diplopia (3) Incoordination (4) Jaundice (5) Muscular (6) weakness (7) Numbness (8) Paresthesias (9) Tremor (10) Paralysis.

iii. Severe toxicity

(1) Arrhythmias (2) Congestive heart failure (3) Pulmonary edema (4)

Convulsions (5) Coma.

b. Chronic toxicity

Chronic toxicity may occur due to repeated occupational exposure with AlP tablets [slow inhalation of PH_3]. Symptoms are (1) Anemia (2) Bronchitis (3) GIT, visual, speech and motor disturbances.

3. Complications

(1) Pericarditis (2) Acute CCF (3) Acute massive GIT bleeding (4) ARDS.

D. Management

Doctors, nurses, and all paramedical staff must take personal protection measures, including full face mask and rubber gloves during management. One should be beware of "spontaneous ignition" also.

(1) **Immediate:**

(i) If there is occupational or accidental exposure to phosphine gas - the patient should immediately be removed to fresh air. As PH_3 is absorbed through the cutaneous route, decontamination of skin and eyes must be carried out thoroughly with plain water as early as possible.

(ii) Confirm airway patency –At the hospital, protect airways with endotracheal tube to prevent aspiration pneumonitis

(iii) Start supplementary oxygen

(iv) Check for pulse

(v) Establish IV access, preferably central venous, to start normal saline and vasopressor therapy as appropriate

(vi) Monitoring of vitals

(vii) Investigations - ECG, chest X-ray, blood glucose, arterial blood gas, electrolytes including magnesium, routine hemogram, LFT and KFT. Repeated or continuous ECG and echocardiography can reveal cardiac dysfunction early.

(2) **Gastric lavage** – In cases of ingestion. But only after endotracheal intubation. $KMnO_4$ [1:10,000] is used as it oxidizes PH_3 to nontoxic phosphate. Repeated 2-3 times.

(3) **AC** - 100 g of **activated charcoal**

(4) **Antacids** – (i) Reduce stomach related symptoms. (ii) Reduce absorption of phosphine

(5) **Fats and oils** - (i) Mainly vegetable oils [coconut oil] and liquid paraffin. (ii) Inhibit PH_3 release from the ingested AlP.

(6) **Maintain adequate renal perfusion and urine output** - Phosphine excretion is -ed.

(7) **Magnesium sulphate** –

(i) May cause up to 50% .in mortality.

(ii) Mechanism of action - (a) cell membrane stabilization ".es incidence of fatal arrhythmias (b) combats free radical stress due to phosphine. (c) Corrects hypomagnesemia and arrhythmias.

(iii) Dose –1g stat, then 1 g every hour for the next 2 h and then 1–1.5 g every 6 h for 5–7 days.

(8) **Cathartics** - Sorbitol [1–2 ml/kg]

(9) **Hemodynamic Support:**

(i) Rationale - Necessary because myocardial injury and hemodynamic instability are major features.

(ii) Objectives - to attain adequate tissue perfusion and oxygenation and physiologic metabolic milieu compatible with life

(iii) Measures undertaken - (a) continuous invasive hemodynamic monitoring (b) IV fluids - 4-6 L of fluids during first 3-6 h, of which 50% must be normal saline. Fluid therapy should be guided by central venous pressure (CVP) or pulmonary artery wedge pressure (PAWP) monitoring. (c) vasoactive agents - For refractory hypotension [Norepinephrine, phenylephrine]. Vasoactive agents with more β-receptor agonist action [dobutamine, dopamine (4-6μg/kg/min)] should be used cautiously as they are prone to inducing arrhythmias. (d) following should be readily available at bedside for emergency use - (I) Anti-arrhythmic agents, (II) DC cardioversion (III) temporary pacemaker. (e) Repeated echocardiography – to assess the reversibility of myocardial injury. (f) Advanced measures - (I) intra-aortic balloon pump (IABP) to mechanically support the heart (II) extracorporeal life support (ECLS) for intractable circulatory collapse.

(10) **Early Identification and Managing Other Organ Failures:**

(i) O_2 therapy – for hypoxia (a) If no response, then rule out methemoglobinemia by (I) multiple wave length co oximetry or (II) plasma level of

methemoglobin. (b) If methemoglobinemia present – IV methylene blue [1% sol^{n}] 2 mg/kg over 5 mins. Repeated if cyanosis is not resolved.

(ii) Endotracheal intubation and mechanical ventilation - for acute lung injury.

(iii) IV sodium bicarbonate - for metabolic acidosis

(iv) Hemodialysis - Not very effective in removing phosphine but is helpful when renal failure, severe metabolic acidosis or fluid overload is present.

(v) Peritoneal dialysis – if hemodialysis not available or possible

(11) **For survivors –** Mortality is very high [35-100%] despite institution of treatment. Following measures for survivors:

(i) If swallowing difficulty - conduct barium swallow study and upper GIT endoscopy for detection of esophageal strictures [late complication]. If present - (a) Endoscopic dilation of esophagus. About 5 dilations are required. (b) Intralesional steroids to augment the effect of dilation. (c) surgical intervention, if all other measures fail.

(ii) Psychosocial counselling – (a) to cope with possible reduction of quality of life due to late complications (b) to prevent repeat suicidal administration.

E. Diagnostic Tests

(1) **Tests for phosphine** – AlP liberates Phosphine [please see above]

(i) Live patients - (a) Gastric aspirate – (I) Procedure - 5ml gastric aspirate + 15ml H_2O"Put in a flask"Cover mouth with filter paper impregnated with 0.1N $AgNO_3$"Heat at 50°C for 15 min"If AlP is present in gastric aspirate, phosphine will form and blacken the filter paper due to formation of silver phosphide. **PH_3+3$AgNO_3$"Ag_3P+3HNO_3.** (b) Face mask – A filter paper is impregnated with 0.1 N $AgNO_3$ and used as a face mask. Patient is asked to breathe in and out of this filter paper for 15 min. If phosphine is present, filter paper will turn black. The test is +ve only if the patient has ingested >6g AlP. Thus it is less reliable

(ii) PM Samples – (a) Use gastric contents as gastric aspirate above. (b) Gas chromatography with a nitrogen–phosphorous detector - is the most specific and sensitive test. It can be used for analysis of airtight samples [viscera and gastric content] collected during autopsy.

(2) -in SGOT and SGPT, leucine aminopeptidase, aldolase, alkaline phosphatase, and albumin. [-ed liver enzymes are due to cell death and loss of cell

membrane integrity].

(3) **Magnesium levels** - Both hypo- and hypermagnesemia may occur. Their pathogenesis is not clear. There is a direct relationship between ECG changes and .Mg levels. The mortality is also related directly to hypomagnesemia.

F. Fatal Dose

(1) **AlP** -

(i) Ingestion of 1-3 tablets [**3-9 g**].

(ii) Since active ingredient [AlP] is only 56% in the tablet, the adjusted fatal dose of pure AlP is **1.5-5g**. Survival is unlikely if more than 1.5 g is ingested.

(iii) Survival has been reported after ingestion of 9.0 g or more. Possible explanations - vomiting, exposure of tablets before ingestion, early availability of supportive care.

(2) **Phosphine** -

(i) The permissible exposure limit of phosphine is **<0.3 ppm** in the working environment.

(ii) Inhalation of **0.25 ppm** (0.35 mg/m^3) of PH_3 for 8 hours can cause serious illness

(iii) Inhalation of **7.2 ppm** (10 mg/m^3) of PH_3 for 10 min is fatal.

(iv) **>50 ppm** - dangerous to life.

(v) **400–600 ppm** - Lethal within **30 min**.

G. Fatal Period

24 hours. Range 1 h-4d

H. Cause of Death

(1) **Within 24 h** - (i) Cardiogenic shock [CVS failure] (ii) hemodynamic instability (iii) Myocardial injury (iv) arrhythmias

(2) **After 24 h** - (i) acidosis (ii) ARDS (iii) refractory shock.

I. PM Appearances

1. Gross

(1) **Garlic like odor** – at mouth, nostrils, stomach contents. Very specific
(2) **Blood stained froth** – at mouth and nostrils
(3) **Congestion** – lower part of esophagus, stomach, duodenum, intestines, all internal organs.

2. Histopathological

Suggestive of cellular hypoxia.

(1) **Stomach** – (i) Congestion (ii) Edema (iii) Leukocytic infiltration (iv) Sloughing of gastric mucosa
(2) **Heart** – (i) Congestion (ii) edema (iii) Fragmentation of fibres (iv) Focal necrosis (v) Leukocytic infiltration (vi) Hemorrhagic myocardial lesions (vii) Myocardial muscles show (a) myocyte vacuolation, (b) areas of myocytolysis and (c) degeneration.
(3) **Lungs** – (i) Congestion (ii) Desquamation of respiratory epithelium (iii) Edema (iv) Lymphocytic infiltration (v) Thickened alveoli (vi) Diffuse vessel injury with edema and atelectasis
(4) **Brain** – (i) Congestion (ii) edema (iii) Disorganization of different layers, (iv) paucity of glial cells, (v) degeneration of neurons and (vi) appearance of necrotic patches.
(5) **Liver** – Centrizonal hemorrhagic necrosis
(6) **Renal** – (i) Congestion (ii) Cloudy swelling of renal tubular epithelia (iii) Acute tubular necrosis.
(7) **Suprarenal** – (i) Congestion (ii) Hemorrhage (iii) Necrosis (iv) Lipid depletion in cortex.

J. ML Importance

(1) AlP is a **restricted use pesticide [RUP]** in India. [for def of restricted use insecticides, please see under MLI of OP]
(2) **Suicidal poison** - Most common cause of suicidal poisoning in northern India. Cases of poisoning were first reported in India in **1980**. Before that poisoning in India was unknown.
(3) **Accidental** – occasionally
(4) **Homicidal** – rare. Difficult because of smell.

V. ZINC PHOSPHIDE

Zinc phosphide [Zn_3P_2] is a popular rodenticide available in India as dark grey tetragonal crystals or crystalline powder.

Salient features:

Zinc phosphide used as rodenticide comes as a black powder containing 75% of zinc phosphide and 25% of antimony potassium tartrate, an emetic to cause vomiting if the material is accidentally ingested by humans. However, it is still effective against rats and mice because they do not have a vomiting reflex.

A. Mechanism of Action

On coming to contact with moisture present in the air, zinc phosphide liberates phosphine [just like AlP]: **$Zn_3P_2+6H_2O"3Zn(OH)_2+2PH_3$-.** Toxicity is due to phosphine. Please see above under Aluminum phosphide.

B. Uses

Grain preservation, rat poison.

C. Symptoms

Same as those by Aluminum Phosphide, but are slower to start, because of slower release of phosphine.

D. Fatal Dose

5 g.

E. Fatal Period

24 h.

F. Management

Same as that of AlP.

G. PM Appearances

Same as those of AlP.

36. Metallic Irritants

I. ARSENIC

Metallic arsenic is not poisonous, as it is insoluble in water and is not absorbed from the alimentary canal. When heated, it volatilizes, combines with oxygen and forms arsenic trioxide (As_2O_3), which is poisonous.

A. Toxic Salts of Arsenic

(1) **Arsenious oxide** [Arsenic trioxide, As_2O_3].

(i) **Vernacular names** - Also known as sankhya, somalkhar, white arsenic [or simply arsenic]

(ii) **Occurrence** - Occurs in two forms – (a) powder and (b) porcelain type solid mass.

(iii) **Physical properties** - (a) White in color, tasteless and odorless; thus could be easily mixed with foodstuffs for homicide. (b) Sparingly soluble in water. (c) Sublimates on heating. (d) Although heavier than water (sp gr 3.669), floats on it as a thin film.

(iv) **Legitimate uses** - **Non-medicinal:** Used in artificial flowers, calico printing, fly papers, fruit sprays, as mordant in dyeing, preservation of timber against white ants, rat poisons, Sheep dips [liquid insecticide used by farmers to protect their sheep against ticks and lice], taxidermy, wall papers and weed-killers.

(2) **Arsenic acid** [H_3AsO_4]

(3) **Arsenic trichloride** ($AsCl_3$)- Butter of arsenic. Colorless poisonous oil

(4) **Arsenic trisulfide** (As_2S_3) comes as the mineral orpiment [hartal in India]

(5) **Copper acetoarsenite** [**Paris Green**, **Emerald Green**, **Schweinfurt Green**] – Once used as a pigment and insecticide

(6) **Copper arsenite** [**Scheele's Green**, $CuHAsO_3$]. Made by Swedish chemist **Carl Wilhelm Scheele** in **1775**. Initially used as a green pigment. Not used now due to its toxicity

(7) **Tetraarsenic tetrasulfide** (As_4S_4) comes as the mineral realgar (manseel in India).

B. Mechanism of Action

Arsenic is absorbed through all portals of entry (oral, nasal, cutaneous).

(1) **Trivalent arsenic** (As^{3+}) has a great affinity for **sulfhydryl groups [SH groups]**. Binds to mitochondrial membrane SH groups"damages them"Cytochrome c is released from the damaged mitochondria"activation of caspases"Apoptosis [**arsenic induced apoptosis**]. Because of -er affinity for SH groups [and also RBCs], the ***trivalent form*** is more toxic than ***pentavalent form*** [same is true of antimony also].

(2) Downregulates gene expression of **BCL2**, a **prosurvival protein**, that protects against apoptosis

(3) **Inhibits pyruvate dehydrogenase (PDH) complex** leading to disruption of oxidative phosphorylation.

C. Toxicokinetics [Absorption, Distribution and Elimination]

1. Absorption

(1) **Average daily intake** - is ½ -1 mg [contained in food and water].

(2) Well absorbed from the GIT (pentavalent As), respiratory tract (arsine), or skin (arsenite).

(3) Upon absorption, it is bound to the protein portion of Hb and **a**-globulins.

2. Distribution

(1) Once absorbed, arsenic is quickly distributed to all organs and tissues.

(2) In early stages, arsenic is found maximally in liver [in fatal cases >1mg%] followed by kidneys and spleen.

(3) Does not cross **blood-brain barrier.** Brain has lowest levels

(4) Inorganic arsenic can cross placenta

(5) **In ch poisoning:**

(i) Found in **muscles** for several days, and in **keratin containing tissues** [skin, hair, nails] for years. Can appear in hair and nails within hours of ingestion. Attaches itself to –SH groups which are abundant in keratin. In intermittent

ch poisoning, there are successive deposits of As in hair and nails, and their sequential analysis can determine even the dates on which As was administered [normal growth of hair 0.4mm/day – **ch 3**].

(ii) Gets incorporated in the **bone** by replacing P.

(6) **Normal conc in hair** – 2ppm

(7) **Normal levels in urine** <0.03mg/L.

3. Elimination

(1) **Mainly by kidneys:**

(i) As methylated As.

(ii) Found in urine within ½ h of ingestion. Then onwards, elimination is continuous for about 10-12 days.

(iii) In acute poisoning 24 h excretion > 100 mg

(2) **Breast milk** – eliminated only in traces

(3) **Bile, feces, sweat, other secretions** – Other routes of elimination

(4) **Has an enterohepatic circulation** – Because of this, As is seen in stomach and intestines, even if given by parenteral routes.

D. Acute Poisoning

Occurs within 24 h of ingestion.

1. Signs and symptoms

a. Ingestion

(1) **GIT:** (i) Odor – Garlicky (ii) Taste – sweetish metallic (iii) Intense thirst, Ptyalism (iv) Nausea, Vomiting [projectile in nature] (v) Throat – feeling of constriction, dysphagia (vi) Burning and colicky pain – in esophagus, stomach and bowels (vii) Abdominal pain (viii) Diarrhea - (a) Accompanied by pain, tenesmus and irritation around the anus. (b) Stools – (I) expelled frequently and involuntarily (II) Color and odor - dark colored, stinking, bloody; later become colorless, odorless and watery resembling rice-water stools of cholera. (c) May mimic bacterial food poisoning or cholera [**Table 1**].

(2) **Ocular:** (i) Conjunctivitis (ii) Lacrimation

(3) **Respiratory** - Irritation of upper airways

(4) **Liver** - Fatty degeneration

(5) **Renal:** (i) Pain - during micturition (ii) Oliguria (iii) Uremia (iv) Urine – contains albumen, casts and RBCs

(6) **Muscular** – Tenderness of muscles

(7) **CNS:** (i) Convulsions and tremors (ii) Formication (iii) Giddiness (iv) Headache (v) Vertigo (vi) General paralysis (vii) Delirium (viii) Coma

(8) **CVS:** (i) Cardiac arrhythmias [ST-T wave changes, prolonged QT interval], ventricular fibrillation (ii) Hypotension (iii) Ventricular tachycardia (iv) .ed vascular permeability, vasodilation and acute circulatory collapse

(9) **Skin** – loss of hair [in case of survival], skin eruptions

(10) **General** – hyperthermia [Hyperpyrexia].

Table 1: Differences between Arsenic poisoning and Cholera

S.No	Feature	Arsenic poisoning	Cholera
1.	Pain in throat	Before vomiting	After vomiting
2.	Sequence of vomiting and purging	Vomiting → Purging	Purging → vomiting
3.	Vomitus	Contains mucus, bile and blood	Watery - without mucus, bile and blood
4.	Stools	Dark colored, blood; later rice watery	Rice watery, no blood; passed in a continuous involuntary jet
5.	Tenesmus	Present	Absent
6.	Irritation and pain around anus	Present	Absent
7.	Voice	Not affected	Rough and whistling
8.	Conjunctiva	Inflamed	Not inflamed
9.	Lab Investigation	Arsenic in stools	No arsenic
10.	Stool microscopy and culture	No *Vibrio cholerae*	*Vibrio cholerae* present
11.	Circumstantial evidence	Arsenic may be found in victim's possession if suicidal; in the possession of enemy if homicidal.	Evidence of having taken infected food.
12.	Number of victims	Generally one	Generally several
13.	Motive	Suicidal, or homicidal motive is usually present	Neither suicidal nor homicidal
14.	Accidental cases	May occur	Generally accidental

Presentation

Depending on dose ingested, and retention of arsenic within the system, the patient may present in 3 major forms

(1) **Fulminant type:**

(i) Dose ingested is >10 times the fatal dose [3-5 g].

(ii) CVS symptoms are prominent, with almost complete absence of GIT symptoms.

(iii) Death occurs within 1-3 h from shock and peripheral vascular failure.

(2) **Gastroenteric type:** (i) dose ingested – around normal fatal dose. (ii) Classic type (iii) GIT symptoms are prominent

(3) **Narcotic type** - CNS and muscular symptoms are prominent; GIT symptoms insignificant.

b. Inhalation [Arsine gas]

(1) Hemolysis [**Fig 36.1**] (2) hemoglobinuria (3) renal failure (4) death is almost instantaneous.

Fig. 36.1: Arsine hemolysis. The bag on the right represents the plasma removed from an arsine poisoned patient compared with normal fresh frozen plasma on the left.

2. Fatal dose

(1) **As_2O_3** - 200-300 mg

(2) **Arsine gas** – 25-30 ppm is lethal in 30 min.

3. Fatal period

1-2 days.

4. Management [Ac arsenic]

(1) Emetics – Ipecac

(2) Gastric lavage – Repeated washings [to remove As adherent to walls] with warm water and milk [acts as a demulcent]

(3) Demulcents – Butter, greasy substances [to reduce absorption]

(4) **Whole bowel irrigation** – If radiopaque material is visualized in the GIT. Continue till the material is not visualized on repeat x-ray

(5) **Continued nasogastric suction** – Must be done because arsenic is resecreted in gastric tract. It has been detected in the GIT 7 days after ingestion

(6) **Activated charcoal** – Arsenic poorly adsorbs to activated charcoal. Thus it should only be administered in heavy ingestions.

(7) Cathartics – Castor oil, $MgSO_4$.

(8) Supportive care in ICU

(9) Chelation therapies:

(i) BAL and BAL analogues [**ch 32**].

(ii) **D-Penicillamine** (Cuprimine, Depen) – May be used with BAL [**ch 32**]

(10) **Immunotherapy** - Arsanilic acid is conjugated to a carrier protein [ovalbumin] and injected in small multiple dosages [100µg] in mice to produce active immunization against arsenic. Serum from such mice is then transferred to other mice [passive immunization]. Both actively and passively immunized mice did not succumb to ordinary lethal dose of arsenic. Shows promise in humans also.

(11) **Symptomatic:** Shock – Glucose saline.

(12) **Older remedies:**

(i) Freshly prepared hydrated ferric oxide.

(ii) Dialyzed iron – For details of both, please see **ch 32** under the heading "chemical antidotes". None is used now.

Memory Aid 1: Arsenic poisoning [main points]

ARSENIC - used in **A**bortion stick, **A**phrodisiac, found in charred bones and **A**shes, **A**ldrich-Mees lines,

Raindrop pigmentation, **R**ed velvety appearance of stomach mucosa, **S**ubendocardial hgs of heart, **E**CG changes, **N**europathy (motor & sensory), optic **N**euritis, **I**ron preparations [older remedy], **C**holera like symptoms

5. PM appearances [Ac arsenic]

a. External

(1) **Signs of dehydration** – **Eyeballs, s**unken; **body**, shrunken
(2) **Skin** – Cyanosed.

b. Internal

(1) **Mouth, pharynx, esophagus** – Generally unaffected, but rarely may be inflamed or ulcerated
(2) **Stomach** – displays major findings.
(i) Mucosa - (a) swollen, edematous and bright red, either diffusely or in patches. (b) Shows erosions and ulcerations (c) Mass of sticky mucus covers mucosa in which particles of arsenic may be seen
(ii) Congestion – Most marked along greater curvature, posterior part and cardiac end of stomach. More prominent at the crests of rugae. Lines of redness run along the walls. Rarely there may be no congestion
(iii) Submucous hemorrhages – in curved lines
(iv) Red velvety appearance – stomach wall is soft and red like a valvet [**Fig 36.2**].
Groups of petechiae scattered over mucosa. Sometimes large submucosal and subperitoneal hemorrhages.
(v) Pyloric region - is especially affected.
(vi) If putrefaction has occurred – yellow streaks are found in the subperitoneal layer of the stomach and to a lesser extent in intestines [As converts to yellow sulphide due to H_2S gas]

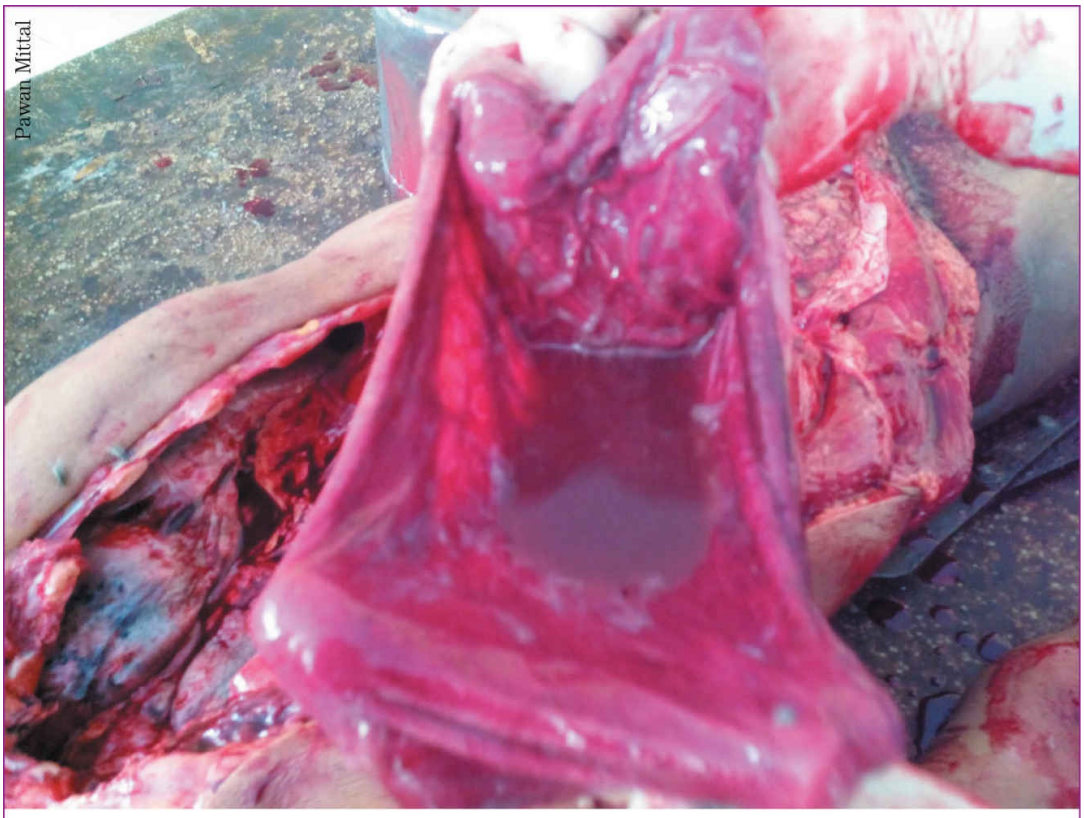


Fig. 36.2: Velvety red stomach mucosa in acute arsenic poisoning.

(3) **Small intestine:**
(i) Appears flaccid
(ii) Mucosa – Inflamed, pale violet. Shows submucous hemorrhages along the entire length
(iii) Contains large flakes of mucus with very little fecal matter
(4) **Cecum and rectum** – slightly inflamed
(5) **Liver, spleen, kidneys:**
(i) Congested, enlarged, show cloudy swelling and if survival is for few days, fatty change.
(ii) Glomerular nephritis
(6) **Hemorrhages** – in all abdominal organs, mesentery and occasionally in larynx, trachea and lungs
(7) **Lungs** – congested with subpleural ecchymoses
(8) **Heart:**
(i) Subendocardial petechial hemorrhages [SEPH] of the ventricles. May be seen even in absence of gastric inflammation.

(ii) Conditions in which SEPH are found - (a) Acute infectious diseases [eg influenza], (b) Heat stroke, (c) Poisonings [Arsenic, Barium, Mercury, Phosphorus] (d) Traumatic asphyxia

(iii) If survival is in days – fatty change

(9) **Brain:** (i) Edema - with patchy necrosis or hemorrhagic encephalitis (ii) Meninges – congested.

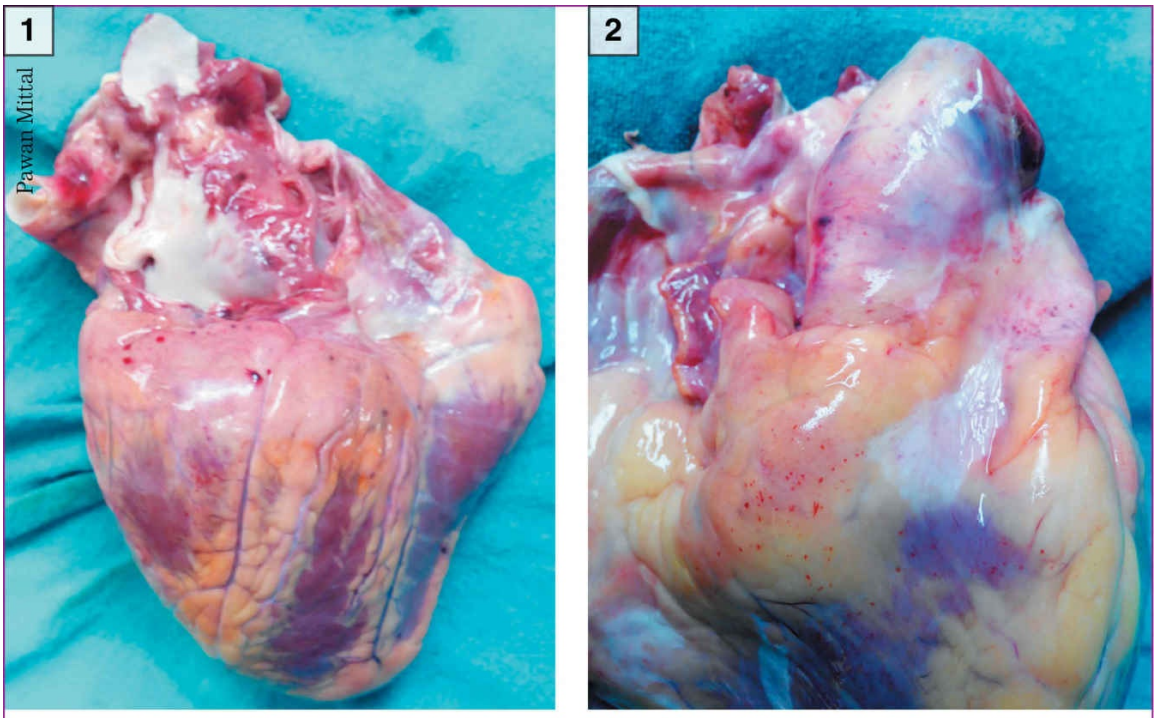


Fig. 36.3: Subpericardial hemorrhages in acute arsenic poisoning **(1)** Anterior surface **(2)** Posterior surface

E. Chronic Poisoning

WHO defines ***chronic arsenic poisoning*** [syn, ***arsenicosis***] as a chronic health condition arising from prolonged ingestion [not less than six months] of arsenic above a safe dose, usually manifested by characteristics skin lesions, with or without involvement of internal organs. Some authorities consider the period of onset as within 4 wks of continuous ingestion.

Salient features:

Occurs in following circumstances:

(1) **Occupational** - due to repeated accidental ingestion of small doses of arsenic

by those working with the metal

(2) **Through contaminated water** - People ingesting arsenic through **water contaminated with arsenic** (as in several parts of West Bengal and Bangladesh)

(3) **Arsenophagists** [please see below]

(4) **Homicidal** – when someone gives repeated doses to his enemy

(5) **Recovery from single heavy dose.**

1. Signs and symptoms

Occur in 4 stages.

a. 1st stage [Gastrointestinal]

(1) **General:**

(i) Circumscribed edema of lower eyelids and ankles [due to localized transudation of intravascular fluid]

(ii) Loss of weight

(iii) Malaise

(iv) Pulse rate-

(v) Temp-

(2) **GIT:**

(i) Loss of appetite

(ii) Gums – (a) red and soft (b) Black line on gums due to arsenic sulphide

(iii) Tongue – coated with a thin, white, silver fur

(iv) Nausea, vomiting [vomitus contains mucus tinged with bile]

(v) Salivation

(vi) Abdominal cramps

(vii) Colicky pain

(viii) Constipation. Sometimes diarrhea.

b. 2nd stage [Dermatological]

(1) **Dermatological:**

(i) Hyperkeratosis – (a) Discrete, multiple, hard wart like growths appear on (I) palms and soles (II) head and trunk [Less commonly]. (b) Due to epithelial hyperplasia (c) Time taken – few months to 30 years (d) Complications – may undergo a malignant change"(I) basal cell carcinoma (II) Carcinoma in

situ [**Bowen's disease**]. Arsenic is teratogenic. Can result in lung and skin cancers, leukemias [please see hematologic changes below].

(ii) Pigmentation [melanosis]– (a) Color - finely mottled brown (b) site – skin flexures of eyelids, temples, neck and other areas [**raindrop type pigmentation**]. Covered parts are also affected (c) Persistence – for months

(iii) Rash – resembling fading measles rash [d/d]

(iv) Flexural eczema

(v) Hair – become dry and fall off

(vi) Nails - (a) irregular thickening (b) brittle (c) Mees' lines. [As in subacute poisoning]

(2) **Eyes:** (i) Congestion (ii) Lacrimation (iii) photophobia (iv) dimness of vision

(3) **Nose:**

(i) Intense coryza [inflammation of mucous membranes lining the nasal cavity]

(ii) Running of mucus

(iii) Nasal septum – painless perforation

(4) **Throat:** Feeling of dryness and itching

(5) **Voice** – hoarse and dusky

(6) **Larynx and bronchi:** inflammation"Spasmodic cough with bloody expectoration, dyspnea

(7) **Hepatic:** (i) Cirrhosis (ii) Hepatomegaly (iii) Jaundice

(8) **Cardiorenal:** (i) Myocarditis (ii) Cardiac failure (iii) Chronic nephritis (iv) Dependent edema.

c. 3rd stage [Neurological]

(1) **General** - edema of lower eyelids and ankles worsens

(2) **CNS:**

(i) Anesthesia, hyperesthesia, Paresthesia

(ii) Encephalopathy

(iii) Polyneuritis [glove & stocking type]– headache, numbness, tingling

(iv) Knee jerks - lost

(3) **Muscles** – Marked tenderness, cramps

(4) **Joints** – arthralgia

(5) **Hematologic:** (i) Anemia – (a) due to bone marrow suppression [aplastic] and hemolysis. (b) type - Normocytic, normochromic. interference with the absorption of folic acid also occurs, which complicates the picture with

megaloblastic anemia. (c) Bone marrow examination shows bizarre nuclear forms and karyorrhexis (ii) Leukemia (iii) Thrombocytopenia

(6) **Impotence**.

d. 4th stage [Neuromuscular]

(1) Peripheral neuritis and muscular atrophy predominate

(2) **General:** (i) Anemia (ii) General emaciation

(3) **Muscular:** (i) Ataxia (ii) Atrophy of extensor muscles [wrist drop, foot drop] (iii) Interossei and intercostal muscles are affected (iv) Muscular weakness (v) Tremors

(4) **Kidneys** - Dysuria

(5) **CNS** - Delusions

2. Cause of death

Cardiac failure.

3. Diagnosis

(1) 24-h urinary As >67 µmol/d [>50 µg/d]; no seafood should have been taken in last 24 h;

(2) High As in hair or nails [Normal <1 µg/g; >2 µg/g indicates exposure.

4. Differential diagnosis

(1) **2nd stage:** (i) Addison's disease [due to pigmentation] (ii) Syphilis (iii) chronic cocaine poisoning [due to perforation of nasal septum]

(2) **3rd stage** - Alcoholic polyneuritis [APN]- APN shows following distinctive features (i) history of heavy alcohol intake (ii) symptoms take more time to appear (iii) Glycosuria

(3) **4th stage:** Lead poisoning [due to wrist and foot drop].

5. Management [Ch arsenic]

(1) Removal from source (2) BAL (3) Vit B_1 for peripheral neuritis (4) Good diet helps quick general recovery.

6. PM appearances [Ch arsenic]

(1) **General** - jaundice

(2) **Stomach:** (i) ch gastritis (ii) rugae may show patchy inflammatory reddening (iii) Patchy hemorrhagic gastritis (iv) acute and ch erosions
(3) **Small intestines** – dilated, reddened, mucosa thickened
(4) Cloudy swelling and fatty degeneration of heart, liver and kidneys
(5) **Liver** – additionally shows severe necrosis,
(6) **Kidneys** – additionally show tubular necrosis
(7) **Brain** – congested, hemorrhagic spots in white matter
(8) **Peripheral nerves** – fragmentation and resorption of myelin, disintegration of axis cylinders.

F. Tests for Arsenic

(1) **Marsh test** – Hydrogen is passed through suspected material (e.g. vomit). Arsenic if present combines with H_2 to form AsH_3, which is passed through a jet and burnt. A thin film of arsenic over porcelain is formed. Developed by the British chemist **James Marsh** (1794-1846) in **1836**.
(2) **Gutzeit test**
(3) **Reinsch test.** Marsh, Gutzeit and Reinsch tests are now obsolete.
(4) Neutron Activation Analysis [NAA] and Atomic Absorption spectroscopy [AAS]. Performed currently.

G. ML Importance

(1) **Postmortem imbibition of arsenic** – If high arsenic content is found in the exhumed body, the defense may take the plea that the arsenic had leached into the body from surrounding soil. There are two **objections** to this argument (a) **Solubility** - Arsenic is found in an insoluble form in the soil. For it to be able to leach inside the body, it must be in a soluble form (b) **Concentration** – Concentration of arsenic found in the body cannot be higher than that in the soil, if arsenic has leached. This is frequently the case. (i) There is a **counter objection** to the first (a) **Bacterial activity** - Some anaerobic bacteria have been demonstrated in grave soil, which can convert insoluble arsenic into soluble arsenic. Furthermore their metabolic activity causes an exchange of soil arsenic with that of sulphur in the hair.
(2) **Homicidal poison** – Most favorite homicidal poison before 1836, when Marsh test was developed.

(3) **Arsenophagists:**

(i) These are people who take arsenic daily in the mistaken belief that it is an aphrodisiac

(ii) Develop tolerance [**ch 31**]. Can take up to 300 mg or more in one dose, without apparent harm

(iii) Develop sings of chronic toxicity

(iv) D/d of chronic arsenic toxicity

(4) **Safe levels in drinking water:**

(i) 10 parts per billion [ppb] or 10µg/L

(ii) **Hydroarsenicism from contaminated groundwater** - has resulted in large scale environmental poisonings [esp in W. Bengal and Bangla Desh.] for the past 5 decades.

(5) **Blackfoot disease** (BFD):

(i) It is an endemic peripheral vascular disease first seen in southwestern coast of Taiwan.

(ii) Peak incidence was noted between 1956 and 1960.

(iii) Associated with the consumption of inorganic As from the artesian wells.

(iv) The incidence .ed dramatically after the implementation of tap water.

6. **Arsenic in drugs of addiction:**

(i) Cocaine [**ch 41**]

(ii) Opium - generally contaminated with arsenic. Opium is adulterated with arsenic in varying quantities because it is believed to be a general tonic and an aphrodisiac. When combined with opium, it is thought to enhance the aphrodisiac quality of opium. Quite often opium eaters present with neuropathy and hepatomegaly due to ch arsenic poisoning. Arsenic levels are -ed in serum, urine, nails and hair of opium eaters with and without neuropathy.

(iii) Homemade brew – shows - As levels.

(7) -**As levels found in** – Alcoholic cirrhosis, alcohol-related liver disease, fulminant hepatitis, Indian childhood cirrhosis, non-cirrhotic portal fibrosis and Wilson’s disease.

(8) Arsenic is a minor component of cigarette smoke. It is thought that it plays a part in development of cancer.

(9) **Seafood** – is exceptionally rich in As. May contain from **2mg/kg** for freshwater fish up to **22mg/kg** for lobsters. Mussels, oysters, prawns, shellfish

all contain As in varying amounts. Urine of persons who consume large amounts of seafood may contain -As [up to 0.7mg/L; normal levels <0.03mg/L], which may mimic ch As poisoning.

II. BARIUM

A. Mechanism of Action

(1) Barium induces **hypokalemia** by two mechanisms (a) It competitively blocks potassium channels, which are responsible for the efflux of intracellular K+ out of the cell. (b) Directly -s cell membrane permeability to Na^+. This leads to a shift of extracellular potassium into the cell. Intracellular trapping of K^+ leads to

(i) Depolarization and ***paralysis***

(ii) -ed vascular resistance and reduced blood flow " hypertension and lactic acidosis

(2) Direct toxicity to skeletal muscle and neuromuscular transmission. Hypokalemia occurs within 2 hours of exposure. Normal serum K_+ is 3.5-5.5 mEq/L; in barium induced hypokalemia it may be reduced to half or even lower.

B. Signs and Symptoms

(1) **Severe GIT irritation** – (a) Nausea, Abdominal pain, vomiting, diarrhea occur within 1 hour of ingestion. (b) Esophagitis (c) hemorrhagic gastritis

(2) **Signs and symptoms associated with hypokalemia** - (a) ventricular dysrhythmia (b) hypertension (c) profound flaccid muscle weakness (d) respiratory failure (e) lactic acidosis, (f) hypophosphatemia (g) rhabdomyolysis

(3) Secondary to tissue ischemia - (a) Altered level of consciousness (b) seizures (c) basal ganglia manifestations.

C. Fatal Dose

1g.

D. Fatal Period

12 hours.

E. Management

(1) **Gastric lavage** – with Na_2SO_4 or $MgSO_4$ (if seen early). Precipitates barium as insoluble $BaSO_4$.

(2) **IV Na_2SO_4 or $MgSO_4$** – Once advised but no more advisable, because it may lead to renal failure as a result of precipitation of barium in the renal tubules.

(3) **Assisted ventilation** – If patient is in respiratory failure

(4) **Correction of hypokalemia** – Administer large doses of K^+.

F. PM Findings

(1) **Stomach, intestines** – congested.

(2) **Heart** - subendo-cardial petechial hemorrhages.

III. COPPER

A. Toxic Salts of Copper

(1) **Copper acetoarsenite** - please see under arsenic

(2) **Copper arsenite** - please see under arsenic

(3) **Copper subacetate** – Also known as **Verdigris** [Old French vert-de-Grèce, "green of Greece". Called so because used by Greek artists as a green pigment in their paintings].

(4) **Copper sulphate** [blue vitriol].

B. Mechanism of Action

(1) **Effect on SH groups:**

(i) Copper inhibits the sulfhydryl groups on enzymes in important antioxidant systems including G-6-PD and glutathione reductase, reducing their free radical scavenging activities

(ii) **Intravascular hemolysis** is caused by the **inhibition of G-6-PD**.

(2) Copper **-s the permeability of cell membranes** by **inhibiting the NA^+/K^+ ATPase pump**.

(3) Copper intoxication can cause **rhabdomyolysis**, as it **damages human skeletal muscle cells**.

C. Toxicokinetics [Absorption, Distribution and Elimination]

(1) **Absorption** - Copper is absorbed by an active process involving a Cu-ATPase in the small intestinal mucosal cell membrane [also known as the Menkes ATPase]

(2) **If mucosa is damaged** – as in acute overdose, the absorption is significantly higher.

(3) **Carriers of copper** - After absorption, copper is rapidly bound to carriers [albumin, ceruloplasmin, and amino acids eg histidine], for transport to the liver and other tissues.

(4) **Local release of copper** - Copper is released locally [in liver and other tissues] in the reduced form from its carrier

(5) **Fate of copper** - (a) In the hepatocyte, copper may either be incorporated into enzymes or eliminated depending on body's needs. (b) **Cu-ATPase** located on trans-Golgi network assists in its incorporation into enzymes. (c) Same **Cu-ATPase** on pericanalicular lysosomes assists elimination as a metallothionein-copper complex, into the biliary system [final excretion in feces]. Biliary excretion of copper is 2000 µg/24 h. Renal excretion is negligible; about 5-25 µg/24 h

(6) **Release of copper in plasma** - (a) Excess copper is released into the plasma from the liver, bound primarily to ceruloplasmin. (b) 1 Molecule of ceruloplasmin binds 6 atoms of copper. (c) 90-95% of serum copper is bound to ceruloplasmin, and 5-10% to albumin; unbound copper and copper bound to other carriers [amino acids] is <1%. (d) **Half life** - (i) Ceruloplasmin bound copper has a plasma half-life of 24 hours. (ii) Half-life of erythrocyte copper is 26 days. (e) The **albumin-copper complex** represents the "free" or toxicologically active copper. (f) **Following acute poisoning**, the excess copper binds only to albumin.

D. Acute Poisoning

1. Signs and symptoms

Symptoms appear within 15-30 minutes. In **mild cases** there is (i) a metallic

taste in mouth (ii) -salivation (ptyalism) (iii) thirst (iv) nausea, vomiting, eructations and diarrhoea (v) burning pain in the stomach with colicky abdominal pain (vi) Vomited matter is **bluish-green**.

In **severe cases** there may be (i) **Hepatotoxicity** – Acute hepatic necrosis and **jaundice** (ii) **Hemolysis** – More common than hepatotoxicity. Seen invariably in those who have undergone liver damage. Manifested by **myoglobinuria**, **hemoglobinuria** and hemoglobinuric renal failure (uncommon in patients who receive adequate volume replacement therapy) (iii) **hematemesis**, **melena** – Due to ulceration in the GIT (iv) **methemoglobinemia**, **methemoglobinuria** (v) **Renal** and **pulmonary toxicity** – Due to oxidative effects of copper ions. Renal toxicity is manifested by **oliguria** (vi) **Hypotension** and **cardiovascular collapse** – due to (a) intravascular volume depletion from vomiting and diarrhea and (b) direct effect of copper on vascular and cardiac cells (vii) **Neurological** – lethargy, seizures, coma. Result from hepatic encephalopathy. In Wilson's disease, neurological manifestations are prominent.

2. Fatal dose

0.15-0.3 g/kg ($CuSO_4$), i.e. about 20 g or a 70 kg man.

3. Fatal period

1-3 days.

4. Management

(1) **Gastric lavage** – is of limited value, because copper salts themselves are emetics. Emesis generally occurs within minutes of ingestion and is protracted. In the past stomach wash with **1% potassium ferrocyanide** [formation of insoluble cupric ferrocyanide], and **egg white** [formation of copper albuminate] was recommended. No more recommended now.

(2) **Antiemetic therapy** – because of protracted vomiting

(3) **Activated charcoal** - Contraindicated. It may hinder the ability to perform gastrointestinal endoscopy to evaluate corrosive effects.

(4) **Most critical steps** before chelation therapy is started are (a) Supportive care, (b) fluid and electrolyte correction and (c) normalization of vital signs

(5) **Chelation therapy:**

(i) BAL – Administered first. Less effective than d-Penicillamine, yet its initial

use is appropriate. Reasons - vomiting or GIT injury prevents oral d-Penicillamine administration. Furthermore, because the BAL-copper complex primarily undergoes biliary elimination, whereas D-peni-cillamine undergoes renal elimination, BAL proves useful in patients with renal failure. When tolerated, D-penicillamine therapy should be started simultaneously or shortly after the initiation of therapy with BAL.

(ii) Calcium disodium ethylenediaminetetraacetate ($CaNa_2EDTA$) – **ch 32**.

(iii) d-Penicillamine - **ch 32**.

(6) **Hemodialysis** – is ineffective. Copper is nondialysable. May be considered in patients with renal failure.

(7) **Liver transplantation** - in life-threatening hepatic failure.

5. PM appearances

(1) **Mouth and nostrils** – Greenish blue froth

(2) **Gastric mucosa** – Congested, eroded, swollen, inflamed. **Color** - greenish blue. **Contents** – greenish blue [**Fig 36.4**].

(3) **Liver** – Soft, fatty

(4) **Kidneys** – Degenerative changes in proximal tubules

(5) **Skin** – Yellow [due to jaundice]

(6) **Blood** - hemolysis.

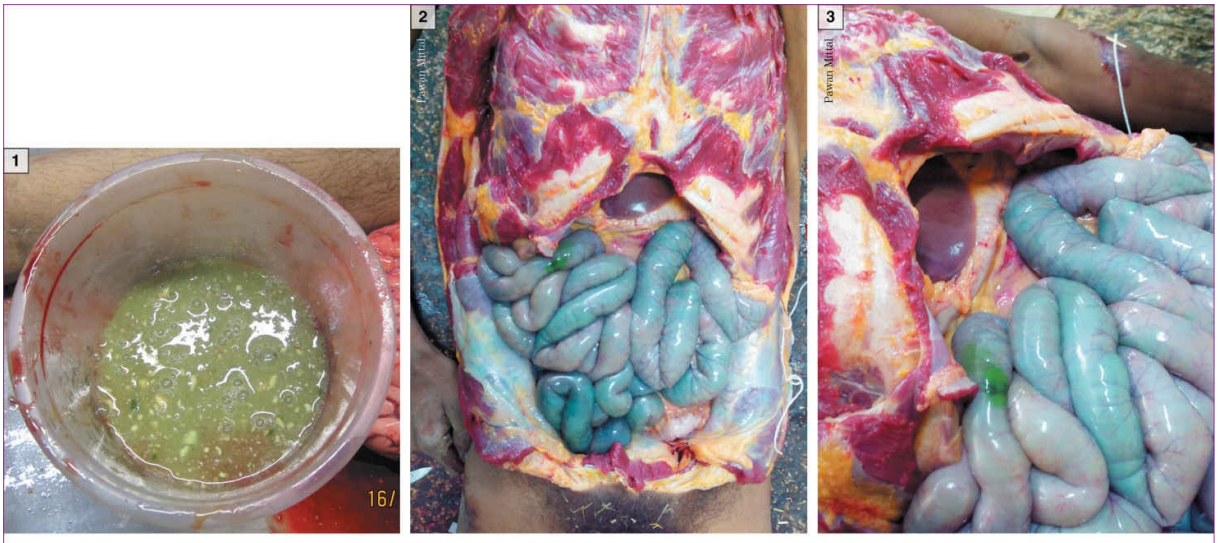


Fig. 36.4: $CuSO_4$ Poisoning **(1)** Bluish green stomach contents **(2)** and **(3)** Bluish green appearance of intestines from outside.

E. Chronic Poisoning

1. Causes

(1) Occurs in workers engaged in **copper factories** (inhalation of copper dust)

(2) **Copper cooking vessels** – If copper cooking vessels are not properly tinned on the inside, food cooked in such vessels will cause formation of copper salts [Acids used in cooking (citric acid, tartaric acid, acetic acid) react with copper and form salts]. Their ingestion over a period of time will cause chronic poisoning.

(3) **Wilson's disease:** Wilson's disease or **hepatolenticular degeneration** is an autosomal recessive genetic disorder affecting approx 1 in 40,000 people. Cause is the presence of abnormal allele of **ATP7B gene** at both loci (located on chromosome 13). A single abnormal copy of the gene is present in 1 in 100 people. They are carriers and do not develop any symptoms. If an abnormal allele is inherited from both parents, Wilson's disease develops. The normal gene codes for ATPase that **transports copper into bile** and incorporates it into **ceruloplasmin**. In the absence of normal gene, **copper accumulates in tissues**. This manifests as neurological or psychiatric symptoms and liver disease. Symptoms usually appear between the ages of 6 and 20 years, but cases in much older patients have been described.

2. Signs and symptoms

(1) **Anemia**

(2) **Green line on gums**

(3) Colic

(4) Peripheral neuritis

(5) Degeneration and atrophy of muscles

(6) **Vineyard's sprayer's lung - Bordeaux mixture** is copper sulfate (1-2%) mixed with lime [$Ca(OH)_2$]. It is sprayed in vineyards to control fungus on grapes, melons, and berries. It is chronically inhaled by vineyard sprayers, who develop **interstitial pulmonary fibrosis** and **histiocytic granulomas** containing copper. Many workers also develop malignancies indicating the carcinogenic effects of copper.

(7) **Chalcosis lentis** - (Gk chalkos, copper) Chronic poisoning may cause deposition of copper in the cornea or lens turning them **greenish-brown**.

(8) **Greenish discoloration of hair:**

(i) Caused due to deposition of Cu over the hair.

(ii) Swimming pools - Copper salts [eg $CuSO_4$] are used in swimming pools as algaecides. Long time swimmers may get **green discoloration of hair**.

(iii) Tap water – Copper leeches from copper fittings by the acidic pH of the water [caused by fluoridation of drinking water]"may turn hair green.

(iv) Exhaust gases containing copper and dusts from the copper processing industry cause green discoloration of the hair [Chalcosis] - [please co-relate with **ch 3**].

F. ML Importance

(1) **Abortifacient**

(2) **Cattle poison**

(3) **Cheshunt compound** - a mixture of copper sulfate and ammonium carbonate used in horticulture to prevent fungal diseases in seeds. May cause poisoning

(4) **Manner of poisoning:**

(i) Accident - (a) Children – may often put the crystals in their mouth attracted by their shining green color (b) Coin ingestion – Indian **`2** and **`5** coins are made of cupro-nickel. Their accidental ingestion may cause poisoning. (c) Cookware - Copper tea kettles and other copper cookware can be a source of copper toxicity if used frequently over a period of time. (d) Vegetables - Copper salts are used to enhance the green color of vegetables"if vegetables not washed properly before eating, can cause poisoning. Food vendors are liable for punishment under The Prevention of Food Adulteration Act, 1954.

(ii) Homicide – Because of their color and taste, salts of copper can not normally be used for homicide. However they can be mixed in blue colored liquids eg sherbets or dark liquids eg Cola drinks

(iii) Suicide – Relatively common, as copper salts are used in many cottage industries

(5) **Medicine** - Copper has been used in medicine for a variety of ailments, which can cause iatrogenic poisoning

(i) Treatment of burns and other wounds

(ii) Contraceptive Pills - -es Cu levels in the body, because of close association between estrogen and Cu levels.

(iii) Copper is a component of dental casting gold alloys, dental amalgams, and IUDs. It corrodes"releases Cu ions into the surrounding tissue"toxicity.

(iv) As an emetic – In the 1960s, copper sulphate (250 mg) was used as an emetic. No more used now because of its toxicity.

(v) Hemodialysis - Copper in dialysis heating coils can be released, especially if acidic water is used in hemodialysis.

(vi) Phosphorus poisoning – in ingestions as well as topical exposures to phosphorous.

(6) **Metal fume fever** [MFF] - Occupational disease recognized for centuries, but first characterized by Potissier in 1822. Caused by **freshly oxidized metallic fumes** (Most commonly with **zinc**, followed by **copper** and iron. Cadmium and tin however produce a more severe illness) having a particle size smaller than 0.5 to 1.0**μ**. Usually caused by welding or melting of metals, soldering, forging and casting (all involve oxidation of metals. **ZnO forms at relatively lower temperatures**, accounting for its frequency as a causative factor). Quite common; more than 1000 cases are reported annually. **Signs and symptoms** [of MFF] - Influenza like syndrome, with sudden onset of chills, fever (104°C), thirst, myalgias and headache within 4-8 hours of inhalation of freshly oxidized metallic fumes. Course is generally benign with symptoms resolving completely within 24-48 hours without any sequelae. **Theories of causation** (a) release of **endogenous pyrogens** (b) production of **antigenic metal proteinates** (c) Delayed hypersensitivity pneumonitis (d) Direct toxic effect on alveolar cells.

IV. LEAD

Lead is a silvery-gray soft metal, used in construction of buildings, bullets, ceramics, electric cable insulations, fusible alloys, hair dyes, lead-acid batteries, pewter, potteries, radiation shieldings, shots, solders, and weights. Lead and its salts are potent poisons.

A. Toxic Salts of Lead

(1) **Lead carbonate** ($PbCO_3$, safeda)

(2) **Lead chromate** ($PbCrO_4$)- has a bright yellow color and is insoluble in

water. As a result used in paints as "**chrome yellow**"

(3) **Lead diacetate** [$Pb(C_2H_3O_2)_2$ - Commonly known simply as **lead acetate**] – Called **sugar of lead** because of its sweet taste. Other names are salt of Saturn, and Goulard's powder (after Thomas Goulard). It was used medicinally in the early 19^{th} century for control of bleeding and diarrhea.

(4) **Lead monoxide** (PbO)- Occurs in two polymorphs, red (**litharge**) and yellow (**massicot**). Used in the manufacture of lead glasses, ceramic glazes and fine dinnerware

(5) **Lead sulfide** (PbS, Galena) – Insoluble and stable at blood pH. Thus least toxic forms of lead

(6) **Lead tetraacetate** [$Pb(C_2H_3O_2)_4$]

(7) **Lead tetroxide** (Pb_3O_4 or $2PbO \cdot PbO_2$)-Also known as red lead, ***sindoor***, **vermilion**. HgS has the same color and has same names. Once both Pb_3O_4 and Hgs were applied by married Indian ladies on their heads. Largely abandoned now because of toxicity

(8) **Tetraethyl lead** [$CH_3CH_2)_4Pb$; TEL] - Once a common **antiknock additive** in petrol. Now largely discontinued because of toxicity. Still used as an additive in aviation fuel for piston engine-powered aircraft.

B. Toxicokinetics

1. Absorption

Absorption by (i) ***oral route*** is less efficient (10%-15% of total absorbed Pb comes via this route) than by (ii) **inhalation** (40-50%). Essential trace elements (Fe, Ca, Zn) **reduce absorption** by competitive absorption. Children have a 3 fold higher lung deposition rates than adults. (iii) **Cutaneous absorption** is poor (iv) **Placenta** – Readily crossed. Causes fetal toxicity when mother is poisoned (v) **Other routes** – Lead bullets or pellets lodged in bones, muscles and joints (souvenir bullets) may get slowly absorbed causing poisoning.

2. Distribution

(1) **Mostly stored in bone:**

(i) Distribution of Pb is beautifully illustrated by the classic 3-compartment model [**ch 31**]. In adults, app 95% of the body Pb is stored in bone (70% in

children). (a) In bones Pb is stored mainly as insoluble phosphate and carbonate. (b) -Ca^{++} levels favor storage; Ca^{++} deficiency causes Pb to be released into blood stream. $CaCl_2$ is thus advocated in treatment.

(ii) Most of the remainder is distributed to the soft tissue pool.

(iii) Very little is present in blood; 99% of that is bound to RBCs

(2) **Toxicity is associated with soft tissue lead uptake** - Most of the toxicity associated with lead is a result of soft-tissue uptake.

(3) **Toxic Lead levels:**

(i) Normal asymptomatic tissue lead levels are 200-500 parts per billion

(ii) Brain lead content in encephalopathy is 1000-2000 ppb.

(iii) Blood lead levels (BLLs) of 100 ppb (>10 µg/dL) are considered toxic. [Normal BLL <10µg/100mL]. Symptoms in adults may begin with BLL 40 µg/dL, but usually begin with levels >50-60 µg/dL.

3. Excretion

(1) **Retention rate** - Adults retain 1-4% and children 33% of ingested lead [because Pb mimics Ca, which is used by growing children]. Rest is excreted.

(2) **Excreted through urine and bile** - Lead is primarily excreted in urine (approx 65%; normal urinary excretion of Pb =80**µ**g/L) and bile (approx 35%). Negligible amount is lost via sweat, hair, and nails.

(3) **Biologic half-lives in various body tissues for lead are**: (a) blood (adults), 25 days; (b) blood (children), 10 months; (c) soft tissues (adults), 40 days; (d) bone (labile, trabecular pool), 90 days; and (e) bone (cortical, stable pool), 10-20 years.

C. Mechanism of Action

(1) **Inhibition of heme synthesis** [**Fig 36.5**]. Heme is synthesized in 9 steps. Enzymes at step 2-3 [porphobilinogen synthase or ALA dehydratase], 6-7 [coproporphyrinogen oxidase] and 8-9 [ferrochelatase] are inhibited by Pb. This causes an overall . in heme synthesis resulting in anemia. Also the substrate catalyzed by the inhibited enzyme shows - [**Fig 36.5**].

(2) **Inhibition of –SH groups** - Pb interferes with the action of –SH groups, which are components of several enzymes, receptors and structural proteins.

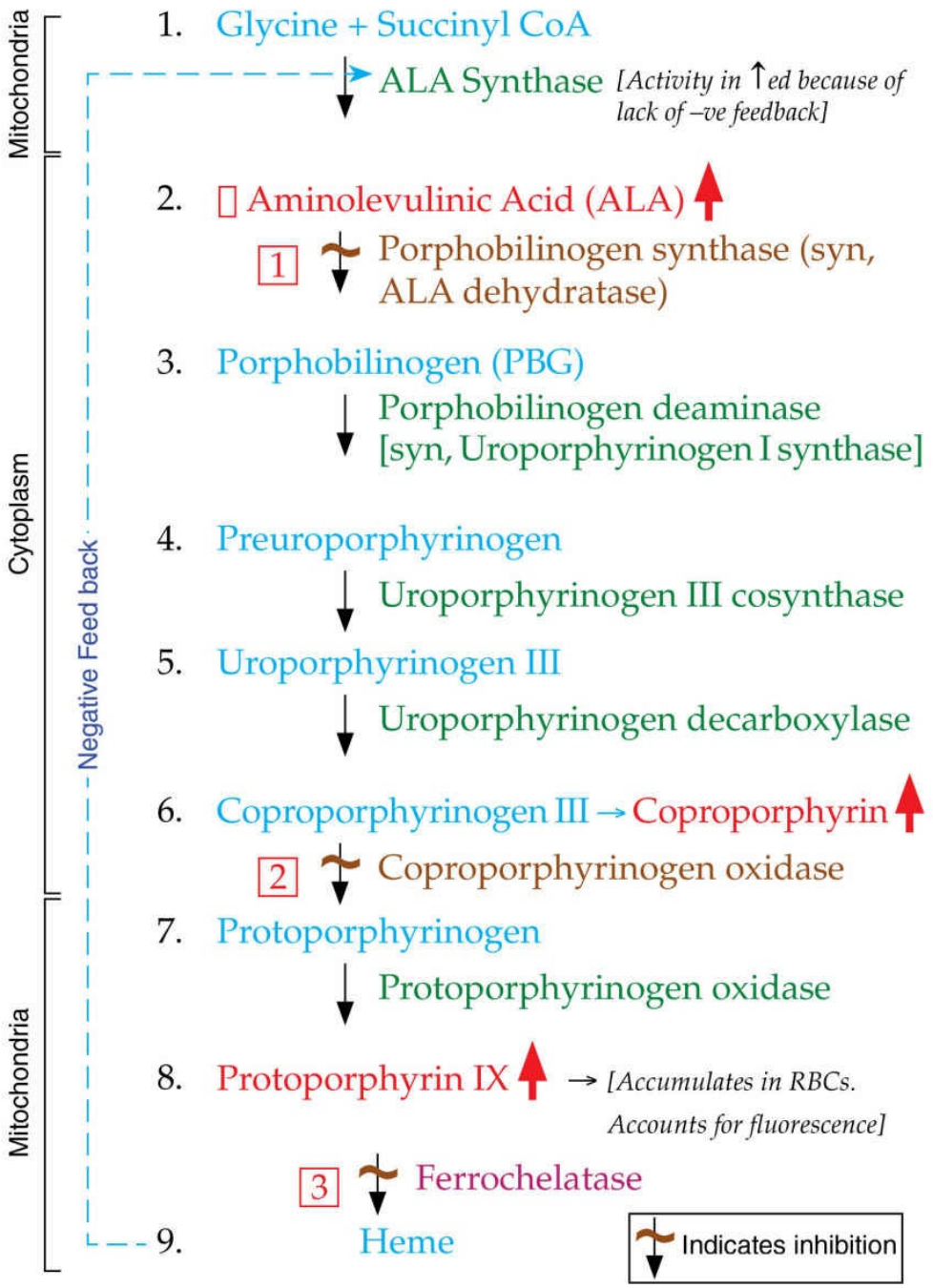


Fig. 36.5: Mechanism of action of lead. Enzymes in brown are inhibited; those in green remain unaffected. Products in red are increased; those in blue are normal. Porphobilinogen deaminase [enzyme at steps 3-4] is used in RNA based assays of blood also (ch. 29)

D. Acute Poisoning

1. Signs and symptoms

Acute poisoning is characterized by (1) an **astringent** and **metallic taste**, (2) headache, (3) dry throat, (4) nausea and vomiting [occasionally diarrhea], (5) thirst, (6) peripheral circulatory collapse [due to loss of water from GIT], (7) neurological signs [(i) **paresthesias** (ii) rarely encephalitis (iii) **cerebellar ataxia** is common in children]. (8) **Hemolysis** causes anemia and presence of hemoglobin in the urine, (9) decreased urine output (damage to kidneys). (10) Death in severe cases.

2. Fatal dose

(1) Lead acetate – 20 g;
(2) Lead carbonate – 40 g;
(3) Tetraethyl lead – 100 mg/kg.

3. Fatal period

1-2 days.

4. Diagnosis

(1) **Lab findings:**
(i) - δ-**A**minolevulinic acid
(ii) - **C**oproporphyrin
(iii) - **P**rotoporphyrin IX [**Fig 36.5**].

Memory Aid 2: Lab findings in Pb Poisoning

ACP – **A**ssistance **C**ommissioner of **P**olice

(iv) **Blood Lead Levels [BLL]** >20**µ**g/100mL. May not be so useful in ch poisoning because Pb migrates to bony compartment]
(v) Urinary excretion of Pb >150µg/L. But if there is renal damage, Pb may not be secreted at all.
(2) **Radiology** – Abdominal X-ray may reveal lead paint chips or other ingested lead foreign material.

5. Management

(1) **Gastric lavage** – with 1% sol^{n} of Na_2SO_4 or $MgSO_4$.
(2) Demulcents
(3) $CaCl_2$ - 5 mg of 10% soln causes Pb to move from blood to bony

compartment, where it lies inert

(4) **Magnesium or sodium sulphate** – 8-12 g. Covert unabsorbed Pb salts to the highly insoluble lead sulphate "hastens fecal excretion

(5) For **convulsions** - (a) Diazepam – 0.1 mg/kg IV [max 10 mg] if patient is convulsing (b) Phenytoin and/or phenobarbital.

(6) **For -Intracranial tension** – Mannitol, steroids

(7) **Chelation therapy:**

(i) If blood lead level (BLL) is >70 µg %, chelation is done with both BAL and EDTA. On the first day of EDTA therapy symptoms may paradoxically worsen [please see above under the heading “distribution”]. To prevent this, administer BAL first. It is a small molecule that crosses into cells, and protects them against the initial - in Pb levels caused by EDTA. Also see **ch 32**

(ii) Penicillamine

(8) Peritoneal and hemodialysis

(9) symptomatic treatment.

6. Cause of death

Gastroenteritis"shock.

7. PM appearances

(1) Signs of acute gastroenteritis – thickened red mucosa with eroded patches

(2) Grayish white deposit on stomach mucosa.

8. Tests

To the suspected material, add hydrochloric acid. White ppt of **lead chloride** will form. It is soluble in boiling water; crystallizes on cooling.

E. Chronic Poisoning

1. Causes

Chronic poisoning **[syn. *colica pictonium*, Devon colic, miner’s disease, painter’s colic, Plumbism (**from Latin plumbum, lead**), or saturnism**] is caused by

(1) **Food and drinking water:**

(i) Drinking water stored in lead cisterns.

(ii) Ghee stored in brass or copper vessels lined inside with lead. Fat in ghee reacts with lead to form lead oleate which is absorbed

(iii) Food cooked in similar vessels

(iv) Tinned food – contaminated with lead from solder

(v) If water supply to homes is through lead pipes or copper pipes with lead-soldered joints.

(2) Use of **hair dyes** and **cosmetics** containing lead

(3) **House dust** from deteriorated lead paint

(4) **Occupational exposure** - Common in people who work with lead, e.g. **p**ainters, **p**ewters, **p**lumbers, **p**otters and **p**rinters. Other professions who may be affected are enamel workers, glass polishers, glassblowers, glass polishers, makers of white lead and smelters. Poisoning caused by continuous inhalation.

(5) **Retained lead ammunition**– In old gunshot wounds, if some bullet or pellets remain lodged in body [souvenir bullet or pellets]

(6) **Sindoor** - Use of sindoor (red lead) applied to scalp. Lead can combine with oils used on scalp to form lead oleate, which can be absorbed.

(7) **Smoking:**

(i) Particularly among workers in Pb based industries.

(ii) Mechanisms - (a) Tobacco plants absorb Pb from soil (b) lead arsenate is used as a pesticide on tobacco crops"gets deposited on leaves (c) General poor personal hygiene habits among smokers [inadvertent contact and ingestion] (d) Inadvertent touching of lips with contaminated hands during smoking.

(iii) Thus lead-handling workers are advised to improve their hygiene by refraining from smoking in the workplace, regularly washing their hands and face, and bathing immediately after their day’s work.

(8) Consumption of **wildlife killed with lead ammunition** - may cause lead intoxication.

(9) **Children** can get poisoned if they continuously chew or lick on leaded objects, eg furniture and walls painted with lead based paints and toys.

2. Signs and symptoms

a. Facial pallor

(1) Facial pallor, particularly circumoral pallor is one of the earliest and most consistent signs

(2) **Causes:** (i) anemia (ii) vasospasm [**Mechanism** – please see above].

b. Anemia

(1) **Initially** – there is polycythemia with polychromatophilia [syn polychromasia – RBCs are of multiple colors, particularly gray-blue. This is due to differing amounts of Hb in each cell, which in turn is due to their premature release from the bone marrow.]

(2) **Other RBC abnormalities:**

(i) Anisocytosis [unequal sized RBCs]

(ii) Nucleated RBCs [sideroblasts]

(iii) Poikilocytosis [abnormally shaped RBCs]

(iv) Punctate basophilia [presence of many dark blue colored pinhead sized spots in the cytoplasm of RBC.]

(v) Reticulocytosis

(3) **Other blood cells:** (i) -Mononuclear cells (ii) .platelets (iii) .polymorphonuclears

(4) **Significant anemia:**

(i) Usually a sign of more chronic poisoning.

(ii) Initially it is microcytic, hypochromic [as seen in Fe deficiency anemia], but later changes to normocytic normochromic.

(iii) Anemia is not a significant feature in organic lead poisoning.

(iv) Causes - (a) .ed Heme synthesis [**Fig 36.5**] (b) .ed survival time of RBCs.

c. Lead line

(1) **Morphology** - A stippled blue line [**Burtonian line** - first described by **Henry Burton** (1799-1849), a British physician] is seen on gums in 50-70% cases.

(2) **Characteristics:**

(i) More common in upper jaw

(ii) Appears within a week of exposure

(iii) Not seen in edentulous persons

(iv) Removal from exposure obliterates line gradually (v) More common in persons with bad orodental hygiene, carious teeth

(3) **Mechanism:**
(i) Subepithelial deposition of black Pb granules
(ii) Bad oral hygiene"food sticking in gums" decomposition " H_2S formed " Reacts with Pb in tissues " forms PbS [black]
(4) **D/d** – similar lines seen in poisoning by bismuth, copper, iron, mercury and silver.

d. Colic and constipation

(1) Also known as **colica Pictonum**, **Devon colic**, or **painter's colic**
(2) **Mechanism** – please see above
(3) Usually appears late
(4) Colic of smooth muscles – occurs in about 85% cases. Blood vessels, intestines, ureters and uterus are involved
(5) Colic occurs at night
(6) Pain is very severe
(7) Individual attacks last a few minutes; keep recurring
(8) Constipation is usual. Diarrhea may also occur.

e. Lead palsy

Weakness of muscles due to lead.

Salient features:

(1) **Sex** – more in men than in women
(2) **age** – more in adults than in children
(3) **Onset** - late
(4) **Frequency** <10% of cases
(5) **Symptoms preceding** – Before actual muscle weakness following symptoms may be seen (i) cramps (ii) Hyperesthesia (iii) Numbness (iv) Tremors
(6) **Muscles affected:** (i) Those which are most prone to fatigue (a) extensor muscles of wrist. [**wrist drop**] (b) Anterior tibial [**foot drop**]. Occurs in ch arsenic poisoning also (ii) biceps (iii) deltoid (iv) rarely (a) eye muscles (b) intrinsic muscles of hand and foot. Associated neuropathy also occurs making the condition worse.
(7) **Recovery** – complete but slow. Occurs when the person is removed from source.

f. Encephalopathy

(1) More common in children

(2) usually irreversible; 85% have permanent brain damage

(3) commonly associated with tetraethyl lead

(4) **Symptoms:** (i) Delirium (ii) Hallucinations (iii) Headache (iv) Insomnia (v) Irritability (vi) Restlessness (vii) Visual disturbances (viii) Vomiting (ix) Convulsions, coma (x) death occurs in about 25% cases.

g. Cardiorenal manifestations

(1) **Hypertension:**

(i) **Mechanism** – (a) arteriolar contraction [please see above] (b) nephritis

(ii) HT ultimately causes permanent arteriolar degeneration.

(2) **Nephritis:**

(i) Chronic arteriosclerotic nephritis

(ii) Interstitial nephritis

(3) **Saturnine gout** [syn, lead gout]- Pb .es renal excretion of uric acid "-Uric acid in blood [hyperuricemia]"Gout.

h. Reproductive system

(1) Sterility and impotence [in both sexes].

(2) Menstrual disorders [Amenorrhea, Dysmenorrhea, Menorrhagia]

(3) **In pregnant females:**

(i) Abortion – generally between 3-6 months [**Mechanism** – please see above]

(ii) Premature labor

(iii) Intrauterine growth retardation.

(4) Occupational exposure of a father just before conception -es the risk of cancer in his offspring through toxic inheritance. [Mech: Pb exposure" genetic change in sperm"cancer in offspring].

i. Eye

Ophthalmoscopic examination reveals retinal stippling. Glistening grayish lead particles are seen.

j. Other systems

(1) Anorexia (2) Drowsiness (3) Dyspepsia (4) Emaciation (5) Exhaustion (6)

Foul breath (7) General weakness (8) Headache (9) Irritability (10) Loss of hair (11) Peripheral neuritis [rare] (12) Vertigo.

Table 2: (with **Memory Aid 3**) A simple mnemonic to remember main features of chronic lead poisoning

ABCDEFGHI:
Anemia, **a**bortion, **a**norexia, **a**rthralgia, **a**trophy of optic nerve
Basophilic stripping, **B**urtonian line
Colicky pain, **c**erebral edema, **c**onstipation, ↑coproporphyrin in urine
Drop [wrist and foot], **D**ry belly ache [**D**iarrhea is very rare]
Encephalopathy, Emaciation, **E**osinophilia
Facial pallor [earliest sign], **F**oot drop [also wrist drop], foul breath, **f**ailure of kidneys, **F**anconi syndrome
Gum (lead line), **G**onadal dysfunction, **G**out
Heart Manifestations, **h**allucinations, **h**eadache, **h**yperesthesia, **h**ypertension
Impotence, **I**nfertility, **I**nsomnia, **i**rritability

3. Diagnosis

(1) History
(2) Clinical features
(3) **Laboratory tests** – As in acute poisoning. Additional tests useful in ch poisoning (a) Normal Pb levels in human seminal plasma are **31 µg/dL**. In chronic poisoning, the levels may be more than double.
(4) **Blood picture** - (a) Anemia [as above] (b) Leukocytosis
(5) **Erythrocyte protoporphyrin** [EP]:
(i) Lead impairs heme-based enzymes
(ii) EP reflects lead inhibition of the heme synthesis pathway.
(iii) It was used as a screening tool in the past, but is no longer considered

sufficiently sensitive.

(iv) Still useful as secondary diagnostic tools for (a) tracking response to therapy (b) distinguishing acute from chronic Pb poisoning [EP levels normal in acute Pb].

(v) EP>35**µ**g/100mL "Chronic Pb Poisoning.

(6) **Free erythrocyte porphyrin (FEP)**: >70 µmol/mol heme.

(7) **Radiology** – Dense transverse bands or lead lines just underneath the metaphyseal plates. They indicate -Pb deposition. Usually develop in 4-8 weeks of heavy exposure.

(8) **X-ray fluorescence** (XRF)- Estimates bone lead, and is thus indirectly a measure of body lead exposure.

4. Prophylaxis

(1) **Factory management should ensure:**

(i) Maintenance of proper ventilation in factories

(ii) Maintenance of personal hygiene of workers

(iii) Periodic medical examination

(iv) Shifting of poisoned worker to administrative side

(2) **Diet** - rich in calcium

(3) **Other measures:**

(i) Small amount of sulphuric acid in water

(ii) Weekly saline purgative.

5. Management

(1) **Removal for source** – Most important. If poisoning is a result of occupational exposure, person should be shifted to another job.

(2) **Chelation:**

(i) **Both BAL and $CaNa_2EDTA$** – If there is lead encephalopathy, BAL is administered first. 4 hours later $CaNa_2EDTA$ is started [1500 $mg/m^2/d$ by continuous IV infusion]. In patients with renal dysfunction limit daily dose to 50 mg/kg.

(ii) **Duration** - Continued for 5 days, followed by a rest period of at least 2-4 days, which permits lead redistribution [please see **3-compartment model** above].

(iii) **Adjuncts** - (a) Folic acid, (b) pyridoxine, and (c) thiamine [10-50 mg/kg] -

the antidotal properties of $CaNa_2EDTA$. Thiamine also improves neurological manifestations

(iv) **Penicillamine – ch 32**.

(v) **Only BAL** – is the chelator of choice in presence of renal impairment as its main route of excretion is bile. Two molecules of BAL combine with 1 atom of Pb to form an insoluble complex" excreted in bile

(vi) **BAL Analogues** – DMSA [**Succimer**], DMPS [**ch 32**]. **Succimer** is increasingly replacing $CaNa_2EDTA$ as the chelator of choice in lead-poisoned children without encephalopathy.

(3) **Calcium gluconate IV** – for colic.

(4) **Magnesium or sodium sulphate –** as in acute poisoning.

(5) **To mobilize Pb from bones:**

(i) Diet – Poor in calcium.

(ii) Ammonium chloride – 1 g ten times daily

(iii) Parathormone

(6) **Surgery** – Rarely surgery may have to be performed.

(7) **Symptomatic**.

6. Cause of death

(1) Encephalopathy

(2) Failure of vital functions (i) Liver failure (ii) Renal failure (iii) Respiratory failure

(3) Intercurrent infection

(4) Malnutrition.

7. PM appearances

(1) **Gums** – Burtonian line

(2) **CNS:**

(i) Brain – (a) pale (b) greatly swollen (c) PAS +ve, pink staining, homogenous material seen in the perivascular spaces

(ii) Peripheral nerves – segmental demyelination

(3) **CVS:**

(i) Heart – hypertrophied

(ii) Aorta, aortic valves - atheromatous

(4) **Stomach and intestines:**

(i) Hemorrhagic and ulcerative changes
(ii) Contracted, thickened
(5) **Liver:**
(i) Contracted
(ii) Hepatocytes – Show eosinophilic intranuclear inclusions [EII]
(6) **Kidneys:**
(i) Contracted
(ii) Proximal tubules - Show EII.
(7) **Muscles** – fatty degeneration [paralyzed during life]
(8) **Bone marrow:**
(i) Hyperplasic of erythroblasts and leukoblasts
(ii) . in fat cells.

8. ML importance

(1) **Abortion** – Lead salts used for abortion, eg **Diachylon** [lead oleate] (pl also see **ch 26**)
(2) **Artificial insemination** - Donors with Pb poisoning [↑ seminal plasma Pb levels] have lower fertility rates than donors with no Pb poisoning or low seminal plasma Pb levels.
(3) **Cattle poison** – Red lead alone or mixed with arsenic
(4) **Drug abusers** - Chronic Pb poisoning may sometimes be seen in drug abusers [because of intentional or unintentional adulteration with lead]
(i) Cocaine – Lead is a contaminant of manufacture in cocaine paste [**ch 40, 41**]
(ii) Marijuana - Lead in marijuana samples is a diluent [**ch 40,41**].
(5) **Employer's liability to pay compensation in cases of occupational disease due to Pb** – Same as MLI of Arsenic above.
(6) **Lead pellets or bullets** -
(i) Ingestion – Suicide has been attempted by ingesting lead pellets [pl see management above]
(ii) Souvenir bullets - These are pellets or bullets which remain in the body after a shooting incident [**ch 13**]. May cause ch lead poisoning.
(7) **Manner** - Suicidal, and homicidal poisoning rare
(8) **Rapidity of poisoning** -
(i) Acute poisoning – rare
(ii) Chronic poisoning is an occupational disease.

V. MERCURY (QUICKSILVER)

A. General

Mercury is a liquid metal, having bright silvery appearance. It is volatile at room temperature.

B. Toxic Salts of Mercury

Mercury occurs in three forms – Metallic mercury, inorganic salt and organic salts. **Metallic mercury** if swallowed as such is not poisonous, as it is **not absorbed from GIT**. It is however absorbed in vapor form, and is very toxic, because inhaled Hg vapors readily cross biological membranes.

1. Organic salts

Organic salts are most poisonous, especially (1) **methyl mercury** and (2) **dimethyl mercury**.

2. Inorganic salts

Among inorganic salts, **mercuric salts** (Hg^{++}) are more poisonous than **mercurous salts**(Hg^{+}). **Mercurous** salts are **less soluble** in water than mercuric salts, accounting for their lesser toxicity.

(1) **Mercuric Chloride** [$HgCl_2$, **Corrosive sublimate**] – White crystalline powder, soluble in water, but much more so in alcohol. Before the advent of antibiotics, used to treat syphilis. Once available as a blue coffin-shaped tablet with the inscription "POISON". This 0.5 g tablet dissolved in water was intended to be used as an antiseptic solution.

(2) **Mercurous Chloride** [Hg_2Cl_2, **Calomel** (Gk. kalos, fair; melas, black; its original preparation involved converting a black powder into white), sweet mercury] - A white, tasteless powder, used chiefly as a fungicide and abortifacient [**ch 26**]. Less toxic than $HgCl_2$ due to its lower solubility in water.

(3) **Mercuric ammonium chloride** - ointment containing 10% $HgNH_2Cl$ is used for eczema. May cause toxicity.

(4) **Mercuric cyanide** – [$Hg(CN)_2$]- Odorless, toxic white powder with a bitter

metallic taste. Once used as an antiseptic. As an antisyphilitic, its solution was applied with a brush on syphilitic sores. Still used in homeopathy as Hydrargyrum bicyanatum

(5) **Mercuric iodide** - (HgI_2, **Protiodide**) Red-orange crystals. Once used as a treatment for syphilis

(6) **Mercuric nitrite**

(7) **Mercuric oxide** (HgO)- Brick red powder. Used as cathode for mercury batteries

(8) **Mercuric sulfate** ($HgSO_4$)- odorless white crystalline powder

(9) **Mercury sulphide** [**cinnabar**, red sulphide of mercury, ***sindoor***, vermilion, HgS. Vermilion is primarily HgS, but broadly it refers to any of various red pigments.]

(10) **Mercury thiocyanate** [$Hg(SCN)_2$] - Best known for its former use in Deewali, as it produces a large, winding "snake" when set on fire. This is known as the Pharaoh's Serpent [After a Biblical story when Pharaoh of Egypt calls his magicians, who turned their staffs into snakes].

C. Mechanism of Action

(1) **Mercury binding** to (i) **sulfhydryl** (ii) **phosphoryl** (iii) **carboxyl** and (iv) **amide** groups - results in widespread dysfunction of enzymes, transport mechanisms, membranes and structural proteins

(2) **Direct oxidative effect of mercuric ions** - results in PCT necrosis. Mercury ion accumulates predominantly in the renal cortex

(3) **Immune mechanism** – results in membranous glomerulonephritis and acrodynia associated with Hg

(4) **Methylmercury inhibits astrocyte uptake of cysteine**"Results in .production of glutathione (a major antioxidant).

D. Toxicokinetics [Absorption, Distribution and Elimination]

(1) **Absorption:**

(i) **Elemental Hg** – (a) GIT [Not absorbed]" nonpoisonous orally. (b) inhalation [80% absorption]. (c) Cellular membranes - diffuses rapidly including the blood-brain barrier and placenta. (d) Half life - 60 days.

(ii) **Inorganic Hg** – GIT [10%], skin and mucus membranes.

(iii) **Organic Hg – Long chain alkyl compounds**"GIT [>50%]; **short chain alkyl compounds**"GIT [90%]

(2) **Distribution –** Mercury distributes widely to all tissues. Major deposition in **CNS**, **kidneys**, liver and spleen. Methylmercury concentrates in **hair** (due to presence of sulfhydryl groups). Its concentration in hair is 250 times more than in whole blood. Within blood, methylmercury **concentrates more in RBC** than in plasma. **RBC:Plasma** ratio of methylmercury is **10:1**, while the same ratio for **inorganic Hg** is **1:1**.

(3) **Elimination –** The total body half life of elemental and **inorganic Hg** is **30-60 days**. In contrast half life of **organic Hg** is **70 days** (due to its enterohepatic recirculation). Inorganic mercury is excreted both through urine and feces. Excretion of organic Hg is predominantly fecal.

E. Acute Poisoning

Signs and symptoms differ according to the type of Hg ingested [elemental, inorganic or organic].

1. Signs and symptoms

a. Elemental mercury

i. Inhalation

Usually occurs following gold refining in a closed room.

(1) **General** – Headache, blurring of vision, Fever, chills [metal fume fever], Erythematous pruritic papular rash, conjunctivitis, salivation

(2) **Pulmonary** – chest pain, cough, dyspnea, interstitial pneumonitis,, necrotizing bronchiolitis, pulmonary edema, pulmonary failure,

(3) **GIT** – Metallic taste, deep red oral mucosa, gingivitis, stomatitis, strawberry tongue, swelling of salivary glands, teeth [loosened]

(4) **Miscellaneous** – Reddened palms and soles, manifestations similar to Kawasaki disease (mucocutaneous lymph node syndrome) especially in children. May be mistaken for scarlet fever.

ii. Ingestion

Absorption is minimal [<0.01% is absorbed]. Large quantities can be ingested

without obvious toxic manifestations. In **1515**, an English nobleman on his honeymoon drank a large amount of elemental Hg by mistake, but did not suffer any harm.

iii. Injection

(1) **S/c or IM** - Abscess formation, ulceration [exuding tiny droplets of Hg].

(2) **IV** – [generally suicidal, or taken with a false belief that it acts as an aphrodisiac] bloody sputum, dry cough, granuloma, pulmonary embolism, tachypnea, thrombophlebitis.

(3) **Intra-arterial** – Can occur inadvertently from arterial blood gas sampling with syringes containing Hg as anaerobic seal. Peripheral embolization with ischemia and gangrene. X-ray in all cases reveals Hg globules.

b. Inorganic mercury

Manifestations occur in 2 phases.

i. First phase

(1) **Immediately after ingestion of corrosive mercury salts** – Hot burning pain, sense of constriction, ashen discoloration of the mucous membrane in mouth and throat. Burning pain extends down to stomach and abdomen.

(2) **Within a few minutes** - Intense epigastric pain, followed by diffuse abdominal pain. Nausea, retching and almost continuous vomiting of mucoid material, which frequently contains blood and shreds of mucous membrane. Hoarse voice.

(3) **Mouth, tongue and fauces** – are corroded, swollen and show a grayish white coating.

(4) Acrid metallic taste, excessive salivation and thirst.

(5) Severe purging, with liquid, bloody feces and considerable tenesmus.

(6) Rapid, weak pulse; breathing shallow and difficult; Pallor; Prostration, circulatory collapse, and death.

(7) Above Signs and symptoms are not seen with mercury compounds of low irritancy or with portals of entry other than the mouth.

ii. Second phase

If death does not intervene, **phase 2** begins in **1-3 days** in untreated cases (unless

vomiting effectively removed most or all poison).

(1) **Oral** - Marked salivation, mercurial stomatitis, glossitis and ulcerative gingivitis within 24-36 hr. Loosening of teeth, necrosis of the jaw.

(2) Severe infections

(3) **Renal** - PCT Necrosis in 2-3 days, transient polyuria, albuminuria, cylindruria, hematuria, anuria, azotemia and renal acidosis. Recovery may occur within 10-14 days, but death may also occur in some cases.

(4) **Late** - After many days after the original exposure (especially in untreated cases), a membranous colitis may appear; dysentery, tenesmus, ulceration of the colonic mucosa, and hemorrhage. Liver necrosis sometimes develops.

2. Fatal dose

1-4 g of $HgCl_2$

3. Fatal period

3-5 days.

4. Management (acute Hg)

a. For inorganic mercury

(1) **Removal of patient** – from further exposure

(2) **Gastric lavage** – Avoid. It is **hazardous** (due to corrosive nature of $HgCl_2$, it can lead to perforations) and **unnecessary** (excessive vomiting already)

(3) **Activated charcoal** [AC]

(4) **Fluid resuscitation** – Done on a priority basis. Otherwise there is danger of cardiovascular collapse is caused by severe gastroenteritis and fluid loss.

(5) **Hemodialysis** and **peritoneal dialysis** – **Not useful** because of high degree of protein binding

(6) **Whole bowel irrigation** – with polyethylene glycol is useful. Progress should be followed with serial radiographs.

(7) **Chelation therapy:**

(i) **BAL** and **DMSA**- 5 mg/kg every 4 hours deep IM for 48 hours; then 2.5 mg/kg every 6 hours for 48 hours; then 2.5 mg/kg every 12 hours for 7 days. When the patient is able to take oral medication, replace BAL with DMSA (Succimer) at 10 mg/kg orally tds x 5 days; then twice a day x 14

days. This regimen is the ***treatment of choice*** for inorganic Hg poisoning

(ii) **D-penicillamine (Cuprimine)** – 1-1.5 g/day orally in 4 divided doses (alternative chelator)

(iii) **N-acetyl-DL-penicillamine** (**NAP**)- An investi-gational analog of D-penicillamine is a better chelator of Hg than D-penicillamine, because of its greater stability

(iv) **DMPS –** 5 mg/kg IV, followed by 100 mg orally bd for 3 weeks.

b. For organic mercury

(1) **BAL** – **should not be used**. BAL comes as a lipid solution and may - Hg mobilization into the brain, causing severe toxicity. Instead

(2) **DMSA** (**Succimer**) and

(3) **DMPS** are used. Both are water soluble analogs of BAL (ch 32).

c. For elemental mercury

(1) **Postural drainage and endotracheal suction** – useful if mercury vapors have been inhaled.

(2) **Surgical excision** – if there has been IM Hg deposition

(3) **Environmental decontamination** – If there is spillage of elemental Hg on solid surfaces, following measures are useful (a) **Adsorb on sand** - and discard in tightly sealed containers (b) **Mercury decontamination kit** – Should be used. Contains **calcium polysulfide**, which converts Hg to HgS (Cinnabar) (c) **Remove absorbent surfaces** – eg. carpets (d) **Do not vacuum Hg** – can volatilize it.

5. PM appearances

(1) **Lips, mouth, tongue and esophagus** - Mucous membrane more or less whitened, swollen and soft.

(2) **Stomach and esophagus:** (i) congestion (ii) corrosion (iii) inflammation (iv) severe hemorrhagic necrosis

(3) **Large intestines** – Mucus membrane inflamed, swollen, corroded and hemorrhagic [lesions seen if person survived for a few days. Due to re-excretion of Hg in the large intestines] – Necrotic areas. Small intestine is also affected but to a lesser degree.

(4) **Heart** – subendocardial petechial hemorrhages

(5) **Lungs** – Thrombosis in capillaries.
(6) **Liver** – congested. Shows cloudy swelling and fatty change.
(7) **Kidney** – Interstitial nephritis
(8) **H/P:**
(i) Severe proximal convoluted tubule (PCT) necrosis, hemorrhagic glomerular nephritis
(ii) Mercury deposition in renal cortex and renal macrophages
(iii) Neuronal necrosis and glial proliferation in cerebral cortex [seen in Minamata disease].

F. Chronic Poisoning (Hydrargyrism)

Clinical pictures are different in inorganic and organic mercury toxicity. Inorganic mercuric chloride mainly affects the renal and gastrointestinal systems. The characteristic neurological feature is a fine tremor, particularly of the hands and fingers.

1. Signs and symptoms

May result from (1) External application of Hg as **ointment** (2) **occupational** exposure (3) **recovery** from a large dose (4) excessive **therapeutic** use of Hg.

a. General

(1) Anemia (2) anorexia (3) loss of weight (4) Inflammation of gums (5) **blue line** at the junction of teeth and gums (6) Sore mouth and throat (7) loosening of teeth (8) GIT disturbances (9) chronic nephritis with progressive uremia.

b. Ptyalism

Common causes of **ptyalism**.

c. Mercurial tremors

Mercurial tremors (syn. ***Danbury tremors*** or shakes, ***Glassblowers tremors*** or shakes, ***Hatter's shakes***) are static and intentional tremors seen in chronic mercury poisoning. The first description of mercurialism in hatters was published by **J. Addision Freeman**, in Transactions of the Medical Society of New Jersey in 1860.

Salient features:

(1) **Tremors** – Two types are seen (i) **Static or resting tremor** - occurs when the muscle is at rest. It is a fine trembling motion, most evident in the upper extremities (ii) **Intentional or ataxic tremor** – (a) occurs when there is purposeful movement of an extremity, or (b) as an aggravation of an established static tremor. **Concussion mercurialis** (**Tetanus mercurialis**) refers to the last stage where tremors are so intense that they prevent daily activities.

(2) **Predisposing factors** – Alcohol predisposes to tremors. Severe shakes are never seen in teetotalers. **Alternative names:** (1) **Hatter's shakes**

(3) **Glassblower's shakes** - Glassblowers often blow colored mercury compounds in ornamental glass. May get poisoned.

d. Mercurial erethism

Mercurial erethism (or erythism) is the name given to neuropsychiatric manifestations seen in chronic mercury poisoning. The classical triad of chronic mercury poisoning is (i) gingivostomatitis (ii) mercurial tremors and (iii) mercurial erethism. Main symptoms seen in erethism are – Anorexia, emotional instability, fatigue, insomnia, loss of memory, mood changes [anxiety, shyness, withdrawal, depression, loss of confidence, nervousness, irritability, timidity], resentment at being observed, rough temper, delusions, hallucinations, suicidal melancholia, manic depressive psychosis (**mad hatter**). The Mad Hatter is a character in Alice's Adventures in Wonderland, a novel written in 1865 by Lewis Carroll. He showed signs of mercurial erethism.

e. Mercuria lentis

Mercurial lentis is a peculiar eye change seen in chronic mercury poisoning due to deposition of mercury through the cornea on to the anterior lens capsule.

Salient features:

(1) **Slit lamp examination** demonstrates a matt-brown reflex from anterior lens capsule

(2) It is bilateral and has no effect on visual acuity. In contrast methyl mercury toxicity results in **visual blurring** and **tunnel vision**.

(3) Mercury pigmentation of the anterior lens capsule may occur secondary to topical medication containing mercury too [Mercuria Lentis Medicamentosus]. These are mainly miotics containing phenyl-mercuric

nitrate (PMN).

(4) **Other eye changes:**

(i) Lens opacities

(ii) Well marked vascularity at the corneoscleral junction.

f. Acrodynia (pink disease)

Acrodynia (also known as calomel disease, erythredema, erythredemic polyneuropathy, Feer's disease, pink disease, Selter's disease and Swift's disease) is a peculiar manifestation of mercury poisoning seen in children.

Salient features:

(1) Hands and feet of the child became painful, paresthetic, peeling, [**Fig 36.6**] perspiring, pink and puffy (Gk acro, extremity; dynia, pain).

(2) **Other symptoms** – Anorexia, hypertension, hypotonia, insomnia, irritability, photophobia, rashes [due to excessive perspiration].

(3) Symptoms of **catecholamine excess** (perspiration, tachycardia, hypertension) occur because mercury blocks the degradation pathway of catecholamines.

(4) **D/d** includes pheochromocytoma and Kawasaki disease.

(5) Acrodynia was relatively common amongst children in the first half of the 20^{th} century, due to the use of **calomel** in **teething powders** (analgesic action) and for **washing nappies** (bactericidal action).

(6) There is lack of correlation of symptoms with mercury levels, so it is thought to be an **idiosyncratic hypersensitivity reaction** to mercury.

(7) Mortality is about 10%.

Fig. 36.6: Acrodynia from mercury poisoning.

Memory Aid 4: Symptoms of ch Hg poisoning

Mnemonic for chronic Hg poisoning"**TABLE** – **T**remor, **A**crodynia, **B**lue-Black line on gums, **L**entis, **E**rethism

g. Hunter russell syndrome

Occurs due to ch exposure to **methyl mercury**. Most disastrous epidemic occurred in 1953 [Minamata disease]. **Symptoms:**

(1) **CNS – Peripheral neuropathy** is a major feature. Major symptoms are akinesia, ataxia, auditory impairment, blurred vision, cerebellar ataxia, constricted vision, dementia, depression, diminished or absent deep tendon reflexes, but up to 40% may have enhanced tendon reflexes, dysarthria, impaired hearing, smell and taste, malaise, memory loss, numbness, olfactory impairment, paresthesia with a peroral and glove and stocking distribution, rigidity, sensory impairments, spasticity, taste impairment, tremors, **visual blurring** and **tunnel vision**, and weakness.

(2) The **immature brain** is especially susceptible to the effects of methylmercury and is often damaged in an irreversible and profound manner.

(3) **Congenital poisoning** - mental retardation, deafness, blindness, dysphasia,

ataxia, microcephaly, and cerebral palsy. However, signs of peripheral neuropathy are not found.

(4) **Pathology** - The symptoms are due to loss of myelinated nerve fibers, autonomic dysfunction, sensory nerve conduction delay, abnormal neuronal migration, and abnormal central nervous system cell division.

2. Diagnosis

(1) **Normal levels of Hg:** (i) Blood - <10 µg/L (ii) Urine - <20 µg/L. (iii) Normal urinary Hg excretion in 24 h is <50 µg.

(2) **Ch exposure** - (i) Blood >35 µg/L (ii) Hair. (iii) Urine >150 µg/L

(3) **MRI** [in patients with ingestions of organic Hg]- **Atrophy** of **cerebellar hemispheres**, postcentral gyri and calcarine area. These findings co-relate with clinical findings of ataxia, sensory neuropathy and visual field constriction respectively.

3. Management (Chronic Hg)

(1) Removal of patient from further exposure

(2) Demulcents

(3) Saline purgatives

(4) Maintain oral hygiene

(5) Chelation therapy – same as in acute poisoning.

G. Tests for Mercury

Suspected solution + A piece of copper wire + Few drops of HCl"A silvery colored coating of mercury on copper wire will form.

H. MLI

(1) **Blood** - Stains of red sulphide of mercury resemble blood stains. They need to be differentiated [**ch 29**].

(2) **Blood transfusions** - Blood conc of Hg may be higher in persons taking a diet rich in sea food. If blood from such individuals is transfused to premature infants [especially multiple transfusions in a short period], there is a possibility of Hg poisoning.

(3) **Component of:**

(i) $HgCl_2$ is a component of cavity fluid for embalming [**ch 9**]

(ii) Hg_2Cl_2 is a component of Vibert's fluid [**ch 29**]

(4) **Criminal abortion** – Mercury salts [eg $HgCl_2$] introduced in vagina for criminal abortion or as contraceptive measures. May cause death.

(5) **Dentistry** – Hg used in dental amalgams.

(i) Patients - Chronic absorption produces grayish blue mucosal discoloration.

(ii) Dentists – Many still knead the amalgam mass in their hands. While squeezing the mass to express out excess Hg, droplets often fall on the floor. Since they are difficult to scoop up, they are allowed to remain their and vaporize causing the Hg conc in the environment to rise. This may cause chronic Hg poisoning in dentists.

(6) **Employer's liability to pay compensation in cases of occupational disease due to Mercury** – Same as MLI of Arsenic above.

(7) **Manner of poisoning:**

(i) Accident– can occur as a result of (a) thermometers breaking in the mouth. It is usually harmless. (b) as a complication of Hg-sealed syringes for arterial blood drawing. It can give rise to **radiopaque shadows** in the head (embolization in brain), chest (embolization in lungs) and other areas of the body. D/d of such shadows are (I) embolization of bullet fragments or lead shots, (II) heavy metal and metal salt deposits (bismuth) in gluteal areas and (III) cysticercosis (c) Disk battery ingestions – Mercury oxide may release if battery opens within GIT causing Hg poisoning.

(ii) Homicide and suicide – rare.

(8) **Quacks** – Mercury salts are quacks' favorites. Acute Hg poisoning may result from the external application of some mercurial preparation, or from the injection into the uterus, or into large abscess cavities of a sol^n of corrosive sublimate. Symptoms of acute Hg poisoning, followed in some cases by death, have resulted from the external application of corrosive sublimate to tumors or ulcers, a method sometimes resorted to by quacks for the destruction of so-called cancer; similar toxic symptoms have resulted from injecting a solution of corrosive sublimate into psoas abscess cavities, and into the uterus after childbirth.

(9) **Therapeutic use of Hg:**

(i) **Mercuric chloride** – Strong sol^n used for washing abscess cavities or irrigating vagina, uterus or rectum. May cause accidental poisoning.

(ii) **Mercurous chloride** – (a) Can cause acrodynia [please see above]. Even today it is sometimes used as a laxative. (b) As an abortifacient, it caused death of pregnant mothers [**ch 26**]

(iii) **Metallic mercury** is generally not poisonous. At one time oral Hg in doses of 100 to 500 g was given for the treatment of **adynamic ileus** and **bowel obstruction**. It usually passed off in the feces harmlessly, but sometimes diarrhea and stomatitis resulted. Occasionally **fatal poisoning** also occurred. Hg was also put as a weight at the end of a gastroduodenal tubes, which were passed to relive GIT obstructions. Sometimes the Hg containing balloons used to rupture, releasing Hg in the GIT. Generally such events were harmless, but occasionally acute appendicitis used to occur. Sometimes Hg used to escape from a perforated appendix and formed **intraperitoneal mercury granulomas**.'

(10) **Thiomersal controversy** – Thiomersal, an **organomercury** compound, is a well established **antiseptic** and **antifungal** agent, and has been used as a preservative in vaccines, immunoglobulin preparations, skin test antigens, antivenins, ophthalmic and nasal products, and tattoo inks. Its use in vaccines **allows the use of multidose vials** instead of single-dose vials, which are more expensive. There have been concerns that its use in vaccines leads to the ***development of autism*** and other ***brain development disorders in children.***

VI. THALLIUM

In the early 1900s, **thallium** [Tl] salts were used as a depilatory and to treat syphilis, gonorrhea, TB and ringworm of the scalp. The dose given (7-8 mg/kg) was very near fatal dose, and caused severe poisoning (**thallotoxicosis**). Because **thallium sulfate** is odorless and tasteless, it was very useful as a rodenticide (Thalgrain, Echol's Roach powder, Martin's Rat Stop liquid). Since it could easily be purchased as a rodenticide, it became a popular homicidal poison too. **Thallium acetate** was once used to check the "night sweats" in tuberculosis. When it was noticed that it caused loss of hair, Sabouraud instituted its therapeutic use for tinea in 1898. It was supplied as a cream (Koremlou cream), containing 7.18% thallium acetate. This practice was abandoned half a century later, when it was realized that it was a potential poison. Thallium is used in electronics industry [App 60–70% of Tl production], pharmaceuticals, in glass

manufacturing and in infrared detectors.

A. Acute Poisoning

Can occur from

(1) Oral ingestion [most common]

(2) Inhalation from contaminated dust from pyrite burners, cadmium manufacturing, lead and zinc smelting, and

(3) As a contaminant of heroin or cocaine. Symptoms occur after a latent period of 12 hours to 12 days.

1. Signs and symptoms

(1) **GIT:**

(i) Abdominal pain [most common]. Accompanied by

(ii) Vomiting and either

(iii) Diarrhea [if irritant effect of Tl is predominant] or

(iv) Constipation [if involvement of vagus nerve is predominant ".d intestinal motility, .d peristalsis]

(v) Severe symptoms, eg hematemesis and bloody diarrhea are more rare.

(2) **Chest** – Tightness and pain [vagus involvement]

(3) **CVS:**

(i) Tachycardia and

(ii) Hypertension [both develop after 1-2 wks. These are due to (a) vagus involvement (b) Tl stimulates ATPase in the chromaffin cells"catecholamines -"sinus tachycardia. Persistent and pronounced tachycardia points to poor prognosis

(iii) **ECG changes** [due to direct myocardial damage] – (a) prolongation of the QTc interval, (b) T-wave flattening or inversion, and (c) nonspecific ST-segment abnormalities

(4) **Skin** – Maculo-papular skin eruption having butterfly distribution on face [very characteristic]

(5) **Coma** – Occurs with large exposures. If recovery occurs, patient may suffer from permanent Guillain-Barré like polyneuritis.

2. Fatal dose

6-40 mg/kg of Tl salts. Death is due to coma with loss of airway-protective

reflexes, respiratory paralysis, and cardiac arrest.

3. Fatal period

24-36 h.

B. Chronic Poisoning

Due to repeated administration [homicidally or taken as medicine]. **Most characteristic lesions** are abdominal pain [seen also in acute poisoning], alopecia and neurologic symptoms [Thallium triad].

(1) **Alopecia:** [**Fig 36.7**]

(i) Most common and classic manifestation of Tl toxicity.

(ii) Typically it is the presenting symptom.

(iii) Begins in 10 days. Total hair loss in 30 days.

(2) **Neurologic symptoms:** (i) painful ascending peripheral neuropathy [appears after 2-5 days]

(3) **Skin:** (i) acne, (ii) palmar erythema, (iii) anhidrosis and (iv) dry scaly skin [results from damage to the sebaceous glands].

(4) **Nails** - Mees lines appear within 2-4 weeks after exposure [also seen in arsenic].

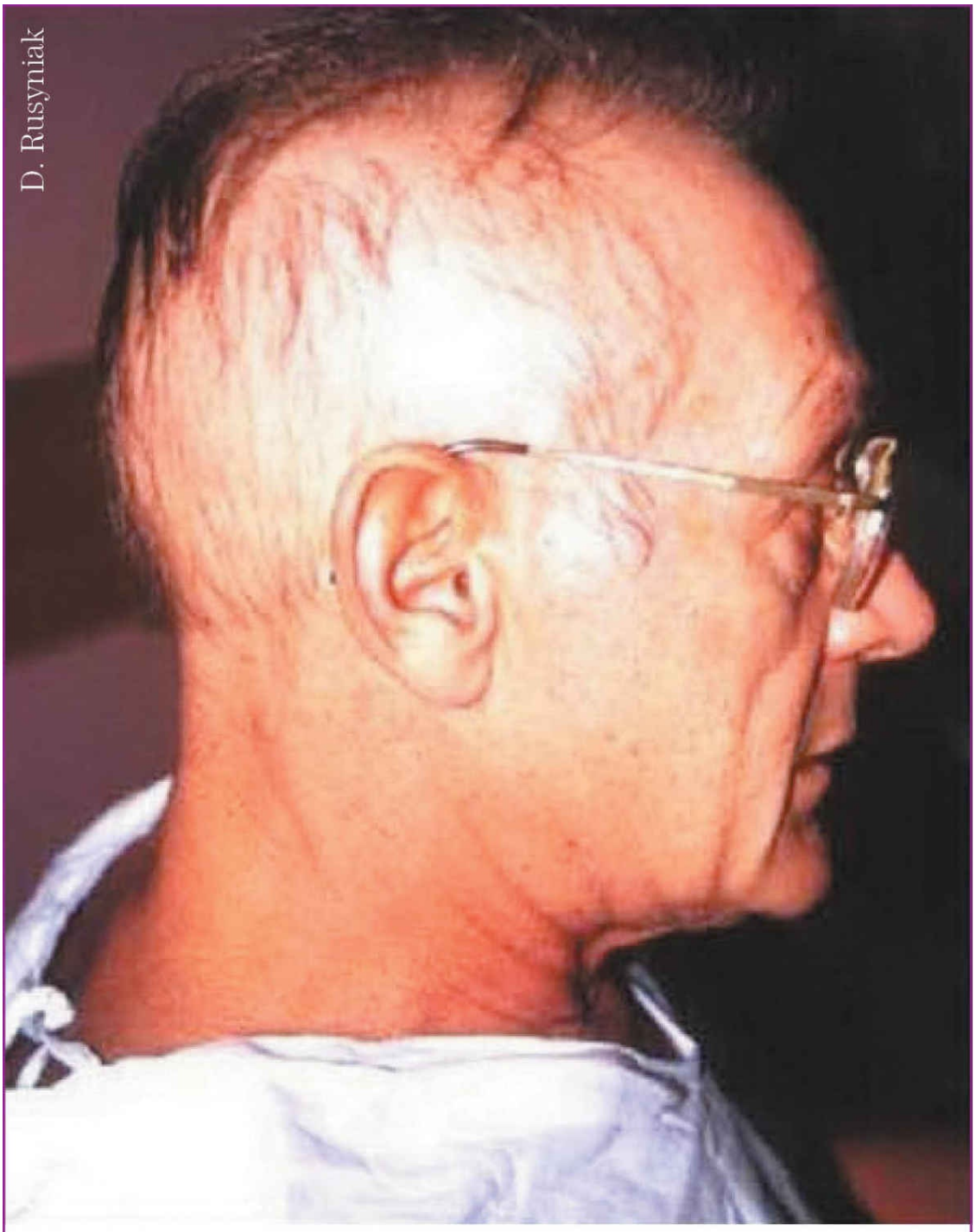


Fig. 36.7: Thallium-induced alopecia.

C. D/d

Must be differentiated from neuropathy seen in

(1) Poisoning by (i) arsenic, (ii) colchicine, and (iii) vinca alkaloids;
(2) Botulism;
(3) Thiamine deficiency; and
(4) Guillain-Barré syndrome.

D. Management

(1) **Gastric lavage** - with **1% KI** or **Prussian blue.** After the lavage Prussian blue is instilled through a duodenal tube directly into the duodenum [since Tl diminishes gastric and intestinal motility]. The dose is 125 mg/kg bd, with 50 ml of 15% mannitol [laxative to counter constipation].
(2) **Activated charcoal** [**AC**]– Since Tl undergoes enterohepatic recirculation, AC may be useful both in acute and chronic poisoning.
(3) **Whole-bowel irrigation** with polyethylene glycol
(5) **Prussian Blue** [potassium ferric hexacyanoferrate]- 125 mg/kg bd orally via a nasogastric tube. Causes -ed fecal elimination, and .ed blood Tl^+ conc. **Mechanism** - Acts as an ion exchanger. Interferes with the enterohepatic circulation by exchanging K^+ ions for Tl^+ ions in GIT"formation of a conc gradient causing"-ed movement of Tl^+ into GIT.
(6) **Charcoal hemoperfusion and hemodialysis** - especially if the patient has renal insufficiency
(7) Forced diuresis.

E. PM Appearances

In fulminant cases, death occurs before any gross changes become apparent. In cases which death occurs days or weeks later, following changes may be seen. Of these ***GIT*** and ***lung*** changes are seen in acute poisoning too.

(1) **Hair** – Alopecia
(2) **Mouth** – Stomatitis
(3) **CNS** –
(i) Cerebral cortical changes, especially in autonomic nervous system. Widespread degeneration of axons and nerve cells in the brain
(ii) Meninges – congested
(4) **CVS** - Fatty degeneration of the heart [seen as a “tabby cat” striation of both ventricles

(5) **Lungs** – Edema
(6) **Kidneys** – Renal damage. Nephrosis
(7) **GIT** -
(i) Stomach and intestines – (a) Signs of gastroenteritis. (b) Mucosa inflamed, ulcerated. (c) Submucosal petechial hemorrhages
(ii) Liver - fatty degeneration
(8) **In cremated bodies** – Thallium can be detected in the ashes.

37. Vegetable Poisons

I. RICINUS COMMUNIS (CASTOR, *ARANDI*)

The pathology of ricin and abrin poisoning was described by **Flexner** in **1897**.

Salient features:

(1) **Habitat** - Ricinus communis [Latin for "common tick" because the seeds resemble ticks (**Fig 37.2**)] grows all over India.
(2) **Plant** [arandi, wonder tree] -
(i) The plant [family Euphorbiaceae, (spurge family)], is native to tropical Africa.
(ii) Grows like a bush [**Fig 37.1**].
(iii) Cultivated in many countries as an ornamental annual plant in gardens.
(iv) It is a shrub, 1–4 m high, branched with green or reddish leaves.
(v) Has clusters of seed pods covered with fleshly spines.
(3) **Leaves** – (i) Glossy, (ii) 15–45 cm long, (iii) long-stalked, (iv) alternate and palmate with 5–12 deep lobes with coarsely toothed segments.
(4) **Fruit** - The fruit [pod] is a spiny, greenish to reddish purple capsule [**Fig 37.1**] containing large, oval, shiny, bean-like, highly poisonous seeds with variable brownish mottling. Contain three seeds per capsule.

Fig. 37.1: Ricinus communis plant

(5) **Seeds** – There are two varieties of seeds (i) Large red seed with brown blotches [**Fig 37.2**] – yield 40% oil. Double the size of small seeds. Used mainly for illumination (ii) Small grey seed with brown spots – (a) Size - 1.2x0.8 cm (b) resemble croton seeds [**Table 1**] (c) yield 37% oil. (d) Better quality. Used for medicinal purposes. Oil does not contain ricin; it remains behind in bean pulp during extraction. Extraction during heated conditions inactivates ricin within the pulp too.

(6) **Poisonous parts** - All parts of the plant are poisonous but seeds are most poisonous [ricin: 1-5%; ricinine: 0.3–0.8%].

(7) **Uses** - The seeds contain between 40% and 60% of a **yellowish oil** (**castor**

oil or **ricinus oil**), which is used as an

(i) Additive - in candles, cosmetic creams, diffusion pump oils, linoleum, lipsticks, lubricants, paints, plasticizers, printing-inks, soaps, transparent paper and varnishes.

(ii) Lubricant - From World War I until the 1960s it was used as a lubricant for jet engines, high-speed automotives, and industrial machinery. Castor plants were cultivated in large quantities until synthetic oils became available for use.

(iii) Food industry - Flavorings, candy (eg chocolate), as a mold inhibitor and in packaging. Castor oil is used, which is not poisonous. In **Nigeria**, the whole beans are eaten as food, after boiling [heat inactivates ricin]

(iv) Medicinal [purgative] and lighting purposes

(v) Manure – seed cake is used

(vi) Fuel, thatching material or for preparing paper pulp – Plant stalks are used

(vii) Silkworm feed - In the silk-producing areas, leaves are fed to the silkworms.

(viii) Ornamental - In countries where synthetic oils have become common, the plant is now grown only for its ornamental value.

Fig. 37.2: Ricinus communis (castor) seeds besides a ₹5 coin for size comparison. Please note that they look like ticks

A. Active Principles

(1) **Ricin** [toxalbumin]

(2) **Ricinine –**

(i) A piperidine alkaloid.

(ii) A ***toxalbumin*** or ***phytotoxin*** is a toxic protein found in certain plants and bacteria, capable of stopping protein synthesis. Common toxalbumins are

ricin, **crotin** and **abrin**.

B. Mechanism of Action

Biochemically toxalbumin is a **Ribosome Inactivating Protein (RIP)**, capable of inactivating ribosomes and hence protein synthesis. **3** types of **RIP** are known – **Type I, Type II** and **Type III**. It is best to study **type II RIP** first.

(1) **Type II RIP** [**syn:** type 2 RIP] is composed of an **A chain** (or effectomer) that is enzymatically **A**ctive (**Memory Aid:** **A**"**A**ctive) and a **B chain** (haptomer) that **B**inds (**Memory Aid.** **B**"**B**inds) the toxin to the surface of cells (**Fig 37.3**) [Type II RIP is thus sometimes referred to as A–B toxin]. Once the RIP **b**inds to the cell surface by the **B** chain, it enters cell by endocytosis. In the cytosol, the A chain inactivates ribosomes"Protein synthesis ceases. One A chain molecule can inactivate approximately 1500 ribosomes per minute, leading to rapid inhibition of protein synthesis and cell death. [**Ex** – Abrin, Crotin, Ebulin [from Sambucus ebulus], Modeccin [from Adenia (Modecca) digitata], Ricin, Shiga toxin (from Shigella dysenteriae) and the related Shiga-like toxin (from certain enterohemorrhagic strains of E.coli), Viscumin (from Viscum album)]. Type II RIPs evolved from type I RIPs.

(2) **Type I RIP [syn:** type 1 RIP**]** consists only of A chain, and is thus active against ribosomes only in vitro; it can not enter the cell on its own. [**Ex** - Agrostin (from Agrosternma githago), Asparin (from Asparagus officinalis).

(3) **Type III RIP [syn:** type 3 RIP**]** - are actually inactive precursors (**proRIPs**) that require proteolysis before they become active. They are the **least prevalent of all 3 types**. Till date, they have been isolated only from **maize** and **barley.**

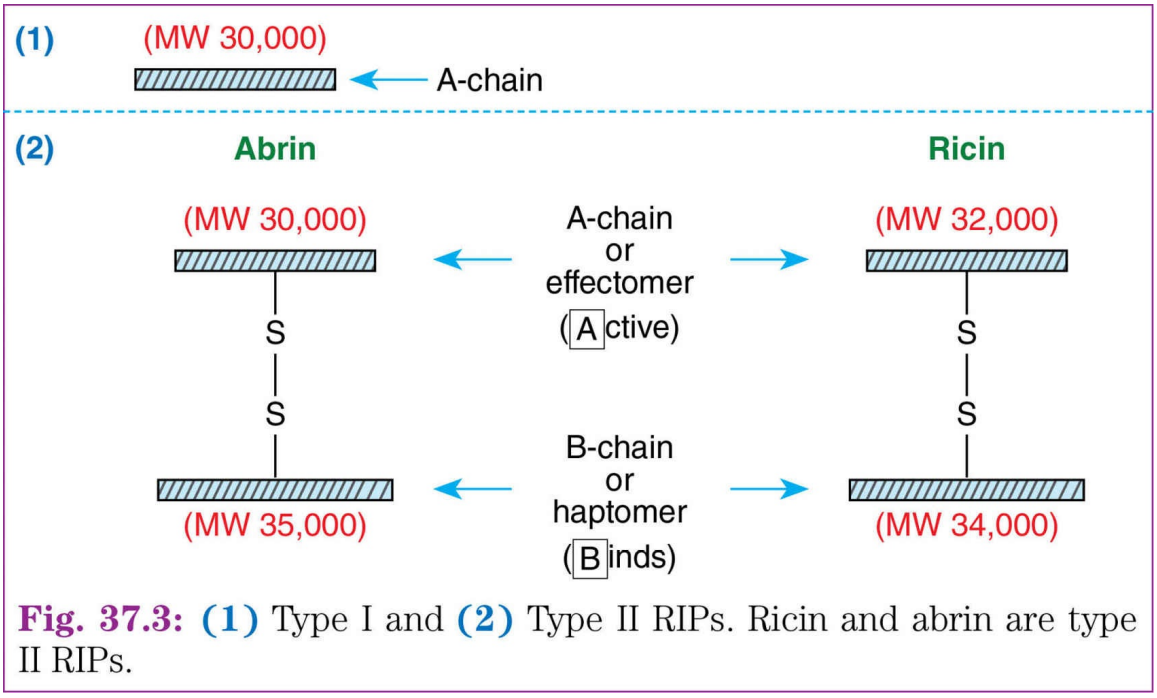


Fig. 37.3: (1) Type I and **(2)** Type II RIPs. Ricin and abrin are type II RIPs.

C. Ricin

After oil is extracted, the **bean pulp** (or fibrous residue) that is left behind contains ricin (approx 1 to 5% of seed's wt). Ricin is a water soluble toxalbumin. It is not present in the oil; castor oil is thus non-toxic (unlike croton oil which is toxic). If oil is extracted under heated conditions, ricin is inactivated. **Ricin** is a **type II ribosome-inactivating protein [RIP]** (Fig 37.3). The seeds also contain **ricinolein**, a triglyceride which is mainly responsible for their purgative action.

D. Signs and Symptoms

1. Ingestion

(1) **Method of ingestion** –

(i) Swallowed - Do not cause poisoning. They pass through the GIT with little or no toxic effects, because the hard seed coat cannot be digested by gastric juices.

(ii) Chewed - Toxic because ricin is released.

(2) **Severity of poisoning** –

(i) Mild - results in nausea, vomiting, diarrhea, and abdominal pain.

(ii) Moderate to severe - GIT symptoms progress to dehydration, hypotension,

liver and renal dysfunction [-BUN, -creatinine], and death.

2. Inhalation

Dust of seeds causes conjunctivitis, cough, sneezing, acute nasal inflammation, dyspnea, arthralgias, fever, respiratory distress and death.

3. Injection

Generalized weakness, myalgias, hypotension, multiorgan failure and death. Anaphylactic reactions may occur.

E. Diagnosis

(1) H/o ingestion of Ricinus seeds
(2) Detection of seeds in vomitus or stool
(3) Occurrence of compatible clinical signs
(4) Detection of ricin or ricinine in antemortem or postmortem samples.

F. Fatal Dose

(1) **Seeds** - 5 well-chewed seeds. Intact seeds are typically not digested and therefore do not release ricin.
(2) **Ricin** –
(i) By injection or inhalation - 20 µg/kg (1.4 mg for an average adult weighing 70 kg). This is less than $1/250^{th}$ of a standard aspirin tablet
(ii) By ingestion - About 1000-fold less [20 mg/kg], due to relatively low bioavailability of ricin (≤1%).
(3) **Castor oil** [unlike croton oil] is not poisonous.

G. Fatal Period

2 days.

H. Management

(1) **Mainly supportive** - IV fluids and vasopressors (eg, dopamine) for hypotension. There is no antidote for ricin
(2) **Activated charcoal** - if vomiting has not begun and airway is secure
(3) **Gastric lavage** - if ingestion has occurred within an hour

(4) Whole bowel irrigation
(5) **Demulcents.**

I. Prophylaxis

Animal studies have shown that anti-ricin antibodies administered by inhalation, 1 h prior to an aerosolized ricin challenge, .es lung damage.

J. PM Appearances

(1) **GIT mucosa** – congested, softened and inflamed. Erosions and submucosal hemorrhages are seen
(2) **Hemorrhages** – in internal organs
(3) **Serous cavities** – contain blood
(4) **Fragments of seeds** – Found in stomach or intestines.

K. ML Importance

(1) **Manner of poisoning:**
(i) Accidental - Necklaces made of castor oil seeds can cause
(ii) Suicide – Can be used. A case is reported of the death of a 49 y old man who injected himself s/c and IV with approx 10 mL of a 'self-made' acetone extract of castor beans, with an intent to commit suicide. He died within 9 h despite being given the best medical care.
(iii) Homicide – Rarely used
(2) **Destruction of unwanted children** – by giving crushed castor seeds in their foods
(3) **Infanticide** – In East Africa, unwanted children are murdered by adding castor beans to their food
(4) **Abortifacient** – Castor oil used as abortifacient [**ch 26**]
(5) **Malingering** – Powder of seeds applied to eye produces conjunctivitis.
(6) **Biological Weapon** – Ricin is codenamed "**Compound W**". During World War II, the U.S. and Great Britain worked together to develop a "W bomb", but abandoned research midway. Remains a potential biological warfare agent.
(7) **Medicinal use of ricin** - treatment of cancer and AIDS.
(8) **Terrorist weapon** – Ricin is a potential terrorist weapon.
(i) **1978 -** The Russian secret police KGB assassinated the Bulgarian defector

Georgi Markov by ricin. A weapon disguised as an umbrella injected a small metal pellet containing ricin into Markov's thigh. More recently ricin has been stockpiled by terrorist organizations.

(ii) **1989** - Iraq reportedly conducted tests to spread ricin as an aerosol.

II. CROTON TIGLIUM (*JAMALGOTA, NAEPALA*)

Croton tiglium (Purging Croton) [family Euphorbiaceae] grows all over India. Name comes from Gk kroton, "tick"; seeds resemble ticks. Seeds are poisonous and contain a toxalbumin **crotin**. They are odorless, dark brown and oval with longitudinal lines (**Fig 37.4**). **Croton oil** [Crotonis Oleum] present in the seeds is about 50-60% by weight of seeds. Like croton seeds, croton oil is also poisonous (unlike castor oil, which is non-poisonous), as it contains **crotin**. Croton oil is carcinogenic also. Physically, croton oil appears as a thick, brown, viscid oil having an unpleasant odor and acrid, burning taste.

Fig. 37.4: Seeds of *Croton tiglium*

A. Active Principles

(1) **Crotin** [toxalbumin] (2) Crotonal [a non-purgative fraction] (3) crotonic acid (4) crotonic resin (5) crotonoleic acid [A mixture of croton resin with inactive fatty acids. A powerful vesicating resin] (6) **Crotonoside** [glycoside] (7) Glyceryl crotonate (8) Tiglic acid [syn, methyl crotonic acid]. Also present are (9) several volatile acids (10) fatty acids (11) tumor-promoting phorbol esters (i) phorbol formate, (ii) phorbol butyrate, and (iii) phorbol crotonate.

Table 1: Differences between castor and croton seeds

S.No	Feature	Castor seeds	Croton seeds
1.	Appearance	Shiny brown with mottled patches. Appear polished.	Dark brown, or dull gray shell, which on scraping becomes black. Appear dull.
2.	Size	Two sizes. Smaller is 1.25×0.85cm. Larger double the size	One size. Almost same as the smaller castor seed
3.	Cross section of lumen	Circular	Slit like with radiating creases
4.	Microscopy of the prismatic cells in the outer coats of seeds	1. **Size -** (i) length of cells - 250µ (ii) size of Lumen - 3µ 2. **cell-walls -** show fine transverse striae giving the cells a ribbed appearance.	1. **Size -** (i) length of cells -300µ (ii) size of Lumen - 1µ 2. **cell-walls -** striae not noticeable. Cells do not show a ribbed appearance.

B. Signs and Symptoms

1. Ingestion

(1) **GIT** - (i) Vomiting and diarrhea (ii) Salivation (iii) Hot burning pain from mouth to stomach

(2) **Systemic** - (i) Vertigo (ii) Prostration (iii) Collapse (iv) Death.

2. Application on skin

Application of oil on skin produces (1) burning, (2) redness, (3) vesication.

C. Fatal Dose

(1) **Seeds** – 4 (2) **Croton oil** – 20 drops.

D. Fatal Period

6 hours -3 days.

E. Management

(1) Gastric lavage (2) Demulcents (3) symptomatic.

F. PM Appearances

(1) **Mucosa of GIT**- congested, inflamed, excoriated

(2) **All internal organs** - congested.

G. ML Importance

(1) **Accidental poisoning** – results from ingesting croton seeds or oil by mistake, or when taken in large doses as medicine (purgative), or by inhalation of dust

(2) **Abortifacient** – Root and oil [**ch 26**]

(3) **Arrow poison** – paste made by crushing the soft parts of plant [not the seeds]

(4) **Ayurveda** - In Ayurvedic system of medicine croton seed oil is used in minute doses in cerebral affections [apoplexy, convulsions, insanity], dropsy, high blood pressure, intestinal obstruction, lead poisoning and obstinate constipation. May cause poisoning if incorrectly used. Formerly also used as a counterirritant in all systems of medicine.

(5) **Suicide and homicide** – Rare. Fruit is boiled in water and added to food

(6) **Poisoning animals** – croton oil has been used to poison fish in tanks. Mixed with beef it has been used mischievously to kill pet tiger belonging to a circus party.

(7) **ordeal poison [ch 31]**

(8) **As a counterirritant** - Diluted with a suitable inactive vehicle, croton oil was once used as a counterirritant.

(9) **Chemical peels** – Due to its caustic exfoliating effects, croton oil [in combination with phenol] has been used in chemical peels. Leads to initial skin sloughing and then eventual regeneration.

(10) As an **additive to fuel** to render it unsafe for drinking – Can be added to fuel (comprising of alcohol) to render it unsafe for drinking (much as methyl alcohol is added to ethyl alcohol to denature it). US did so in **World War II** to prevent sailors from drinking fuel (comprising of grain spirits). Reportedly a number of sailors distilled alcohol away from the mixture and still consumed it!

III. ABRUS PRECATORIUS (*RATTI*)

Abrus precatorius (Ratti, Gunchi, Jequirity, Crab's Eye, Rosary Pea) is a slender, perennial climber found all over India that twines around trees, shrubs, and hedges. In **1891**, **Hellin** discovered the agglutinating properties of abrin.

Salient features:

(1) **Leaves** – compound, feather like; 1-2.5 cm long; 10-15 pairs of narrow leaflets.

(2) **Flowers** – small and pale violet, arranged in clusters, with a short stalk.

(3) **Seed pods** – 2.5-5 cm long, borne in clusters. Green when immature; on maturing, become dry and brown; split open while remaining on vine with 4-6 seeds exposed. All parts of the plant are poisonous.

(4) **Seeds** – (i) Size - Pea sized. (ii) Color - Bright scarlet color with a large black spot at one end [**Fig 37.5**]. Rarely the color may be white, yellow, blue, or all black. (iii) Odorless and tasteless. (iv) Weight - Supposed to be quite uniform in weight. In India, it was traditionally used as a standard weight (0.12125 grams) by jewelers.

Fig. 37.5: *Abrus precatorius* seeds.

A. Active Principles

(1) **Abrin** [a toxalbumin similar to **viperine snake venom**] (2) **Abrine** (N-methyl L-tryptophan) [an amino acid] (3) N,N-dimethyl-L-tryptophan [plant growth inhibitor] (4) Hypaphorine (5) **Precatorine** (6) **Choline** (7) **Trigonelline**.

B. Mechanism of Action

(1) **RIP** - Abrin has a structure similar to ricin, and acts essentially in the same manner.

(i) Abrin has 2 chains A and B [**Fig 37.3**]. **A chain** [molecular weight 30 kDa] has **251 amino acid residues**, and is divided into three folding domains.

(ii) **B chain** [molecular weight 35 kDa] is a galactose-specific lectin. Consists of **268 residues**. Both are linked by a disulphide [S-S] bond between **Cys247** of the **A chain** and **Cys8** of the **B chain**. A single molecule of abrin is sufficient to kill a cell.

(2) **Abrin-induced endothelial cell damage** -

(i) Many clinical features can be explained by it.

(ii) Endothelial damage "- in capillary permeability "fluid and protein leakage"tissue edema [vascular leak syndrome]. This is also a recognized feature of ricin poisoning.

(3) **Direct toxic effect on CNS**.
(4) **Agglutination of RBCs.**

C. Signs and Symptoms

1. Topical contact

(1) **Ophthalmic exposure** - (i) severe inflammation of the conjunctiva (ii) localised necrosis. (iii) An infusion instilled in the eye may ran through the tear duct into the throat.
(2) **Dermal exposure** - No reports of human or animal toxicity.

2. Inhalation

(1) **Human toxicity** - has not been reported.
(2) **Rats exposed to abrin dust** -
(i) Onset of symptoms - 18–24 h
(ii) Mild poisoning - general malaise, lethargy, anorexia, piloerection, respiratory difficulties.
(iii) Severe poisoning - exudation of blood-stained fluid from nostrils.

3. Ingestion

(1) **Immediate** [onset few hours – 1 day] –
(i) GIT - (a) Severe irritation (b) Abdominal pain (c) Nausea and vomiting (d) hemorrhagic gastritis (e) Bloody diarrhea, Rectal bleeding
(ii) Pupils – constricted
(iii) Pulse – rapid, weak
(iv) Dyspnea
(v) General - (a) Cold perspiration (b) Trembling of hands (c) Weakness, general prostration, faintness, vertigo
(2) **Delayed** [onset 2-5 days] –
(i) Delayed cytotoxic effects in CNS, liver, kidneys and adrenal glands.
(ii) Convulsions.

4. Injection

Injected in the form of sui [**Fig 37.6**]
(1) **Local** - (i) Painful swelling and edema (ii) ecchymosis (iii) Inflammation (iv)

Oozing of hemorrhagic fluid and necrosis around site of puncture

(2) **General** – (i) General weakness, faintness, vertigo (ii) inability to make movements, (iii) no desire to take food (iv) tetanic convulsions (v) temp. (vi) drowsiness, coma and death in 3-5 days. Symptoms resemble viperine snake bite.

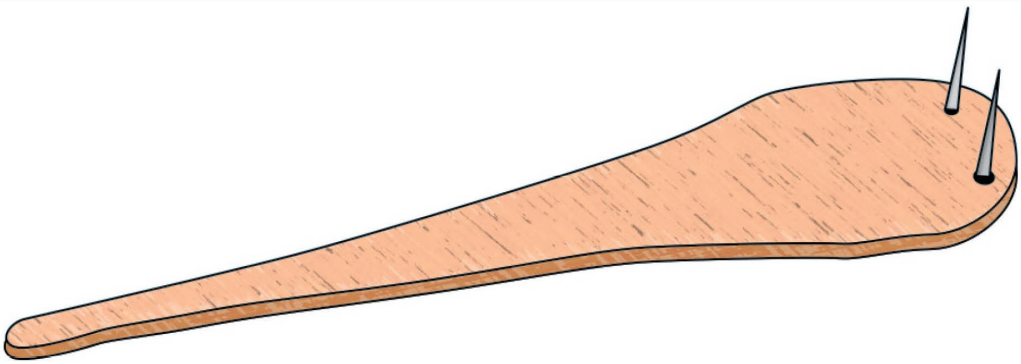


Fig. 37.6: *Suis* mounted on a wooden handle

D. Fatal Dose

(1) **Abrin** [IV]- 0.1–1.0 µg/kg.

(2) **Seeds** [ingestion]- 1-2 crushed seeds [i.e. if they are chewed]. As many as 20 unchewed seeds may be harmless.

E. Fatal Period

3-5 days.

F. Management

1. Topical contact

Eyes -wash eyes with running water for ten minutes. Rest as in ingestion below.

2. Inhalation

(1) Remove from exposure.

(2) Maintain clear airway; ensure adequate ventilation ensured

(3) Treat pulmonary edema conventionally. Rest as in ingestion below.

3. Ingestion

(1) **Remove all seed particles** from the mouth
(2) **Collect remaining seeds** or plant material for identification
(3) **Induce vomiting** and send it for chemical examination
(4) **gastric lavage**
(5) **Activated charcoal**
(6) **Sodium bicarbonate** – 10 g orally/d"maintains alkalinity of urine; prevents agglutination of RBCs, and thus blockage of renal tubules
(7) **Antiabrin antibodies** – no role in management [in all routes of exposure]; very limited role in prophylaxis [please see below]
(8) **Hospitalization** - Patient should be hospitalized for several days because severe symptoms can develop several days after ingestion.

4. Injection [*Sui*]

Sui (needle) should be dissected out. Rest as above.

G. Cause of Death

Cardiac failure.

H. Prophylaxis

(1) **Hyposensitization** - Mice injected with increasing sub-lethal doses of seed extract develop tolerance. Not similarly tested in humans.
(2) **Abrin Toxoid** - Rats administered with abrin toxoid were protected against aerosolized abrin
(3) **Ascorbic Acid** – Repeated bolus doses of ascorbic acid, sodium ascorbate and calcium ascorbate over a 48-hour period "-es survival time.
(4) **Anti-Abrin Antibodies** – Very limited role.

I. PM Appearances

(1) **Puncture marks** - as in snakebite
(2) edema and petechial hemorrhage [in quick deaths] and purulent sepsis and necrosis [in late deaths] in the area where sui was introduced
(3) Fragments of sui in the skin
(4) **Internal organs** – congested; show hemorrhages

(5) **Stomach** – In ingestions, gastric lining shows congestion with submucous hemorrhagic spots

(6) **Other Mucus membranes** – pleura, pericardium, peritoneum may show petechial hemorrhages

(7) **In inhalation** – Lungs strongly congested.

J. ML Importance

(1) **Cattle poison** –

(i) Seeds are used.

(ii) Leather workers use this method to obtain leather from cattle

(iii) Cattle of enemy may be killed for revenge

(2) **Homicidal poison:**

(i) In the form of Sui or sutari [needle]

(ii) Method of preparation of Sui - (a) Seeds are decorticated (b) Mixed with datura, opium, glass powder and onion"powdered. Sometimes used alone (c) A paste is made by mixing this powder with spirit and water (d) Sharp pointed spikes [suis, needles] about 15 mm long and weighing 90-120 mg are made from this paste (e) Dried in the sun

(iii) Method of killing - (a) Two such suis are mounted on a wooden handle [**Fig 37.6**] (b) The handle is struck on the back of a cattle or human. Sometimes needles are kept between fingers and the person is slapped. (c) Needles lodge inside the flesh; the person thinks he has been struck only with a blunt instrument [or slapped] and after picking up a little fight allows the assailant to go away (d) the injection site suppurates and person dies within 3-5 d.

(iv) Advantage to killer – Since symptoms resemble viper snake bite, killer is not under suspicion.

(3) **Malingering** –

(i) Powdered abrus introduced in eye produces strong conjunctivitis. Used by persons [especially army jawans, where getting leaves is difficult] to obtain medical leave.

(ii) Unilateral conjunctivitis should arouse suspicion.

(4) **As contraceptive and abortifacient** – Because of irritant action. Used by vaids and hakims and sometimes by patient herself. Action not reliable.

(5) Arrow poison.

IV. CAPSICUM ANNUUM (CHILI)

(1) The **Capsicum** genus is a member of the Solanaceae family which includes tomatoes, potatoes, and the deadly nightshades. This genus includes chili peppers, red peppers, and paprika, but not black pepper.
(2) ***Capsicum annuum*** (chilies, red pepper, lal mirch) has a pungent odor and taste and is widely used in India and around the world as a condiment. In India, it is widely used in preparing curries and chutneys. Though it causes intense irritation, it is not fatal. The active principles are (i) **capsaicin** and (ii) **capsicin**.

A. Signs and Symptoms

(1) **On oral ingestion** – Burning pain in mouth, difficulty in swallowing, pain in abdomen
(2) **On eye contact** – intense blepharospasm, inflammation, lachrymation
(3) **On dermal contact** –Hunan hand - Intense burning pain, hyperalgesia, erythema and dermatitis, after handling chili powder with bare hands. Common in cooks, who prepare food with chilies without using gloves. **Hunan hand** is so named because it was common in **Hunan province of China**. Capsaicin releases **Substance P**, an undecapeptide (peptide composed of 11 amino acids) from afferent sensory neurons, causing pain (Substance P is the neurotransmitter for communication of pain and itch sensation from the periphery to the CNS). The symptoms are due to nerve receptor stimulation and not local injury to the skin.

B. Management

(1) Wash local skin with ice-cold water for 15 minutes
(2) emollient creams
(3) irrigation of eyes
(4) give demulcents and ice cubes to suck if symptoms due to ingestion.

C. ML Importance

(1) **Robbery** - Chili powder is thrown in eyes to facilitate robbery

(2) **Burning of chili powder** - by superstitious people to scare away ghosts and spirits
(3) **Introduced in vagina** - for infidelity
(4) **For obtaining confession of crime** – chili powder introduced in vagina, urethra etc
(5) **Pepper spray** - used by ladies in the west to drive away potential rapists
(6) **Homicidal asphyxia in children** - by forcibly introducing pepper in the nostrils and mouth of unwanted young children
(7) ***Hyderabadi goli*** - Police torture. A lathi is liberally plastered with chili powder and is thrust up the victim's anus to extort confession.
(8) **Datura seeds** - must be differentiated from chili seeds (see chapter on deliriant poisons).

V. SEMECARPUS ANACARDIUM (DHOBI'S MARKING NUT)

Semecarpus anacardium (Hindi: Bhilwa, Sanskrit: Agnimukh) [family Anacardiaceae] is a native of India.

Salient features:

(1) **Tree** – Moderate sized deciduous tree with large stiff leaves.
(2) **Fruit (nut)** – (i) ripens from Dec-Mar (ii) 1 inch long, ovoid, oblong or heart shaped [**Fig 37.7**], smooth and shining, black when ripe, seated on a fleshy orange-colored cup [**Fig 37.7**]. Has a rough projection at the base. (iii) has a thick cellular pericarp which contains a bitter, irritant juice which is acrid, brownish and oily. It turns black on exposure to air. It is used in India as a substitute for marking ink for clothes by washermen [Dhobi Nut, Dhobi's marking nut]. The tree sometimes is called "**Ink tree**".

Fig. 37.7: (1) *Semecarpus anacardium* nut. **(2)** Nuts seated on fleshy orange colored cup.

A. Active Principles

(1) **Bhilawanol** [15-17%] – A mixture of cis- and trans isomers of urushiol
(2) **Semecarpol** [0.1%].

B. Signs and Symptoms

(1) **External application** –
(i) Produces a dark colored lesion which resembles a bruise [**artificial bruise**].
(ii) Eczematous eruptions of the neighboring skin with which it comes in contact
(iii) Irritation, itching
(iv) Painful blister which contains acrid serum.
(v) Later an ulcer is produced.
(vi) Skin may slough off.
(2) **Ingestion** -
(i) GIT - Blisters on throat, severe GIT irritation with vomiting and diarrhea
(ii) RS – Dyspnea
(iii) CVS – Cyanosis, Hypotension, Tachycardia
(iv) CNS - Reflexes absent, Delirium, coma and death.

C. Fatal Dose

5-10g.

D. Fatal Period

12-24 h.

E. Management

(1) **If applied externally** –

(i) Wash with lukewarm water and water. If patient has presented with suspected fabricated bruises, preserve for chemical analysis.

(ii) Apply antiseptics.

(2) **If ingested** -

(i) Gastric lavage, (ii) demulcents. (iii) Preserve gastric lavage for chemical analysis in all cases.

F. PM Appearances

(1) Mouth, palate, throat, other oral structures – inflamed, show blisters

(2) Stomach – congested. May show fragments of seed.

G. MLI

(1) **Artificial bruises** – are made on arms, thighs, breasts etc to bring about a false charge of assault.

(2) **Criminal abortion** – juice is applied to the cervical os.

(3) **Infidelity** – juice is introduced in the vagina as a punishment.

(4) **Injury** - Juice is sometimes thrown on the body of other to cause injury

(5) **Malingerers** – put the juice inside their eye to produce ophthalmitis

(6) **Manner of poisoning** -

(i) Accidental poisoning – Common. Poisoning may occur because the nut is used in Indian medicine for a variety of disorders eg allergic dermatitis, ascites, asthma, colitis, cough, diarrhea, dyspepsia, leprosy, leukoderma, piles, poisonous bites, scaly skin, tumors and worms. The oil is topically applied on swollen joints and traumatic wounds. May result in false bruises.

(ii) Suicidal and homicidal poisoning – Rare

(7) **Occupational poisoning** - Dhobie mark itch (**Dhobie mark dermatitis**) is

urushiol dermatitis seen in Indian washermen who use **marking nut** in their work. Has been reported even in persons, who wear clothes laundered and marked in this way. The latter mode was the cause of dermatitis affecting several English soldiers stationed in pre-independent India.

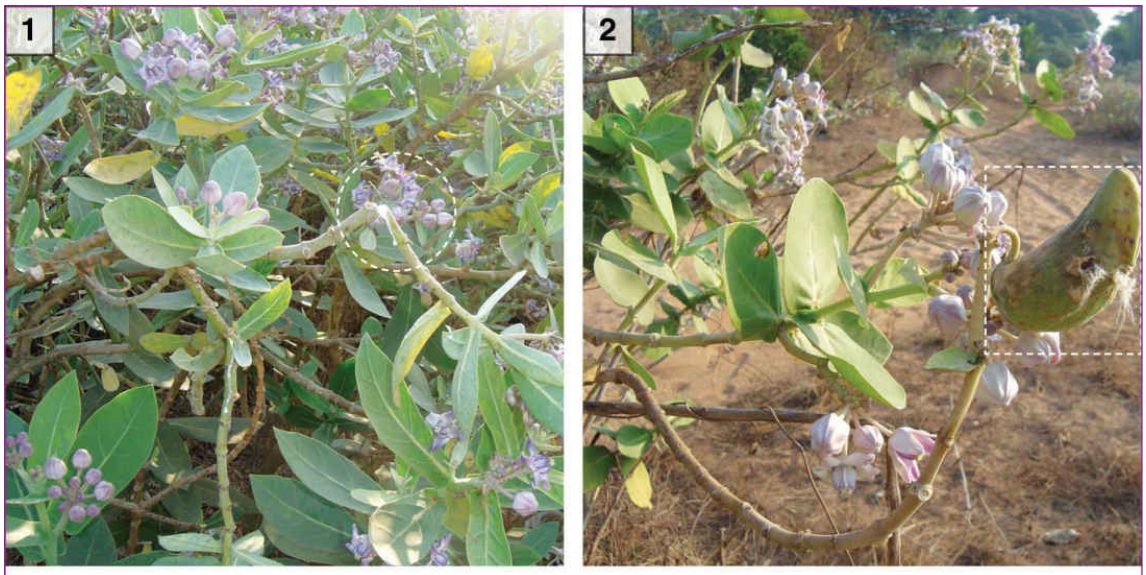


Fig. 37.8: (1) *Calotropis gigantea*. A stem has been broken to show the irritant juice coming out of it. **(2)** Plant showing fruit, with two punctures showing long silky hairs inside. Please see text for its uses.

VI. CALOTROPIS GIGANTEA (AKDO, MADAR)

A. Active Principles

Calactin, calatoxin, calotropin, trypsin, uscharidin and uscharin. They have CNS depressant and anticonvulsant activity.

B. Signs and Symptoms

1. Dermal application

(1) Redness (2) vesication and bullous eruptions.

2. Instillation in eye

Instillation of juice in the eye is often done for local eye ailments. Produces

1.keratitis, 2.conjunctivitis, 3.corneal edema, 4.dimness of vision.

3. Ingestion

(1) Acrid bitter taste
(2) Burning pain in mouth, throat and stomach
(3) Salivation
(4) Stomatitis
(5) Vomiting and diarrhea
(6) Pupils – dilated
(7) **In animal models** – cardiotoxic and hepatotoxic. In rats and sheep produces tracheal exudates, pulmonary edema, tachycardia, transitory cardiac arrhythmias, ascites, liver hemorrhages, hydropericardium, flaccid heart, and pale juxtamedullary renal cortex.
(8) Tetanic convulsions
(9) Collapse and death.

C. Fatal Dose

Uncertain.

D. Fatal Period

6-12 hours.

E. Management

(1) Gastric lavage (2) demulcents (3) symptomatic.

F. PM Appearances

(1) Pupils – dilated
(2) Mouth – stomatitis, froth
(3) nostrils – froth
(4) GIT – mucosa congested
(5) Brain, meninges, abdominal viscera - Congested.

G. ML Importance

(1) **Artificial** or **false bruises -** are produced by its juice [**ch 12**].
(2) **Cattle poison** –
(i) Smeared on a cloth, and pushed into the rectum of animal [cf ch 38"snake venom]
(ii) Given with fodder
(3) **Criminal abortion** – ingested or applied locally by abortion stick
(4) **Folk medicine** -
(i) Indian medicine - Flowers, leaves, root and juice are used. Leaves are used in body pain, burns, jaundice, mumps, rheumatism, sinus fistula and snake bite. Juice is used as a depilatory, in skin diseases and as a vesicant. May cause poisoning if injudiciously used.
(ii) In several countries, juice is instilled in eyes for ailments like chalazion. Can produce corneal edema and disturbances of vision.
(5) Infanticide
(6) **Manner of poisoning** -
(i) Suicidal poison
(ii) Accidental– White flowers used in India for worshipping Lord Shiva. May cause accidental poisoning, and ocular toxicity [conjunctivitis].
(iii) Homicide – rare because of bitter taste
(7) Arrow poison
(8) **Snakes** - Root of calotropis procera is thought to be highly poisonous to snakes, which cannot stand even its smell. It is thus kept by snake charmers in their baggage.

38. Animal Poisons

I. SNAKES

Snakes are elongate, legless, carnivorous reptiles of the suborder ***Ophidia*** [Class - Reptilia; Subclass: Lepidosauria; Order – Squamata (Scaled reptiles); Suborder – Ophidia (syn, Serpentes)] that are distinguished from legless lizards by their

lack of eyelids and ***external ears***. Living snakes are found on every continent except Antarctica and on most islands.

A. Classification

(1) There are about **3,500 species of snakes** in the world, but only about **350 species are venomous** and only the minority of these are likely to cause significant envenoming in humans.

(2) In India **330 snake species** exist. Of these **70 are venomous** [40 land snakes, 30 sea snakes].

(3) All snakes dangerous to humans may be divided into venomous and non-venomous.

Table 1: Difference between venomous and non-venomous snakes

S.No.	Trait	Venomous snakes	Non-venomous snakes
1.	General appearance	Stout, dull colored	Slender, brightly colored
2.	Head	triangular	Rounded or oval
3.	Head scales	1. Small (in **vipers**). Large in others 2. Large (a) pit between eye and nostril → **Pit viper** (b) Third supralabial touches the eye and nasal shields (Fig 38.1) [**cobra** or **coral snake**] (c) (i) No pit ii) third supralabial does not touch the eye and nasal shields. iii) Central row of scales on back enlarged and hexagonal in shape [Fig 38.3] iv) 4th infralabial is the largest (Fig 38.4) [**Kraits**]	Large with exceptions as mentioned in adjacent column
4.	Belly scales	Large and cover entire breadth [Fig 38.2]	Small, like those on the back. May be larger, but do not cover the entire breadth of belly
5.	Anal plate and subcaudal scales	Single row	double rows [Fig 38.2]
6.	Teeth	Two long fangs and a row of smaller teeth	Several small teeth arranged in rows, but no fangs
7.	Fangs	Canalized, like hypodermic needles [vipers] or have grooves [cobra]	Short and solid. Contain no canal or groove
8.	Poison glands	Present	Absent
9.	Saliva	Contains toxic polypeptides and enzymes	No
10.	Tail	1. may be rounded or flattened 2. tapers abruptly	1. Always rounded 2. tapers gradually
11.	Habits	Mainly nocturnal	diurnal

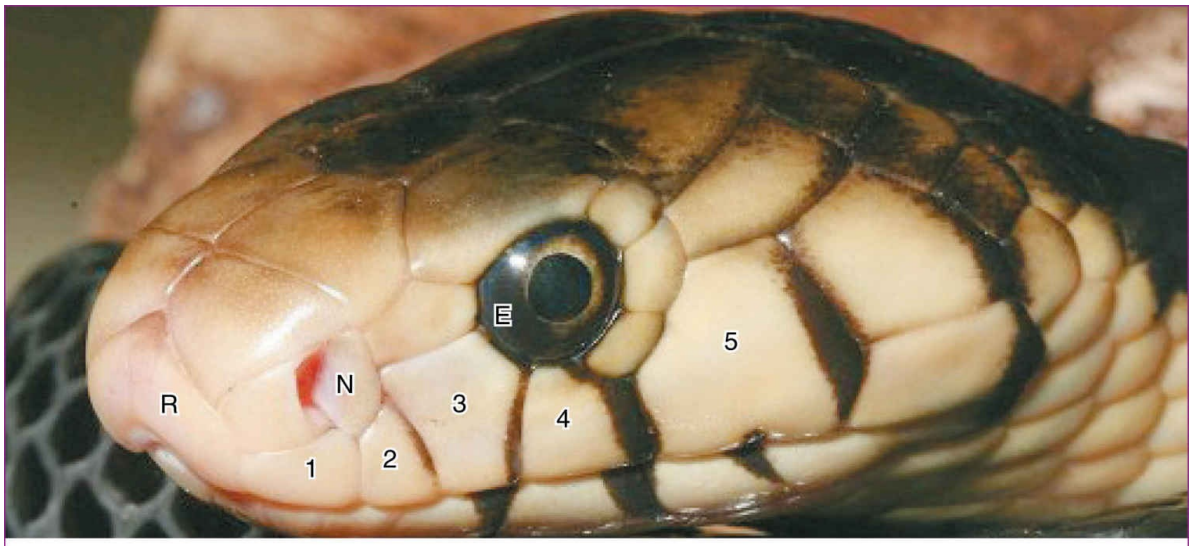


Fig. 38.1: Third supralabial (3) touches both eye (E) and nasal shields (N) in cobra and coral snake. Supralabials are counted numerically from the front, the foremost being called the **rostral scale** (R) or shield. It corresponds to the **mental scale** in the lower jaw [please correlate with **Fig 38.4**]. The term pertains to the rostrum, or nose. Counting of supralabials (1-5) is done from before backwards as shown.

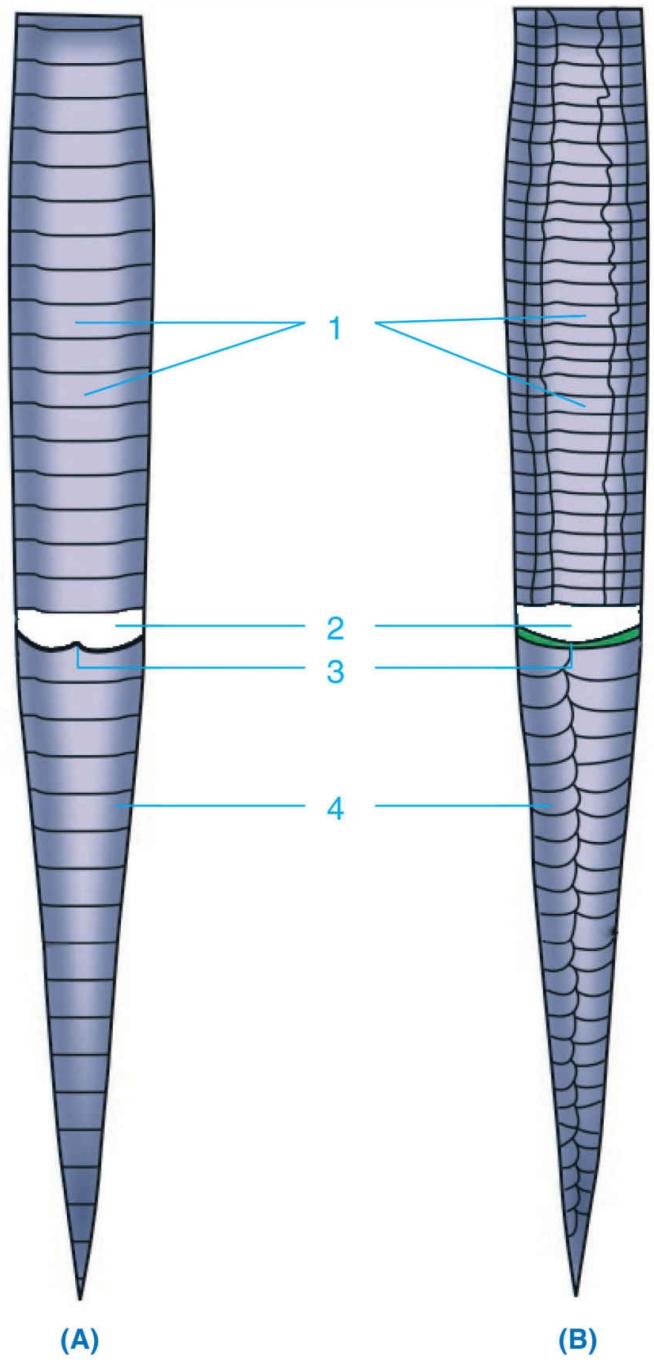


Fig. 38.2: Belly scales in **venomous (A)** and **nonvenomous (B)** snakes. (1) Belly scales (2) Anal scale (3) Cloaca (4) Subcaudal scales.

Fig. 38.3: Large hexagonal scales in the midline on the dorsum of **krait**. Blue colour has been artificially added to highlight the scales.

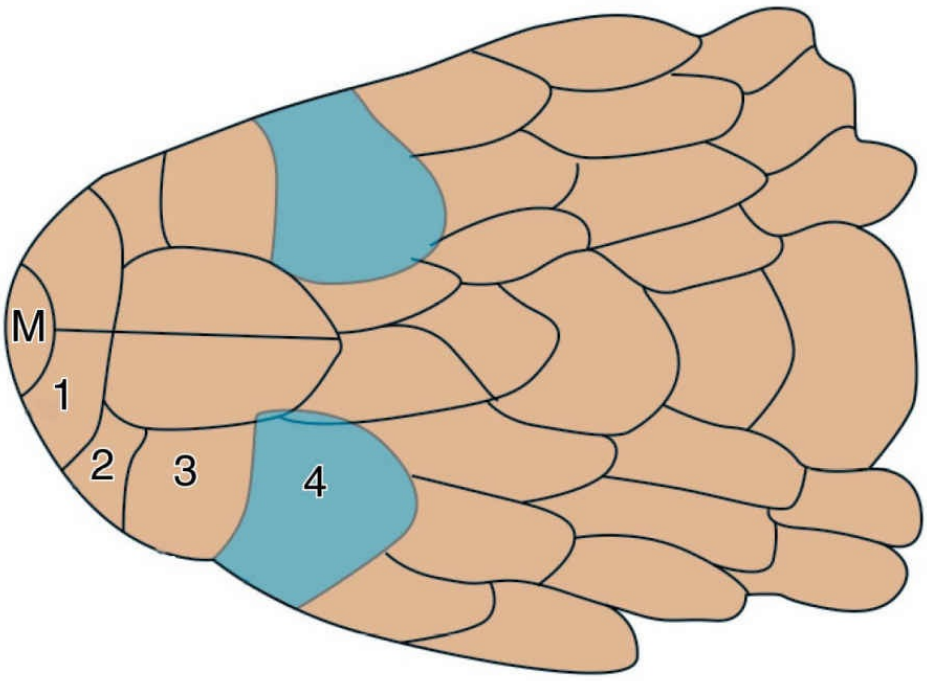


Fig. 38.4: Undersurface of the lower jaw of krait. **Infralabials** (1-4) are counted numerically from the front, the foremost being called the **mental scale** (M) or shield. It corresponds to **rostral scale** of the upper jaw [please correlate with **Fig 38.1**]. The term pertains to mental nerve which supplies chin. 4th infralabial (in blue) is the largest.

Venomous snakes

All venomous species of the world are grouped in just **five** families:

a. Colubridae

(1) Fixed and rear-fanged (Opisthoglyphous).

(2) Only few are dangerous to humans [**ex.** Boomslang, mountain racer, tree snake].

(3) The family includes about 2/3rd of all snake species on earth. Includes some non-poisonous also, eg rat snake.

b. Elapidae

(1) Fixed and front-fanged (Proteroglyphous).
(2) Cobras, kraits, mambas, coral snakes.
(3) They are a major global cause of snakebite.

c. Atractaspididae

(1) Side-fanged. (2) Include burrowing asps (3) Not a major cause of snakebites.

d. Viperidae

(1) Folding and front-fanged; fangs are like hollow syringe needles, [Solenoglyphous].
(2) Most important cause of snakebite globally.
(3) **Classification** -
(i) Divided into 4 subfamilies of which 2 are major – (a) **Viperinae** [true, typical or "Old World" vipers, eg African puff adders, European adders, Gaboon vipers, Russell's vipers and saw-scaled or carpet vipers] (b) **Crotalinae** [pit vipers eg, bushmasters, copperheads, cottonmouths, green pit vipers, habus, jararacusus, Malayan pit vipers, mamushi, rattlesnakes, water moccasins]. In addition, there are 2 monotypic [containing only one biological type] subfamilies (c) **Azemiopinae** [Fea's viper] and (d) **Causinae** [night adders].
(ii) Most US venomous snakes belong to Crotalinae. Both major Indian vipers [saw scaled viper and Russell's viper] belong to Viperinae.

e. Hydrophidae

[Gk. hydros, water; ophis, serpent]
(1) Fixed and front-fanged [like elapids], but habitat is water.
(2) Sea snakes. evolved from terrestrial ancestors [elapidae]; now adapted to a fully aquatic life; unable to move on land, except for the genus Laticauda [sea kraits], which retain ancestral characteristics, allowing limited land movement.

2. Non venomous snakes dangerous to humans

Pythons [family Pythonidae] and Boas [family Boidae] kill by constricting around chest causing traumatic asphyxia.

Table 2: Difference between Cobra and Viper

S.No.	Feature	Cobra	Viper
1.	Body and neck	Long and cylindrical	Short body; narrow neck
2.	Head	1. Small 2. Seldom broader than body 3. covered by large scales	1. Large and triangular (due to the venom glands located in both temporal regions) 2. Broader than body 3. covered by numerous small scales.
3.	Pupil	Round	Vertical
4.	Upper jaw	Carries poison fangs and other teeth	Carries only poison fangs
5.	Fangs	Short, grooved, fixed	Long, canalized, mobile. Retract posteriorly when the mouth is closed.
6.	Venom	Neurotoxic	Hemotoxic
7.	Tail	Round	Tapering
8.	Eggs	Lays eggs (oviparous)	Gives birth to young ones (viviparous)

B. Venomous Snakes of India

1. Commonly encountered

Saw scaled viper, **Russell's viper**, **Common cobra [Indian cobra]** and **common krait** are traditionally referred to as the "**Big four**", because they are responsible for virtually all snakebites in India.

a. Saw scaled viper

Zoological name - Echis carinatus [Gk Echis, viper; Latin carinatus, like a keel, referring to keeled scales]. 8 species of Echis are currently recognized.

b. Russell's viper

Zoological name - Vipera russelli, or Daboia russelli. Genus is named after the Hindi Daboia meaning "that lies hidden"; species in honor of **Patrick Russell** (1726–1805), a Scottish herpetologist who first described many of India's snakes

c. Common cobra

Common Names – (i) Bengali – Gokhra (ii) Hindi, Sanskrit, Oriya, Marathi – Naag, Kala saanp.

d. King cobra

The king cobra (Ophiophagus Hannah, Humadryad, Nagraj, Rajnag) is not a "true cobra" despite its name. True cobras belong to genus naja; it belongs to its own genus. It is the sole member of genus Ophiophagus.

e. Common krait

Common Names - (i) Bengali - Kalach (ii) Gujarati - Kala taro. (iii) Hindi - Karait.

f. Banded krait

Zoological name - Bungarus fasciatus. [from Telugu bungarum, gold, an allusion to the yellow rings around its body; Latin fasciatus, banded]

g. Sea snakes

Habitat - They are found in warm coastal waters from the Indian Ocean to the Pacific. Also in rivers, estuaries and even fresh water lakes.

C. Snake Venom

(1) Venoms of different snakes differ in antigenic structure, composition and toxicity

(2) **Cobra venom** – faint transparent yellow, slightly viscous, when exposed to

sun, it becomes slightly turbid

(3) **Russell's viper venom** – white or yellow

(4) **Composition** -

(i) Venom is the saliva of snake.

(ii) Composed of **90% proteins**, polypeptides and many other organic and inorganic substances. Together they are classified as enzymatic and non-enzymatic components. Enzymes produce local and systemic effects and non-enzymatic components produce lethality. **Proteome** [from **prote**ins and gen**ome**] is the entire set of proteins expressed by a genome, cell, tissue or organism. ***Venom proteome*** is the entire set of proteins present in snake venom. The study of snake venom proteomes is **snake venomics**.

(iii) No venomous snake has all components. Most snakes have 6-12 of these enzymes in their venom.

(iv) Each of these enzymes has its own special function. Some aid in the digestive process, while others specialize in paralyzing the prey.

Absorption and excretion

(1) **Transport** - Snake venom travels through lymphatics and superficial veins [that is why a light tourniquet may be useful – please see below].

(2) **Routes of absorption** - Poisonous only when injected; harmless when taken by mouth, as it is not absorbed through stomach. Oral sucking of venom may not harm the rescuer, if he accidentally swallows poison, but if he has ulcers in the mouth, venom can get absorbed from there and cause harm.

(3) **Excretion** - by kidneys, milk, salivary glands and mucus membranes.

II. SNAKEBITES

A. General

(1) When a snake bites, it normally leaves two faint impressions, the distance between them being 8 mm to 4 cm.

(2) A side swipe may produce a single puncture, and also small marks of other teeth

(3) The exact number of fang marks vary because of (i) Glancing bites (ii) Multiple fangs (iii) Multiple strikes (iv) Protection from clothing and shoes

(4) Non venomous snakebites leave a number of small impressions in a row.

(5) **Season** - During summer months, venom output is more and the venom is more toxic than during winter.

(6) **Dry bite** - When a snake bites, but venom fails to get injected, it is known as a "dry bite". Approx **20%** of all snakebites are dry bites, except in sea snake bites, where the incidence is **80%**. Causes - (i) Layers of clothing or shoes- over the bitten part (ii) When a snake bites but does not inject venom (iii) Superficial bite - Discharge orifice of a viper is usually well above its tip. Thus if the fang has not penetrated deep enough, most or part of the venom may be ejected superficially or externally without entering the wound. (iv) sideswipes - rather than head-on bites. Dry bites occur from other venomous animals also eg spiders such as tarantulas.

B. Signs and Symptoms

Most snakebites are from non-venomous snakes, simply because they outnumber venomous snakes almost ten times [please see above under classification].

Venomous snakebites

Ophitoxemia (Gk. ophis, serpent; toxon, poisoning) is poisoning by snake venom. In more than 50% of cases, inadequate venom is injected, producing mild symptoms. **Signs and symptoms depend** upon

(1) **Snake dependent factors:**

(i) Species and size of snake

(ii) Condition of its fangs and venom glands

(iii) Pathogens present in the snake venom

(iv) **Nature of bite** – Location, number and depth of bite. 98% of bites occur over the extremities

(v) Length of time the snake holds on

(vi) Amount of venom injected

(vii) Extent of anger or fear of snake [motivation of snake]

(2) **Victim dependent factors** - (i) Age and size of victim (ii) Victim's sensitivity to the venom

(3) **Community dependent factors** - First aid and medical care immediately given.

a. General [seen with all venomous snakes]

(1) **Fright** – Most common, especially the fear of rapid and unpleasant death. Following features are seen [within a few minutes of bite] due to fright (i) Hypotension (ii) Pulse feeble (iii) RR- (iv) Semiconsciousness with cold clammy skin.

(2) **Gas gangrene**

(3) **Tetanus**

(4) **Psychological shock** and death [due to fright]

(5) **Fang marks:**

(i) Two in number – (a) most common. (b) Distance between the two puncture wounds varies from 8 mm to 4 cm depending on species (c) Occasionally other teeth marks are also visible (d) Lower jaw does not leave any fang marks [**Fig 38.5**]

(ii) One – (a) Less common. (b) Seen in sideswipes

(iii) More than two – (a) less common. (b) Seen in (I) multiple bites (II) Single bites by snakes having more than 1 set of fangs

(iv) Depth - 1-8 mm, depending of species and strength of bite. Vipers generally cause deeper bites than Elapids, because they have longer teeth.

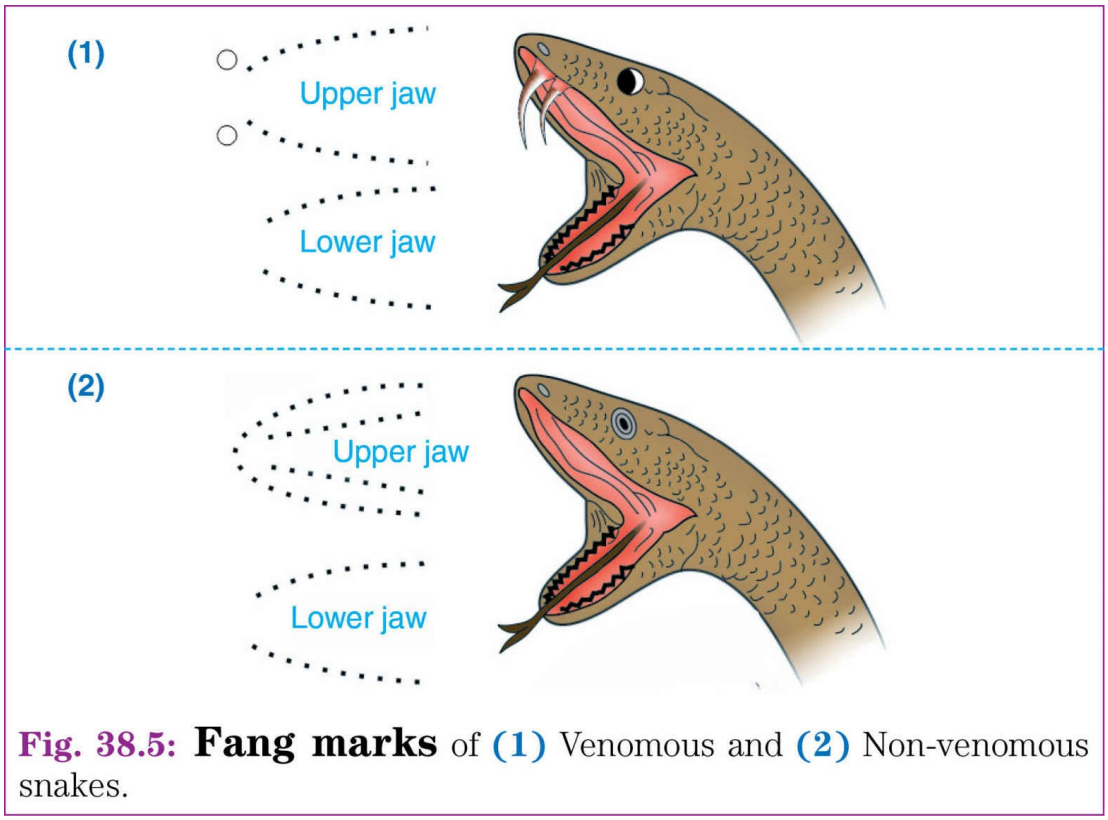


Fig. 38.5: Fang marks of **(1)** Venomous and **(2)** Non-venomous snakes.

b. Specific

i. Cobra

(1) **Local** – manifestations start within 6-8 min. Local manifestations in elapidae [cobra, krait] are minimal in sharp contrast to viperidae [Russell's viper, saw scaled viper] in which they are quite marked.

(i) A small reddish wheal or bullae - develops at the site of bite

(ii) Tenderness – of bitten area with slight radiating burning pain and oozing of bloodstained fluid

(iii) Swelling – minimal or even absent

(2) **Systemic** – Symptoms appear after 30 min

(i) Nausea, vomiting – early symptoms

(ii) After ½ -1 h"Excessive salivation, headache, vertigo, paresthesia around the mouth, myalgia, irritability

(iii) CNS depression- (a) Patient feels sleepy (b) slightly intoxicated, (c) weakness of legs, (d) reluctant to stand or move.

(iv) Paralyzing effects – (a) Paralysis is first detectable as ptosis and external ophthalmoplegia, in that ocular muscles are most sensitive to neuromuscular blockade. Other ocular symptoms [due to involvement of oculomotor nv] are - blurring of vision, diplopia, dysconjugate gaze, strabismus (b) later, paralysis of lower limbs, trunk, neck and head [head falls forward, inability to raise head], palate, vocal cords [inability to speak (dysphonia)], jaws, tongue, muscles of deglutition [dysphagia], absent gag reflex. (c) After 2h"complete paralysis

(v) Respiratory arrest – Causes (a) paralysis of intercostal muscles and diaphragm (b) obstruction of upper airway by the paralyzed tongue (c) inhaled vomitus

(vi) Stoppage of heart

(vii) Convulsions, coma.

(viii) Cause of death - respiratory failure [causing hypoxia and acidosis]

(ix) If recovery occurs – skin and cellular tissues surrounding the bite mark undergo necrosis.

Memory Aid 1: Signs and symptoms of cobra bite

COBRA causes **PARALYSIS**

Convulsions, **C**oma [towards the end]

Ophthalmoplegia, **O**cular symptoms [blurring, diplopia, dysconjugate gaze, strabismus]

Bulla at the **B**ite site

Respiratory arrest

Airways obstructed by paralyzed tongue

Paralysis, **P**tosis, **P**aresthesia around the mouth,

Area tender [bitten area]

ve**R**tigo

Absence of swelling at bitten site [in contrast to viper]

Lameness [inability to walk due to weakness of legs]

m**Y**algia,

Salivation

Irritability

Stoppage of heart [towards the end]

ii. Krait

(1) Time of bite - Most patients are bitten while asleep on the floor of their huts.
(2) Site - Because people are bitten while lying down, the target may be any part of the body, including the head, neck, and shoulders.
(3) Local –
(i) local signs are minimal or insignificant.
(ii) invisible or scarcely perceptible puncture marks
(iii) mild tenderness, itching, numbness, paresthesia
(iv) negligible or no local swelling.
(v) Painful enlargement of local lymph nodes [rare].
(4) General –
(i) Abdominal pain [very common]
(ii) Fasciculations
(iii) Feeling of drowsiness and intoxication
(iv) Paralysis - develops within 2 hours or be delayed for 12 hours.
(v) Sometimes severe generalized muscle pains with moderately elevated plasma myoglobin concentrations [due to generalized rhabdomyolysis].
(vi) Urine – shows albumin.
(5) Fatality - in the absence of antivenom treatment and assisted ventilation >

75%.

iii. Viper

(1) **Local:**

(i) Swelling - around bite. Quickly spreads around to involve entire limb and even adjacent trunk. Followed by malignant edema of the entire limb

(ii) Pain, paresthesias, reddening, tenderness

(iii) Peristent bleeding - from bite site

(iv) Regional lymphadenopathy

(v) Bruising – over (a) path of superficial lymphatics (b) lymph nodes

(vi) Blisters – (a) in and around bite site start appearing within 12 hours. (b) Spread around to involve entire limb. (c) Contain either clear or blood stained fluid. If patient survives, they heal in about 4 weeks.

(vii) Extensive necrosis - of skin, s/c tissues, muscles in 10-15% cases. Followed by extensive suppuration, sloughing

(viii) -ed intracompartmental pressure – due to edema. In tight fascial compartments [eg anterior tibial compartment] causes (a) -ed pain on stretching intracompartmental muscles (b) s/c anesthesia (c) Severe pain and (d) tense swelling

(2) **Systemic:**

(i) Hematologic abnormalities – most characteristic of viper bites.

(ii) Pathology - (a) DIC"fibrin is used up" defibrination"Blood becomes incoagulable. (b) Primary pathological fibrinolysis [PPF] (c) widespread microvascular fibrin deposition [microthrombi] (d) Blood – shows (I) early hemoconcentration followed by (II) .RBC and (III) .platelets (IV) -bleeding time (V) -clotting time (e) Urine – contains blood, protein, sugar

(iii) Main hemorrhagic manifestations - (a) Bleeding in (I) Anterior pituitary (II) floor of the mouth (III) Genitourinary tract (IV) GIT and (V) tympanic membrane (b) Ecchymosis and petechial hemorrhages over entire body (c) Epistaxis (d) Gingival bleeding (e) Hematuria (f) Hemoptysis (g) Intracranial hemorrhages [SAH presenting as severe headache, intracerebral hemorrhage presenting as hemiplegia, unconsciousness and convulsions] (h) Intravascular hemolysis [resulting in hemoglobinuria, acute renal failure] (i) rectal bleeding (j) Retroperitoneal and intraperitoneal hemorrhages [presenting as abdominal distension, peritonism, tenderness]

(k) Subconjunctival hemorrhages

(iv) General – headache, dizziness, weakness

(v) CVS - (a) Hypotension (b) tachycardia (c) Hemorrhagic shock

(vi) Temp-

(vii) Pupils - dilated, insensitive to light, blurring of vision

(viii) Respiratory depression

(ix) Scalp, mouth, tongue – tingling and numbness

(x) Paralysis is not seen – this differentiates viper bite from elapid bites [**Table 3**]

(xi) Abortions - in pregnant females

(xii) Cause of death – hemorrhagic shock.

Table 3: Differences between a elapid and a viperine bite

S.No	Feature	Elapid bite	Viperine bite
1.	Onset of signs and symptoms	Within 10-20 min	Within few seconds to 10 min
2.	Local reaction	Minimal	Extensive
3.	Gangrene	Early wet type	Slower onset, dry type
4.	Feeling of intoxication	Marked	Not marked
5.	Gait	Staggering	Not so.
6.	Speech and deglutition	Lost	Not so
7.	Tongue and larynx	Paralyzed	Not involved.
8.	Paralysis	Seen	Not seen
9.	Saliva	Hypersalivation with trickling of saliva from mouth	Salivation absent
10.	Pupils	Normal	Dilated. Do not react to light
11.	Respiration	Slow, weak, labored	Quick and labored
12.	Blood pressure	Normal	Hypotension
13.	Pulse	Initially normal, later irregular	Weak, irregular
14.	Coagulability of blood	Not affected	Greatly affected
15.	Hemorrhagic manifestations	Absent	Prominent feature
16.	Death	From respiratory paralysis	Hemorrhage, circulatory failure

iv. Sea snake

(1) **Local reaction** -

(i) Little or none.

(ii) The bite is usually painless and may not be noticed by the swimmer.
(iii) Teeth may be left in the wound.
(iv) Minimal or no local swelling, and involvement of local lymph nodes is unusual.
(2) **After ½ - 1 h** –
(i) Headache, generalized pain
(ii) Generalized aching, stiffness and tenderness of the muscles,
(iii) Trismus is common
(iv) A thick feeling of the tongue, thirst, sweating and vomiting.
(v) Generalized rhabdomyolysis - Myoglobin and K^+ are released from damaged skeletal muscles. Hyperkalemia develops within 6–12 hours
(vi) Weakness of muscles [persists for several months]. Passive stretching of the muscles is painful.
(vii) Marked polymyositis with a "limb-girdle" distribution [muscles of shoulders and hips]
(3) **Later** – progressive flaccid paralysis, beginning with ptosis [as in elapids]. Patient remains conscious until the respiratory muscles are sufficiently affected to cause respiratory failure.
(4) **Renal failure** [due to -myoglobin and -K^+]
(5) **Serum and plasma** - appear brownish
(6) **Lab tests** -
(i) Muscle enzymes-
(ii) Plasma K^+-
(iii) Myoglobinaemia and myoglobinuria [develop 3–8 h after the bite]
(iv) **Urine** - dark reddish brown [Coca-Cola colored]. Tests for Hb in urine are +ve [due to presence of myoglobin]
(7) **Death** – due to respiratory paralysis or cardiac arrest [due to -K^+].

C. Diagnosis

(1) If snake has been killed and brought, it is easy to identify [**Table 1**, **Table 2**].
(2) **Snake venom components** – as mentioned above may be detectable
(i) Can be detected in aspirates, biopsies, CSF, serum, body fluids, urine and wound swabs
(ii) Collect and send for analysis (a) Skin and underlying tissues surrounding

fang marks [in PM only] (b) Wound and blister aspirates (c) Serum (d) Urine

(3) **Radioimmunoassay (RIA)** – Most sensitive and specific test [detection level 0.4µg/L]. Snake venom components like cholinesterase and thromboplastin can be detected

(4) **Enzyme immunoassay (EIA)** - Detection level 5µg/L. Simpler. More widely used.

(5) **Immunological detection** - by ELISA

(6) **Urine** – Venom is detectable, even when the patient is treated with antivenin

(7) **Animal testing** – swab from fang marks or an extract from skin injected into a small animal [eg a frog] and see for toxicity.

D. Management

1. Field management [first aid]

(1) **Reassure** the victim.

(2) **Do not tamper** with the bite wound in any way.

(3) **Immobilize** the bitten limb using a splint or sling.

(4) Avoid elevation of an extremity – as it may hasten absorption.

(5) During transport to hospital, whole patient should be immobilized, especially the bitten limb, because any muscular contractions will promote spread of venom.

(6) Avoid harmful and time-wasting treatments.

(7) Because species diagnosis is critically important, the snake should be taken along to the hospital if it has already been killed. However, if the snake is still at large, do not risk further bites and waste time by searching for it.

(8) **Sutherland wrap** [**syn** - Pressure Immobilization, The splinting and crepe bandaging method]:

(i) Developed and advocated by **Struan Sutherland** in Australia.

(ii) **Procedure** [**Fig 38.6**] - An elasticated, stretchy, crepe bandage, app 10 cm wide and 4.5 meters long is used. If that is not available, any long strips of material can be used. The bandage is bound firmly around the entire bitten limb, starting distally around the fingers or toes and moving proximally, to include a rigid splint. The bandage is bound as tightly as for a sprained ankle, but not so tightly that the peripheral pulse (radial, posterior tibial,

dorsalis pedis) is occluded or that a finger cannot easily be slipped between its layers. When applied in this manner, it exerts a pressure of about **55mmHg** [ideal in snakebite; for reasons please see below]. The bandages should not be released until the patient is under medical care in hospital, and antivenom treatment has been started.

(iii) **Recommended** - in bites by cobras, king cobras, kraits or sea snakes [localizes venom, slows down absorption].

(iv) **Not recommended** - in viper bite, because its venom has locally-necrotic effects. Pressure-immobilization localizes the venom, -ing the locally-necrotic effects.

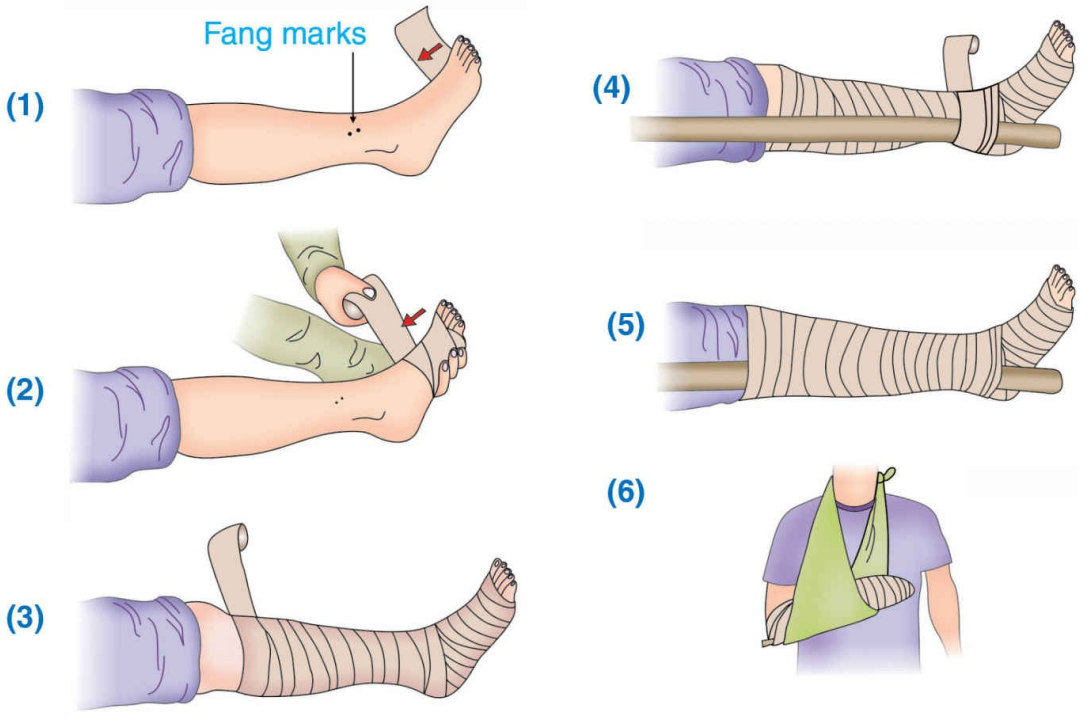


Fig. 38.6: Pressure-immobilization technique (**Sutherland wrap**). **(1-5)** Application on lower limb; **(6)** Same technique applied on upper limb.

2. Management in the hospital

a. Admit in ICU

(1) **Monitor closely** – cardiac rhythm, oxygen saturation, urine output, vital signs.

(2) Obtain history

(3) **Evaluate progression of envenomation objectively**

(i) Mark level of swelling in the bitten extremity

(ii) Measure limb circumferences every 15 min until swelling has stabilized.

(iii) During this period of observation and monitoring, the extremity should be positioned at approximately heart level.

(4) **Establish large-bore IV access** - in one or two unaffected extremities.

(5) **Remove measures applied in the field** [eg constriction bands or tourniquets etc] as soon as IV access has been obtained. Keep in mind that release of such ligatures may result in hypotension or dysrhythmias when stagnant acidotic blood is released to the central circulation.

(6) **Fluid resuscitation** -

(i) Isotonic saline (20–40 mL/kg IV) should be initiated if there is any evidence of hemodynamic instability

(ii) 5% albumin (10–20 mL/kg) - if patient fails to respond to saline.

(7) **Blood** - should be drawn for typing and cross-matching and for laboratory evaluation as soon as possible.

b. Polyvalent antisnake venom [PAV] therapy

(1) The key to management of venomous snakebite is the administration of specific antivenom or PAV. However it should never be started as a matter of routine, because of it being potentially hazardous.

(2) **PAV** - Prepared in India by (a) Bharat Serums & Vaccines Ltd, Mumbai (b) Central Research Institute, Kasauli (c) Haffkine Biopharmaceutical Co, Mumbai (d) Kings Institute of Preventive Medicine, Chennai (e) Serum Institute of India Ltd, Pune (f) VINS Bioproducts Ltd, AP.

(3) **Method of preparation of PAV:**

(i) By hyperimmunizing horses [or sheep] against the venom of 4 common poisonous snakes (a) Cobra (b) Common Krait (c) Russell's Viper (d) Saw Scaled Viper

(ii) Plasma obtained from them is concentrated and purified

(iii) Serum is lyophilized and sealed in vials

(iv) Lyophilization - (a) It is a dehydration process used to preserve sera. (b) **Process** – Sera frozen"High vacuum applied"Frozen water sublimates"Sera reduced to powder with no water content"microorganisms and enzymes

cannot degrade sera"Can be stored for 5 y.

(4) **When sera is needed -** it is rehydrated (reconstituted) by adding 10 ml of water per vial]. Half life of reconstituted serum is 90 h. If the reconstituted serum is opaque or turbid, it should not be administered [indicates precipitation of proteins"loss of activity, -ed risk of adverse reactions.

(5) **Cost of one vial** - `350-500

(6) **Production** - 5 lakh vials/y].

(7) Useful when given within 4 h of bite; lesser value after 8 h and of doubtful value after 24 h.

(8) Efficacy [**Table 4**].

(9) **Indications for antivenom administration:**

(i) Viperid bites - (a) any evidence of systemic envenomation [systemic symptoms or signs, lab abnormalities] (b) significant progressive local findings [e.g., soft tissue swelling crossing a joint or involving more than half the bitten limb].

(ii) Elapid bites - first sign of any evidence of neurotoxicity [cranial nerve dysfunction (e.g., ptosis) or peripheral neuropathy].

(10) **Best time of administration** – within 4 h. Less value after 8 h, and of doubtful value >24 h.

(11) **Hypersensitivity testing** -

(i) By s/c or intradermal inj or intraconjunctival instillation of diluted antivenom was once tried.

(ii) Not recommended now because (a) delay the start of antivenom treatment (b) risky, because may presensitize patient to PAV (c) are poor predictors of early anaphylactoid reactions (d) In serious Envenomations, PAV therapy has to be administered anyway

(12) **Complications:**

(i) Allergic reactions

(ii) Blindness

(iii) Serum sickness [fever, chills, urticaria, myalgias, arthralgias, and possibly renal or neurologic dysfunction] may develop 1–2 weeks after antivenom administration. Treat with systemic glucocorticoids [eg, oral prednisone, 1–2 mg/kg daily] until all findings resolve; the dose is then tapered over 1–2 weeks. Also oral antihistamines and analgesics.

Table 4: Efficacy of PAV

Venom	Each vial of PAV [10 ml] neutralizes following amount
Cobra	6 mg
Russell's Viper	6 mg
Common-Krait	4.5 mg
Saw scaled Viper	4.5 mg

Fig. 38.7: **(1)** Polyvalent snake venom available in India. **(2)** in Africa

c. Other treatments

(1) **Vasopressors** (eg. dopamine) – Given only after aggressive volume resuscitation and antivenom administration. Invasive hemodynamic monitoring [central venous and/or pulmonary arterial pressures] may be helpful, although obtaining access is risky if coagulopathy has developed.

(2) **Acetylcholinesterase Inhibitors** – useful in Neurotoxic Snake Envenomation.

(i) Patients with clear, objective evidence of neurotoxicity after snakebite (e.g., ptosis or inability to maintain upward gaze) should receive a trial of **edrophonium** or **neostigmine**. (a) Pretreat with **atropine**: 0.6 mg IV [children, 0.02 mg/kg; minimum of 0.1 mg] (b) Follow with Edrophonium: 10 mg IV (children, 0.25 mg/kg) or Neostigmine: 1.5–2.0 mg IM (children, 0.025–0.08 mg/kg)

(ii) If objective improvement is evident at 5 min, continue neostigmine at a dose

of 0.5 mg (children, 0.01 mg/kg) IV or SC every 30 min as needed, with continued administration of atropine by continuous infusion of 0.6 mg over 8 h (children, 0.02 mg/kg over 8 h).

(iii) Maintain vigilance regarding aspiration risk and secure the airway with endotracheal intubation as needed.

d. Wound care

Application of a dry, sterile dressing and splinting of the extremity with padding between the digits.

e. General measures

(1) Tetanus immunization

(2) **Prophylactic antibiotics** (e.g. cephalosporins) – especially if misguided first aid efforts have included incisions or mouth suction.

(3) **Pain control** - with acetaminophen or narcotic analgesics. Salicylates and nonsteroidal anti-inflammatory agents should be avoided because of their effects on blood clotting.

(4) **Muscle-compartment syndrome:**

(i) Most snake envenomations involve s/c deposition of venom.

(ii) Sometimes however, venom can be injected more deeply into muscle compartments, particularly if the offending snake was large and the bite occurred to the lower leg, forearm or hand.

(iii) If swelling in the bitten extremity raises concern that subfascial muscle edema may be impeding tissue perfusion (muscle-compartment syndrome), intracompartmental pressures (ICPs) should be checked by any minimally invasive technique [eg wick catheter or ICP monitor].

(iv) If any ICP is high (>30–40 mmHg), the extremity should be kept elevated above heart level while further antivenom is given.

(v) IV mannitol (1 g/kg) - to . muscle edema if the patient's hemodynamic status is stable. (vi) Fasciotomy - If after 1 h of IV mannitol, the ICP remains elevated.

3. Treatments on which opinion is divided

Application of tourniquet

(1) **Conflicting evidence** - There is conflicting evidence regarding the use of tourniquets. Some workers believe that there is no convincing evidence of its efficacy. But proponents think it could be because in most cases, tourniquet is not properly applied. They assert that positive results have been seen with properly applied tourniquets.

(2) **Technique:**

(i) Correct technique - Application of **55mm Hg pressure and no more**, since it would only block lymphatics [and some superficial veins], but not arteries and deep veins [BP is 120/80 mm Hg]. Venom spreads mainly through lymphatics and superficial veins. The best way to do this is to use a sphygmomanometer cuff and inflate it to 55mm Hg.

(ii) Earlier recommendations to ensure the right pressure were (a) tighten until the pulse can just be felt (b) it should be possible to force a finger between the tourniquet and the limb (c) if the part becomes bluish or cold, the tourniquet is too tight

(iii) Multiple tourniquets were sometimes recommended

(iv) The use of a **crepe bandage** is generally effective if sphygmomanometer is not available.

E. Fatal Dose

Please see [**Table 5**].

Table 5: Fatal dose and total yield of venom (in one bite) in various snakes

Snake	Fatal dose in terms of dried venom	Total yield in one bite in terms of dried venom [bracket denote the yield in number of fatal doses]
Krait	6 mg	20 mg [x3]
Saw scaled viper	8 mg	25 mg [x3]
Cobra	15 mg	200-350 mg [x20]
Russell's viper	20 mg	150-200 mg [x10]

Memory Aid 2: Fatal doses of venoms from lowest to highest

Kill **S**nakes by **C**utting **R**apidly [or **R**uthlessly]

F. Fatal Period

(1) Common Cobra – 8 hrs
(2) Common Krait– 18 hrs
(3) Russell's Viper – 3 days
(4) Saw scaled Viper – 5 days.

G. Cause of Death

(1) Cobra - Respiratory paralysis.
(2) Viper – Hemolysis, Hemorrhage.

H. PM Appearances

1. Common to all poisonous snakes

(1) **Fang marks** – 1, 2 or several depending on the type of bite
(2) **Washings from the site of the bite and tissues underneath** – show venom

components [please see “Diagnosis” above for details].
(3) **Internal organs** – congested.

2. Elapids

(1) **Local changes** – minimal (2) **Brain** - congested.

3. Vipers

(1) **Local changes** - extensive local cellulitis, discoloration and swelling.
(2) **Hemorrhages** – Prominent. Occur from puncture, mucus membranes, into bowel, lungs, and almost all other tissues; Purpuric spots on pericardium
(3) **Kidneys** – inflamed, hemorrhagic.

4. Sea snakes

(1) Signs of rhabdomyolysis
(2) **Kidneys** – congested, tubules blocked with myoglobin.

I. ML Importance

(1) **Manner of death** –
(i) Accidental - most common
(ii) Homicidal - (a) throwing snake on the bed of a sleeping person (b) Infanticide – may be committed in this way
(iii) Suicidal - very rare. **Queen Cleopatra** committed suicide by snake bite.
(2) **Cattle poison** – Procedure - A cobra is shut up in an earthen vessel containing a banana"Heat is applied to the vessel"Snake is irritated"Bites fruit"Venom is injected into banana pulp"Pulp taken out"Smeared on a rag"Rag thrust into the animal’s rectum using a split bamboo [cf **ch 37**"calotropis; ch 43"Nerium oleander for similar uses].
(3) **Excretion** - occurs through milk, saliva, urine and mucus surfaces. There are cases on record where a young child died after suckling mother’s breast who was bitten by a poisonous snake.
(4) **Ingested venom** - Snake venom if ingested is not poisonous, as the venom proteins are digested.
(i) Animals killed by snake venom may be eaten without ill effects
(ii) A rescuer who sucks snake venom from wound may not be poisoned if he swallows poison. However if he has minor abrasions in mouth, the poison

may be absorbed from there and secondary poisoning may occur in the rescuer.

III. SCORPION

Scorpions are predatory arthropods of class Arachnida.

A. Mode of Action

Scorpion venom opens neuronal sodium channels [**Memory Aid 3**]**. This causes (i) repetitive and spontaneous depolarization of** both **s**ympathetic and **p**arasympathetic nervous system, causing an autonomic storm **[pl co-relate with signs and symptoms].**

Memory Aid 4: MOA of scorpion venom

Sco **r p**ion venom causes – **r**epetitive and **s**pontaneous **s**ympathetic and **p**arasympathetic depolarization

Typically the parasympathetic effects are transient***, but sympathetic are more*** prolonged**. (ii) "catecholamine release from adrenal gland (iii) catecholamine induced cardiac hypoxia (iv)" renin secretion at the juxtaglomerular apparatus. (iv) Hyperkalemia, hyperglycemia, "insulin secretion, "aldosterone.**

B. Signs and Symptoms

Most stings occur on extremities. There are significant differences in signs and symptoms produced by different species. Stings from M. tamulus [Indian red scorpion] produce following symptoms.

(1) **Local** - (i) edema (ii) erythema (iii) pain [severe, excruciating] (iv) regional lymphadenopathy. Last 1-2 h.

(2) **Systemic** - (i) Nausea, vomiting (ii) extreme restlessness (iii) Allergic reactions, including anaphylaxis (iv) Cyanosis (v) Pupils - dilated (vi) Fever (vii) Paralysis (viii) Respiratory depression

(3) **Sympathetic** - (i) cardiogenic shock (ii) BP- [BP. if parasympathetic predominates] (iii) MI (iv) myocarditis (v) pulmonary edema (vi) arrhythmias, tachycardia

(4) **Parasympathetic** - (i) priapism (ii) sweating (iii) vomiting

(5) **Other** - (i) coagulopathy (ii) pancreatitis (iii) stroke

(6) **Rare** - (i) Intracerebral hemorrhage [Mechanism - venom induced autonomic storm"hypertension, vasculitis].

(7) **Death** – if occurs is preceded by convulsions and coma. Mortality except in children is negligible.

Memory Aid 5: Signs and symptoms of scorpion stings

All S C O R P I O N S cause MI, but fe w dilate pupils

Allergy

Salivation, sweating

Cyanosis, cardiogenic shock, coagulopathy,

Oedema

Regional lymphadenopathy, restlessness, respiratory depression

Pain, paralysis, pulmonary edema, priapism, pancreatitis

Inspirations slow [respiratory depression]

St **O** rm [autonomic]

Nausea & Vomiting

Stroke

Convulsions and coma precede death

MI , Myocarditis

fever

dilated pupils

Fig. 38.8: Priapism in a child in scorpion bite.

C. Fatal Period

Few hours.

D. Diagnosis

ECG – (i) Peaked T waves in leads V_2-V_6. (ii) Q wave, ST segment elevation in leads I and aVL (iii) Left anterior hemiblock.

E. Differential Diagnosis

Snake bite – Snake bite shows two punctures; scorpion stings shows one. Confirmation by ELISA.

F. Management

(1) **First aid -** (i) Immobilize limb (ii) Tourniquet – Technique and pressure as in snakebite (iii) Local washing – with weak soln of ammonia, borax or $KMnO_4$ has doubtful value (iv) Tetanus toxoid (v) local wound care (vi) topical antibiotic to the wound.

(2) **Supportive care** – Pain control - (a) ice packs (b) local anesthetic injection

(c) oral analgesics - (I) Paracetamol (II) Barbiturates control both pain and convulsions.

Memory Aid 6: Main drugs in scorpion stings

SCOR **P** IO**N** **D**IES **A**T **F**OOT - **P**razosin, **N**ifedipine, **D**ehydroemetine, **D**opamine, **A**protinin, **F**urosemide

G. Cause of Death

(1) Cardiac failure (2) pulmonary edema.

H. PM Appearances

(1) **Local** - (i) Stung area is usually a limb. (ii) reddened and edematous. (iii) Shows one hole in the center. (iv) Skin, s/c and muscle around the hole should be excised and sent for chemical analysis

(2) **Viscera** – congested.

IV. SPANISH FLY (CANTHARIDES)

Spanish fly (Lytta vesicatoria, Cantharis vesicatoria) is an emerald-green beetle (**Fig 38.9**) in the family Meloidae.

Salient features:

(1) **Size - 2x0.5 cm**.

(2) **Active principle** - Cantharidin [5%], a strong irritant.

Fig. 38.9: Spanish Fly

A. Mechanism of Action

Cantharidin, an anhydride of **cantharidic acid**, is almost insoluble in water but readily soluble in acetone (1:40). Its chemical formula is $\mathbf{C_{10}H_{12}O_4}$. It is a potent inhibitor of protein phosphatases types 1 and 2A.

B. Signs and Symptoms

1. Applied Externally to the skin

(1) Redness (2) burning pain (3) blisters

2. Ingested

Symptoms begin within 2–4 hours of ingestion.

(1) **Burning** in the mouth, severe thirst

(2) **dysphagia, dysarthria** (difficulty in speech)

(3) **blistering** of the lips and tongue

(4) **Upper GIT symptoms** – nausea, vomiting, hematemesis, retrosternal burning [due to irritant effects on the esophageal and gastric mucosa]

(5) **Lower GIT symptoms** - intestinal colic, bloody diarrhea, tenesmus (if

poison passes into the lower GIT in large amounts)

(6) **Renal symptoms -** severe costovertebral angle pain, dysuria, scanty urine though there is an - desire to pass urine [strangury], hematuria

(7) **Terminal events** are **priapism** (painful erection of penis), **abortion** (in pregnant women), **convulsions**, **coma,** peripheral vascular collapse and death. Because it causes **priapism**, cantharides gained reputation as an aphrodisiac. But the erection of penis is pathological, painful and devoid of any sexual pleasure.

Priapism

Priapism is due to the engorgement of the dorsal corpora cavernosa, resulting in dorsal penile erection and ventral penile flaccidity.

Salient features:

Etiology - (i) Drugs [Anticoagulants, antihypertensives, Cantharides, drugs of abuse (e.g., alcohol, cocaine, marijuana), phenothiazines, sedative-hypnotics, SSRI] (ii) Immunosuppressive disorders (iii) Intracavernosal injections [papaverine, phentolamine, and prostaglandin-1] (iv) Leukemia (v) Sickle cell disease (vi) Spinal trauma.

C. Fatal Dose

10 to 80 mg.

D. Fatal Period

24-36 hours.

E. Management

(1) **Control of pain** – pethidine

(2) **control of emesis**

(3) **Stomach tube** - A soft No. 14 Levine tube passed with caution. **Stomach wash** done and tube retained. The dysphagia may be so marked that oral feedings will have to be given through it for 7 to 10 days. **Dilute milk** (50% water and 50% milk) given (**demulcent** as well as a nutritional)

(4) **vasopressor agents** – eg norepinephrine for peripheral vascular collapse

(5) Replacement of fluids.

F. Cause of Death

Renal failure.

G. PM Appearances

(1) **Mouth** – Shows inflammation and vesication

(2) **Mucosa of esophagus, stomach** and **intestines** – congested, swollen, show ulcerations

(3) **Shiny green particles** – found sticking to the stomach mucosa

(4) **trachea, bronchi** – contain blood stained mucus

(5) **Lungs** – edematous

(6) **Kidneys, ureters, bladder, urethra** – Show inflammation

(7) **Heart** – surface of the heart and endocardium shows hemorrhages.

H. ML Importance

(1) **Accidental poisoning** -

(i) Cantharidin is a **rubefacient** (produces redness of the skin by causing dilation of the capillaries) and **vesicant**.

(ii) As a **counterirritant** it was once employed in the treatment of pleurisy, pneumonia, arthritis, neuralgias, rheumatism and various dermatological conditions (warts and molluscum contagiosum) but has been abandoned because of its excessively toxic effects.

(iii) May cause accidental poisoning, if used therapeutically

(2) **Aphrodisiac** – Frequently taken as aphrodisiac or given to partners for causing sexual excitement. **Case study** - On Apr 27, 1954 **Arthur Ford** of Britain, who was infatuated with two female colleagues gave Spanish fly to them in coconut candies. Both died and autopsy revealed Spanish fly in their stomach. Ford was convicted of manslaughter and sentenced to **5 y** in prison.

(3) **Criminal abortion**

(4) **homicide** [rarely].

39. Food Poisoning and Poisonous

Foods

I. POISONING DUE TO BIOLOGICAL CONTAMINANTS

A. Bacterial Contamination (Bacterial Food Poisoning)

1. Infection type

In ***infection type*** of food poisoning [syn, ***long incubation type***, because incubation period is long], the bacteria multiply within the body and cause typical symptoms like diarrhoea and vomiting.

2. Toxic type

In the ***toxic type*** of food poisoning [syn, ***short incubation type***, because incubation period is short], bacteria have already produced toxins in the food, and the patient ingests preformed toxins.

3. Botulism

The third type of food poisoning is **botulism**. Though it is caused by preformed bacterial exotoxins produced by ***C. botulinum***, it is not clubbed with toxic type of food poisoning, because unlike it, its major feature is not diarrhoea and vomiting but **muscle paralysis**.

Salient features:

(1) **Most dangerous** type of food poisoning.

(2) Clostridium botulinum is an **anaerobic**, **gram-positive spore-forming** rod.

(3) **Seven** immunologically distinct neurotoxins (A to G) are produced which **block the release of acetylcholine** (ACh) from presynaptic fibres, causing **flaccid paralysis** [Botulinum toxin is used as a therapeutic agent for cervical torticollis, strabismus, and blepharospasm]. Foodborne botulism in humans is caused by **toxin of types A, B, and E**. **Type E** is associated with consumption of seafood products.

(4) **Nature of the toxin**– Botulinum toxin in solution is colorless, odorless, and tasteless. Botulinum toxin is denatured by heat at 80°C for 30 min. and 100°C for 10 min. Thus, foodborne botulism is always transmitted by foods that are

not heated thoroughly, before eating.

(5) **Symptoms and Signs:**

(i) Almost every type of food has been associated with outbreaks of botulism, but the most commonly implicated foods are vegetables, particularly "**low acid**" (ie, higher pH) vegetables such as beans, peppers, carrots, and corn.

(ii) Symptoms appear within 12-48 hours of ingestion

(iii) Initially **GI symptoms** predominate – nausea, vomiting, diarrhea, abdominal pain with distension

(iv) Later, **neurological** symptoms due to **cholinergic blockade** – Visual disturbances, dry mouth, dysarthria and dysphagia are 4 most common neurological symptoms

(v) Mortality has fallen from 60% to 5% now.

(6) **D/D** – Disease most commonly confused with botulism is **Guillain-Barré syndrome**.

(7) **Fatal dose of botulinum toxin - Most poisonous substance** known. Fatal dose - 1 ng/kg.

(8) **Treatment**–Symptomatic. Trivalent (A,B,E) antitoxin.

4. Prophylaxis (of bacterial food poisoning)

(1) Meat inspection at slaughter houses.

(2) Control of rodents.

(3) Regular check up and removal of food carriers from food industry.

(4) Food should be properly cooked.

5. Treatment (of bacterial food poisoning)

(1) **Glucose saline infusion**. Mainstay of therapy.

(2) **Antibiotics -** only in prolonged (>5 days), profuse (>6 stools/day) or bloody diarrhea, in fever (> 38.5^0C), or severe abdominal pain. **Of no use in toxic type**.

(3) **Anti-diarrheals** - loperamide, diphenoxylate, bismuth sub-salicylate, probiotics, or prebiotics are of **little value**.

6. PM appearances in bacterial food poisoning

(1) **Mucosa of stomach and intestines** - intensely congested and swollen. May be **ulcerated**.

(2) **Microscopic examination** – Liver shows **fatty degeneration**.
(3) **Culture** – **Causative organism** can be isolated from blood and viscera.

B. Fungal Contamination (Mycotoxicosis)

Mycotoxins are toxic secondary metabolites produced by fungi (molds, and yeasts).

Salient features:

(1) The term 'mycotoxin' is usually reserved for toxins produced by fungi that colonize crops.
(2) Mycotoxins are **heat stable** and survive cooking
(3) Major mycotoxins of interest are:

Ergot

Ergot is the dried ***sclerotium*** (a compact mass of hardened fungal mycelium containing food reserves) of fungi belonging to genus Claviceps (most prominent member C. purpurea) [**Fig 39.1**].

Salient features:

(1) The fungus grows on cereals like bajra, barley, oats, rye, wheat etc.
(2) It gradually replaces the grain, and forms a curved, black, compact mass about 1 cm long and 0.5 cm thick (resembles droppings of a rat).
(3) Has a peculiar odor and a disagreeable taste.
(4) Contains about 30 alkaloids. Most important are (i) ergotoxine (ii) ergotamine [a precursor of LSD] and (iii) ergometrine.
(5) If the sclerotia are not removed from contaminated grain by beating or sieving, humans may accidentally ingest them causing poisoning.

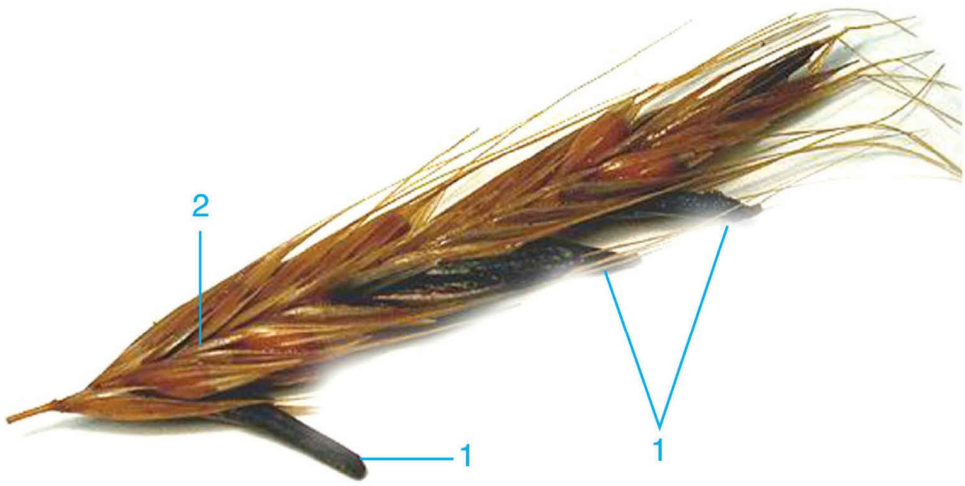


Fig. 39.1: Ergot (1) growing on rye (2). Note curved black mass, which is the dried sclerotium of ergot.

a. Mechanism of action

(1) Ergot alkaloids stimulate adrenergic receptors, both peripherally and centrally.

(2) Directly stimulate muscle fibres.

b. Acute poisoning

i. Signs and symptoms

(1) **GIT** - thirst, abdominal colic, vomiting, diarrhea.

(2) **Resp** - tightness in chest, difficulty in breathing.

(3) **CVS** - tachycardia; pulse - rapid, weak; BP-↑.

(4) **CNS** - confusion, giddiness, numbness and tingling in hands and feet, paresthesias, pupils dilated, dimness of vision.

(5) **Muscular system** - exhaustion, marked muscular weakness, cramps, convulsions, twitching in muscles.

(6) **Hematologic** - Bleeding from nose and other mucus surfaces, especially after a large dose.

(7) **Misc** - cold skin.

ii. Management

(1) Gastric lavage

(2) Activated charcoal
(3) Emetics [**ch 32**]
(4) cathartics
(5) **Nitroprusside** - for hypertension and severe ischemic changes
(6) **Benzodiazepines** - for convulsions [**ch 32**]
(7) **Vasodilators** - eg nitrites

c. Chronic poisoning

Due to repeated consumption of ergot alkaloids [infected rye (**Fig 39.1**) , ergoline-based drugs, migraine preparations containing **ergotamine tartrate** (Migril)]. Known as **ergotism** or **ergotoxicosis**.

i. Signs and symptoms

(1) **GIT** - nausea, vomiting, diarrhea, cramping abdominal pain
(2) **CNS** - headache, vertigo, ataxia, numbness and tingling of skin, paresthesias, sensation of **insects creeping under the skin [formication** - pl also see **ch 41**"cocaine bugs**]**, psychosis [hallucinations, madness], convulsions [**St. Vitus' dance**], coma
(3) **Vascular** - vasospasm, cold skin,[3] Intermittent claudication, Intense burning sensation and gangrenous extremities [**Holy Fire**, ***Ignis Sacer*, St. Anthony's Fire**], dry gangrene of the fingers, toes, ears, nose etc, thrombosis [rarely].

ii. Management

Same as for acute, except for stomach lavage, emetics, cathartics and activated charcoal.

d. Fatal dose

2-10 g.

e. Fatal period

one to several days.

f. PM appearances

Not characteristic
(1) **Internal organs** - congested

(2) **Blood vessels** - Degeneration of intima of smaller arterioles, thrombus formation.

g. MLI

(1) Ergot is used as an **abortifacient**

(2) Excessive and long use of **anti-migraine drugs** may produce ergotism

(3) Consumption of bread made of **contaminated rye** may cause ergotism. Seller of such grains may be prosecuted **U/s273, IPC** [Sale of noxious food or drink (**ch 31**)]

(4) Ergot administration during postpartum period (for reduction of blood loss) may be associated with **puerperal psychosis**.

II. POISONS OF VEGETABLE ORIGIN

A. Lathyrus Sativus [Grass pea, *Kesari dal*]

Lathyrus sativus is an annual leguminous crop [pulse] cultivated in India and several other geographical regions for animal and human consumption.

Salient features:

(1) Belongs to family Fabaceae [older name – Leguminosae], commonly known as the legume family, pea family, bean family or pulse family. Vicia faba also belongs to the same family.

(2) Popular in India, because (i) grows in conditions of extreme drought, famine and in poor soil (ii) high protein content of seeds (iii) resistant to pests. Seen as an 'insurance crop' as it produces reliable yields when all other crops fail.

(3) Staple food for low income groups in India

(4) **Toxic principles:** **β**-N-**o**xalyl-L-**α**, **β**-**di****a**mino-**p**ropionic acid [**β–ODAP**] also known as **β**-N-**o**xalyl-**a**mino-L-**a**lanine, [**BOAA**]. It is a glutamate analog present in seed cotyledons as free amino acid (1%).

(5) **Removal of toxin:** (i) Prolonged cooking .es the amount of **β**-ODAP to the less toxic isomer, **α**-ODAP. (ii) **β**-ODAP is water soluble; boiling grass peas for 1 hour followed by decanting the water removes about 70% of the ß-ODAP from the pea.

(6) **Toxic amounts** – If more than 30% of total diet contains kesari dal, and is taken for >6 m, spastic paralysis results.

1. Signs and symptoms

(1) Causes a form of spastic paralysis known as **neurolathyrism**.

(2) **Incidence** - in epidemic areas is 4%.

(3) **Epidemiology:**

(i) Men [especially young men between 10-20 y]are more susceptible than women.

(ii) Neurolathyrism is rare in well-nourished adults and postmenopausal women.

(4) **Onset** - 3 types

(i) Acute (ii) subacute (iii) insidious

(5) Main signs and symptoms are:

(i) **CNS** – (a) **Initially** - pain in the back and weakness of legs; difficulty in sitting down and getting up (b) **Later** – Gait is affected. -ed muscle tone in the legs [thigh extensors, thigh adductors, gastrocnemius] + muscle weakness"inversion of the feet"lurching, cross-legged gait [scissors gait]. Legs tremble and dragged along with difficulty. Patient may walk on tiptoes, and must use a stick to walk. (c) **Still later** – Complete spastic paraplegia (d) Evidence of peripheral neuropathy - does not usually occur unless the patient has severe neurolathyrism. (e) Superficial sensation, rectal sphincter tone, cranial nerve function, upper extremity muscle tone, and mental activity are usually normal. (f) There is no atrophy or loss of tone of muscles. There is no reaction of degeneration. (g) Knee jerks are -ed, ankle clonus is marked and Babinski’s sign is +ve.

(ii) Miscellaneous - GIT distress, urinary frequency and urgency, sexual dysfunction, neurasthenic symptoms [insomnia, thirst, short-term memory loss].

2. Prevention and treatment

(1) Exclusion of pulse from diet.

(2) If used, it must be parboiled.

(3) Massage and application of electricity.

3. PM appearances

Death is rare.

(1) **Macroscopic** - Lateral columns of spinal cord show sclerosis.

(2) **Histopathology** - Degeneration of the corticospinal pathways (eg, lateral

pyramidal tracts) distal to the mid-thoracic level with preservation of anterior horn cells in the spinal cord.

B. Poisonous Mushrooms

1. Signs and symptoms

Symptoms are collectively known as **phalloides syndrome**.

(1) **General:**

(i) Cyanosis

(ii) Pulse – slow, bradycardia

(iii) Respiration - labored

(2) **Irritant symptoms** – Delayed for 6-12 h.

(i) Salivation, nausea, vomiting, diarrhea

(ii) Throat – sense of constriction

(iii) Stomach - Burning pain

(iv) There may be acute pseudoobstruction and dilation of the colon in the absence of any mechanical obstruction [**Ogilvie's syndrome**].

(3) **Neurotic symptoms** – (i) Cramps (ii) Delirium (iii) Diplopia (iv) Giddiness (v) Headache (vi) Pupils – constricted (vii) sweating, (viii) Twitching of limbs (ix) coma, convulsions, collapse, death

(4) **Hepatic and renal toxicity** – develops in 3-6 days, in cases of survival.

Fig. 39.2: *Amanita muscaria.*

2. Fatal dose

One mushroom.

3. Fatal period

24 hours.

4. Test

Meixner test -

(1) Sample (stool, gastric lavage) + methanol" centrifuge"filter

(2) Add 2 drops of filtrate to a newspaper

(3) Encircle the spot with a pencil and dry it

(4) Add a drop of conc. HCl to the dry spot"**blue color**"Amatoxins present. Phallotoxins do not give blue color.

Memory Aid 1: Meixner test

Meixner test is done in **M**ushroom poisoning

5. Management

(1) Stomach wash – with $KMnO_4$.

(2) Activated charcoal

(3) Forced diuresis, Hemodialysis

(4) Benzyl penicillin 3-10 lakh units daily

(5) Atropine sulphate

(6) **Antidotes:**

(i) Anti-phalloidin antibodies

(ii) Cimetidine - Potent cytochrome P450 system inhibitor. Protects liver against alpha-amanitin. Dose 4-6 g/day.

(iii) Iridoid glycosides - These are a class of secondary metabolites found in a wide variety of plants and in some animals. Named after Iridomyrmex genus which contains a defensive chemical iridomyrmecin. (a) Aucubin - derived from the leaves of Aucuba japonica. Found to be protective against Amanita phalloides. (b) Kutkin - Kutkin is a mixture of iridoid glycosides picroside I and kutkoside isolated from the roots of Picrorhiza kurroa. Found to be protective against Amanita phalloides. Efficacy is superior even to silibinin.

(iv) N-acetylcysteine - Same dose as in paracetamol poisoning

(v) Silibinin - A semi-purified fraction of silymarin derived from milk thistle [Silybum marianum].

(vi) Thioctic acid - Once thought to be useful in hepatic damage. But now obsolete.

(7) Management of acute liver failure.

(8) Symptomatic

6. PM findings

(1) **GIT** - inflammation of mucosa.

(2) **Heart, kidneys** - Fatty degeneration. Kidneys may be pale and swollen.

(3) **Liver** - Shows most characteristic findings as mushrooms are hepatotoxic. Central zonal necrosis, centrilobular hemorrhage, fatty degeneration and steatosis. Findings of hepatic failure, eg anasarca, pulmonary edema, scleral

icterus and serous effusions are seen.

(4) **Brain** - Congestion, petechial hemorrhages in serous membranes and in substance of brain, especially if neurotic symptoms were present during life.

7. ML importance

Poisoning is usually accidental. Rarely homicidal.

C. Argemone Mexicana

Argemone mexicana (Kutila, Mexican poppy, Phirangi Datura, Pila-Dhatura, prickly poppy, Satyanasi, Sial Kanta, Ujar kanta) grows wild all over India in the cold season**.**

Salient features:

(1) It is an annual herb, belonging to the family Papaveraceae [Another poisonous plant of this family is P. somniferum. Please see **ch 40**], which grows wild all over India during winters [**Fig 39.3**]

(2) **Plant:**

(i) It grows 1-4 ft high, with spreading branches.

(ii) Leaves – sessile, spiny, thistle like

(iii) Flowers - 1-3 inch across and are colored yellow.

(iv) Capsules – Prickly oblong or elliptic 2-4 cm long. Contain seeds

(v) Seeds - (a) blackish brown, round and netted and resemble those of mustard [Brassica nigra] (b) Differences - (I) Argemone seeds have spiny edges. Mustard seeds do not have such spiny edges. (II) When seeds of A. mexicana are pressed on a slide, they burst with a report; mustard seeds collapse quietly.

(3) **Alkaloids** - All parts of plant are poisonous. The extract of the whole plant (latex) contains two alkaloids Berberine and Protopine.

(4) **Katkar oil:**

(i) A. mexicana seeds yield 22-36% of a pale yellow, nauseous, bitter, non-edible oil [Katkar oil or Argemone oil].

(ii) The oil is rich in two alkaloids sanguinarine and dihydrosanguinarine, the former being more toxic than the latter. They are mainly responsible for the poisonous symptoms (a) Mechanism of toxicity - sanguinarine inhibits Na^+-K^+-ATPase activity of heart"degenerative changes in cardiac muscle

(iii) Other alkaloids of lesser importance are cheletrythrine and coptisine.

(iv) If katkar oil is heated to 240°C for 15 minutes, it loses its toxic properties.

(v) Uses - (a) used medicinally in several countries in the treatment of dropsy, jaundice and skin diseases. (b) given as an aperient [laxative] (c) used for lighting lamps, as it is very cheap.

(vi) Abuses – used as an adulterant of mustard oil. Causes abnormal permeability of blood vessels resulting in **epidemic dropsy**.

Fig. 39.3: *Argemone mexicana.*

1. Signs and symptoms

(a) Local application

Application on scalp causes burning and fall of hair.

(b) Systemic

Consumption of adulterated oil causes **epidemic dropsy**. Symptoms appear gradually

(1) **General:**

(i) Marked edema of the legs

(ii) General anasarca [due to -ed BV permeability. Not attributable to CCF in most cases].

(iii) Pyrexia

(iv) Anemia

(2) **GIT** - (i) Loss of appetite, (ii) nausea, vomiting, dyspepsia, diarrhea

(3) **CVS** - (i) BP. (ii) pulse – feeble and rapid (iii) CCF

(4) **Liver** – enlarged, tender. Not attributable to CCF in most cases

(5) **Resp system** – Dyspnea

(6) **Skin and mucosa:**

(i) Bluish mottling (Erythrocyanosis) [due to dilatation of peripheral vessels]. Blanches on pressure"May develop into s/c telangiectasis or hemangiomata. As erythrocyanosis resolves, it leaves post inflammatory hyperpigmentation.

(ii) Small fleshy dark red warty growths may be noticeable on various body parts, and also on cheek, gums, nose and tongue

(iii) Tingling

(iv) Hyperesthesia

(7) **Neuromuscular** - (i) Calf muscles – tender; (ii) Tendon reflexes feeble or absent

(8) **Eyes** - Dimness of vision [in 10% cases]. Due to -IOP

(9) **Death** – CCF due to myocardial damage.

2. Lab

(1) -ESR

(2) **X-ray** – moderate to severe pulmonary congestion.

3. Treatment

(1) Good diet

(2) Supportive treatment especially of heart

4. PM findings

(1) Body shows generalized anasarca, or local edema of legs
(2) Heart - Myocardial damage, dilatation
(3) Liver - enlarged
(4) Skin – may show mottling
(5) Gastric contents show +ve tests for argemone oil.

III. POISONS OF ANIMAL ORIGIN

Poisonous animals and **venomous animals** are different. Former cause poisoning when eaten; the latter attack and inject venom in human victims. Please also see **ch 31** "introduction to poisons". In this chapter, only poisonous animals would be discussed. They are poisonous fish and mussel.

A. Poisonous Fish

(1) **Ichthyotoxicosis** [Gk. ichthyo, fish] is poisoning produced by poisonous fishes.
(2) **Ichthyotoxin** is a natural toxin produced by fish.
(3) **Ichthyosarcotoxic fishes** are those fishes that produce ichthyotoxicosis. They contain a poison in the flesh [Gk. sarx, flesh] including musculature, viscera, skin, or slime.
(4) **Ichthyotoxicology** is the study of natural toxins produced by fish, their cause, detection, and effects, and the treatment of conditions produced by them.
(5) **Ichthyotoxicity** is poisoning caused to fishes, eg by sessile animals such as sponges, coelenterates or tunicates which produce toxins to deter potential predator fish.

IV. MEDICOLEGAL ASPECTS OF FOOD POISONING

A. Information to Public Health Authorities

Doctor must inform all cases of food poisoning to public health authorities, as well as to police, because such cases are liable to be registered u/s **269, IPC**

(negligent act likely to spread infection dangerous to life), **272, IPC** (Adulteration of food or drink intended for sale) and **284,** IPC (negligent conduct with respect to poisonous substance).

40. CNS Depressants

I. INTRODUCTION

[A] ***CNS depressants*** are psychoactive drugs which temporarily diminish the function or activity of brain. Since they lower or "down" the functional activity of brain, they are sometimes referred to as "**downers**". In contrast, **stimulants** are referred to as "**uppers**" (increase the function or activity of brain). Depressants are widely used as prescription medicines and as **drugs of abuse**. [B] ***Hypnotic*** (***soporific***) drugs are psychoactive drugs whose primary function is to induce sleep. [C] **Narcotic** – (1) **Medical definition** – Same as hypnotic [from Gk. narke, numbness, stupor]. (2) **Legal definition** - Drug that is totally prohibited, or one that is used in violation of strict governmental regulation. Narcotic drug is defined in **S.*2***(xiv) of The Narcotic Drugs and Psychotropic Substances Act, 1985 [NDPSA 1985] as follows - "Narcotic drug" means coca leaf, cannabis (hemp), opium, poppy straw and includes all manufactured drugs. [D] ***Sedatives*** are psychoactive drugs that induce sedation by reducing irritability or excitement. A sedative drug in a high dose would act as a hypnotic.

II. CLASSIFICATION

CNS depressants may be classified as follows:

(1) General Anesthetics – Chloroform, Ether

(2) Alcohols (a) Ethyl alcohol (b) Methyl alcohol (c) Other alcohols (e.g. Isopropyl alcohol)

(3) Opium and its derivatives

(4) Sedative-hypnotics (a) Barbiturates

(i) Long acting
(ii) Short acting
(iii) Ultra-short acting (b) Hydrocarbons (e.g. Kerosene, Naphthalene, Turpentine) (c) Paraldehyde (d) Chloral Hydrate (e) Bromides - potassium bromide (f) Quinazolines – Methaqualone (g) Phenytoin sodium (h) Benzodiazepines – Diazepam, Fletazepam, Flurazepam, Lorazepam, Oxazepam, Prazepam, Chlordiazepoxide (i) Propanediol carbamates – Meprobamate (j) Piperdinediones – Glutethimide.

III. ALCOHOLS

A. Ethyl Alcohol(C_2H_5OH)

Ethyl alcohol [EA, ethanol, ETOH, grain alcohol, aqua vitae, water of life], is a transparent, volatile, flammable, colorless liquid, having a characteristic alcoholic odor and a burning taste.

Salient features:

(1) **Sp gr - 0.79**.
(2) **Caloric content - 7 Kcals/g** [29 kJ/g]
(3) It is a powerful psychoactive drug used in alcoholic beverages.
(4) **Frequency of use** – Alcohol is a socially acceptable drug. Among socially acceptable drugs, it is the 3nd most common drug of abuse after caffeine and nicotine [Please also see chapter on deliriant poisons].

1. Common forms of alcohol

(1) **Absolute alcohol** [syn, anhydrous alcohol, dehydrated alcohol] is 99.5% ethyl alcohol.
(2) **Rectified spirit** [syn, neutral spirit, rectified alcohol] is 95% ethyl alcohol.
(3) **Methylated spirit** or **denatured alcohol** is 90% ethanol + 9.5 parts methanol+ 0.5% crude pyridine.

Memory Aid 1: Concentrations of various forms of alcohol

ARM: **A**bsolute [**99.5%**] "**R**ectified spirit [**95%**]"**M**ethylated Spirit [**90%**]

(4) **Standard drink** – Definition of standard drink differs from country to country. But 10 g of absolute alcohol is usually taken as one standard drink or 1 unit.

2. Proof spirit

Proof spirit (100 proof) is defined in three ways

(1) **Most common definition** – That conc. of ethanol, which weighs exactly 12/13 of the weight of an equal volume of distilled water at 11°C. It is 57.06% ethanol by volume

(2) In the US, it is twice the conc. of alcohol by volume. Thus 50% ethanol is 100 proof

(3) that minimum conc. of ethanol which when mixed with gunpowder would ignite (57.06%). Spirits weaker than 57.06% are **underproof**; stronger are **overproof**.

Fig. 40.1: 40% alcohol is 80 proof; 44%, 88 proof, and 95%, 190 proof. Pl see labels encircled in red.

3. Concentration of ethyl alcohol in beverages

Table 1 gives the alcoholic content of some common alcoholic beverages.

Table 1: Some common alcoholic beverages and their alcoholic content

Alcoholic beverage	Alcohol content in % (v/v)
Beers (lager)	3.2-4.0%
Malt liquor	3.2-7.0%
Ales	4.5%
Caffeinated alcoholic beverages (CABs)	5%-12%
Porter	6.0%
Stout	6.0-8.0%
Table wines	7.1-14.0%
Sparkling wines	8.0-14.0%
Sake	14.0-16.0%
Fortified wines	14.0-24.0%
Aromatized wines	15.5-20.0%
Aquavit	35.0-45.0%
Okolehao	40.0%
Brandies	40.0-43.0%
Gins	40.0-48.5%
Vodkas	40.0-50.0%
Tequila	45.0-50.5%
Whiskies	40.0-75.0%
Rum	40.0-95.0%

4. Absorption

(1) **Digestion prior to absorption** - Ethanol is not digested prior to absorption and is absorbed as such.

(2) **Molecular size** - Its small molecular size allows it to pass readily through membranes by simple diffusion.

(3) **From mouth and esophagus** – Only traces are absorbed.

(4) **From stomach and intestines** - About 20% is absorbed from the stomach and 80% through small intestine. Absorption is rapid; about 60% of alcohol taken on an empty stomach is absorbed in 30 minutes and 90% in one hour.

(5) **Appearance in blood** - It can be detected in the blood within 2-3 minutes of ingestion. Maximum blood levels are achieved within 45-90 minutes of ingestion [30 min if stomach is empty]. **Table 2** gives various conditions, where absorption of alcohol is - or .. Most conditions where absorption is - can be explained by the fact that it passes immediately to small intestines, where absorption is maximum (and vice versa). **B**lood **a**lcohol **c**oncentration is often abbreviated as **BAC** or **BAL** [level].

(6) V_d - 0.5-0.7 L/kg)

(7) Because of first-pass metabolism by gastric and liver alcohol dehydrogenase (ADH), oral ingestion of ethanol leads to lower blood levels than would be obtained if the same quantity were administered IV.

(8) **Absorption phase** is followed by **elimination phase**, when rate of elimination is higher than that of absorption.

Table 2: Various conditions when absorption of alcohol is increased or decreased

S.No.	Absorption of alcohol ↑ed	Absorption of alcohol ↓ed
1.	When the concentration of alcohol is between 10%-20%	When the concentration of alcohol is below 10% or above 20%. When conc > 40% absorption very slow (pyloric spasm, secretion of mucus due to gastric irritation, reduced gastric motility)
2.	When alcohol is swallowed rapidly, and in large volumes	When alcohol is slowly gulped
3.	When alcohol is taken on empty stomach	When alcohol is taken with food, especially that rich in fat and proteins [gastric emptying delayed] or spicy food [irritated mucosa]
4.	When alcohol is taken with carbonated soda (bubbles greatly ↑ the surface area carrying alcohol)	-
5.	When drink is warm (gastric capillaries dilated)	When drink is ice cold
6.	In habitual drinkers	In novices
7.	In patients of gastrectomy	-
8.	With simultaneous ingestion of parasympathomimetic drugs (e.g. prostigmine)	With simultaneous ingestion of parasympatholytic drugs (e.g. atropine), or with sympathomimetic drugs (e.g. adrenaline and benzedrine)
9.	drugs which ↑ gastric motility (e.g. insulin)	Drugs which ↓ gastric motility
10.	-	Achlorhydria and chronic gastritis
11.	↑ed sympathetic tone [stress] – stress and anxiety may have unpredictable effects	↑ed vagal tone [nausea, anxiety]
12.	Drinking before noon	Cigarettes [slowed gastric emptying]

5. Distribution

(1) Alcohol is distributed to the tissues in proportion to their water content [**Table 3**].

(2) **Blood brain barrier** – crossed by ethanol easily.

(3) **Solubility in fat -**

(i) Alcohol is not distributed to adipose tissue as it is insoluble in fat.

(ii) Thus same amount of alcohol given to an obese person [or a woman] produces -er blood levels, as their aqueous compartments are smaller.

(iii) In general females acquire **25% higher blood levels** from same amount of drinks.

(4) **As arterial blood passes through the lungs -** some alcohol is lost by diffusion into the alveolar air.

(5) **Venous blood alcohol** – is about **10% lower** than arterial blood in absorption phase. Since alveolar air is in equilibrium with arterial blood, this accounts for a -er conc of alcohol in alveolar breath compared to venous blood. After one hour or more of drinking, both arterial and venous blood show same concentration.

(6) **Equilibrium of arterial blood level with muscle** – occurs in about 2 hours.

(7) **RBCs -** contain . alcohol than plasma; thus whole blood conc is slightly . than in plasma or serum.

Table 3: Ratio of Blood alcohol concentration [BAC] and various body organs/fluids at equilibrium (shaded rows indicate where organ/sample concentration is lesser than that of blood)

S.No	Body organ/fluid/sample	Blood:sample concentration
1.	Urine	1:1.3
2.	Saliva (pure, as obtained by special cannulae in the parotids)	1:1.21
3.	CSF	1:1.2
4.	Sweat	1:1.2
5.	Vitreous	1:1.2
6.	Tears	1:1.14 (range 1.08 to 1.20)
7.	Plasma or serum	1:1.12 to 1:1.2
8.	Saliva (mixed with mucous secretions from the mouth, as is likely to be obtained in ordinary laboratory conditions)	1:1.02 to 1:1.08
9.	Mother's milk	1:1
10.	Brain	1:0.85
11.	Liver	1:0.85
12.	Alveolar air	1:1/2100

6. Metabolism and excretion

(1) About 90% of alcohol absorbed is oxidized in liver; 10% is excreted -

(i) Breath – 5%

(ii) Urine – 5%

(iii) Feces, milk, saliva, sweat, tears – negligible amounts. Peculiar odor of alcoholic is due to alcohol in breath and sweat.

(2) **Metabolism** -

(i) First to acetaldehyde by ADH and then

(ii) To acetic acid by aldehyde dehydrogenase [AlDH]

7. Mechanism of action

(1) **At organ level** -

(i) Brain - CNS depressant (a) Causes an irregularly descending type of depression; higher centers within cerebrum are depressed first, followed by "midbrain " reticular activating system "thalamus"spinal cord "medulla [respiratory center is inhibited last of all resulting in death]. (b) Within the cerebrum, frontal lobes are depressed first [they are sensitive even to low conc, resulting in mood changes]; followed by "occipital lobes [visual disturbances]"cerebellum [loss of co-ordination].

(ii) Other organs – EA has direct effects on cardiac muscle, thyroid and hepatic tissue.

(2) **At molecular level** -

(i) GABA receptors - EA binds directly to GABA receptors in the CNS " causes sedative effects similar to those of benzodiazepines, which bind to the same GABA receptor.

(ii) N-Methyl-D-aspartate (NMDA) receptors - (a) EA potently and selectively inhibits N-methyl-D-aspartate (NMDA) type of inotropic glutamate receptors [NMDARs] (b) prolonged ethanol exposure produces a compensatory ‘upregulation’ of NMDAR functions. (c) These changes underlie the development of ethanol tolerance, dependence as well as acute and delayed signs of withdrawal.

8. Acute poisoning

(1) **Cause** - Serious acute alcohol poisoning occurs usually as a result of deliberate heavy drinking.

(2) **Constancy** - Signs and symptoms are not constant in all people; at the same blood concentration, they would vary according to whether the person is a beginner, moderate drinker or heavy drinker.

(3) **Phases** - **Seven phases** of intoxication are known. **Table 4** must be referred for most common signs and symptoms. Main text includes additional features and explanations.

a. Signs and symptoms

(1) **Stage of sobriety [**BAC=0-50 mg%; **Table 4]** –

(i) No significant effect or (ii) mild euphoria (iii) easy socialization. (iv) Roughly midway is the legal limit of driving in India [30 mg%].

(2) **Stage of euphoria** [BAC = 50-100 mg%]–

(i) Higher centers of brain are inhibited " (a) .ed attention span (b) .ed cognitive function (c) .ed inhibitions [feeling of well-being] (d) .ed sensory perceptions (e) -ed self confidence (f) -ed sociability (g) -ed talkativeness (h) Alteration of judgment

(ii) Alcohol flush reaction [AFR]- (a) Flushing of the face, neck, and shoulders after the ingestion of alcohol, often accompanied by nausea, dizziness, and palpitations. (b) Very common in Asians with almost 50% involvement of some Mongoloid groups [for this reason, also known as **Asian flush syndrome**, **Asian flush**, **Asian glow** and **Oriental flushing syndrome**] (c) caused by a defective allele of AlDH, which encodes for aldehyde dehydrogenase [please see “metabolism and excretion” above]. Its deficiency causes accumulation of acetaldehyde, which is responsible for AFR. The defective allele originated among Han Chinese in central China, and was positively selected in the past, because - concentrations of acetaldehyde conferred protection against parasitic infections especially Entamoeba histolytica. (d) The reaction also occurs when alcohol is taken by people receiving treatment with alcohol sensitizing drugs such as disulfiram (Antabuse), which inhibit aldehyde dehydrogenase. (e) associated with - risk of esophageal cancer in those who drink (f) Also with auto-brewery syndrome [please see below].

(iii) Pulse – rapid, full and bounding.

(iv) BP – slight - especially at systolic level.

(v) Temp – usually - at this stage. During later stages it may be subnormal.

(vi) Muscular system - (a) Fine movements affected (b) muscular incoordination [mild]. (c) If driving - Incidence of vehicular accidents is -ed [Roughly midway is the legal limit of driving in UK (80 mg%)].

(vii) Slurring of speech begins [-es in all subsequent stages].

(viii) **Nystagmus** – due to effects of alcohol on vestibulocochlear system. Two types are described – AGN and PGN. (a) **Alcohol Gaze Nystagmus (AGN)** - Seen in a vertical position, with subject sitting or standing. (I) Two types – Horizontal gaze nystagmus [HGN] and Vertical Gaze Nystagmus [VGN]. HGN is an involuntary jerking of the eye that occurs naturally as the eyes

gaze to the side. Under normal circumstances, nystagmus occurs when the eyes are rotated at high peripheral angles. However, when a person is impaired by alcohol, nystagmus is exaggerated and may occur at lesser angles. In the HGN test, the examiner observes the eyes of a suspect as he follows a slowly moving object such as a pen [thus also known as **pen test**] or small flashlight, horizontally with his or her eyes. The examiner looks for three indicators of impairment in each eye: if the eye cannot follow a moving object smoothly, if jerking is distinct when the eye is at maximum deviation, and if the angle of onset of jerking is within 45 degrees of center. If, between the two eyes, four or more clues appear, the suspect likely has a BAC ≥80mg%. VGN is demonstrable at higher levels. (b) **Positional Alcohol Nystagmus [PAN]-** Seen in a lying down position (I) Nystagmus is seen when the head is placed in a sideways position. (II) Two types – PAN I [nystagmus to the right when the right side of the head is down, and to the left when the left side is down] and PAN II [opposite directions as in PAN I]. (III) Time of appearance - (A) PAN I occurs first [within ½ h; lasts for 3-4 h]. Occurs during rising and peak BACs; begins at **40 mg%**; but most often observed at ≥80mg%. (B) PAN II appears later [1-2 h after disappearance of PAN I; lasts for 4-5 h, i.e. up to 10 h after drinking episode] during the 'morning after' stage [hangover] as the BAC is .ing as alcohol is being eliminated from the bloodstream, and the vestibular canals are beginning to regain balance. It is claimed to be a true, objective after-effect of alcohol and form a part of the so-called hangover syndrome. (IV) How to elicit – person made to lie down; head turned to either side. PAN is best elicited with eyes closed [seen through eyelids as mild flutter]. Opening the eyes will block PAN if its intensity is low, but not if intensity is high.

(3) **Stage of excitement** [BAC = 100-150 mg%]–

(i) Higher centers of brain – Inhibited further (a) Lack of self control and loss of critical judgment - may cause the person to disclose secrets (b) .ed concentration (c) Emotional instability (d) Excitement (e) Lack of self control (f) Loss of critical judgment (g) Comprehension, memory and reasoning impaired (h) dizziness (i) Mental confusion (j) Sleepiness

(ii) Eyes – (a) Visual acuity .ed – (I) In dim light objects may not be distinguished at all; strong light is needed. (II) After being dazzled by

strong light, it takes longer to see clearly again (b) eyelids – swollen and red (c) Conjunctivae - congested (d) Pupils – dilated. [contracted in later stages, and then dilate once again just before death. Similar situation is seen in chloroform. Mechanism is the same.

Memory Aid 2: Pupils in alcohol poisoning at the stage of excitement

D''C''D. Same as for chloroform [pl see above]. **MacEwen sign** [pl see below] is elicited at the stage of contraction.

(iii) Time and space perception – Altered [person may underestimate speed of objects and distances travelled]

(iv) Muscular system - (a) Ataxia [mild] (b) Slowed reaction time.

(4) **Stage of disorderly behavior** [BAC = 150-200 mg%]–

(i) Higher centers of brain – Inhibited further (a) Normal good manners - forgotten

(ii) Eyes – (a) Visual acuity, eyelids, conjunctivae - same as in earlier stages (b) Diplopia (c) Pupils - may now be unequal. Reaction to light has lost its briskness. Contract slowly when light is shown, and equalize. Then dilate slowly even if light is continually shown.

(iii) Muscular system - (a) Ataxia [worsened], Staggering, unsteady gait (b) Disorientation (c) emotions exaggerated (d) Mental confusion exacerbates (e) Vertigo

(5) **Stage of stupor** [BAC = 200-300 mg%]–

(i) .ed response to painful stimuli

(ii) Drowsiness

(iii) Inability to stand or walk

(iv) Inertia

(v) Loss of muscular co-ordination

(vi) Vomiting

(6) **Coma** [BAC = 300-500 mg%]–

(i) Anesthesia

(ii) Eyes: Pupils – now contracted. **MacEwen sign** +ve. An irritating stimuli eg pinching, slapping or pulling beard or hair causes them to dilate with slow return back to contraction even though the patient does not regain consciousness to any degree. It may take up to 20 min to reach their previous contracted state. Such mild stimulus would not cause dilatation if

coma was caused by a structural disease. Also known as **MacEwen's sign or MacEwen's pupil**.

Memory Aid 3: McEwen sign

Memory aid C"D"C.

Discovered by Scottish surgeon **Sir William MacEwen** (1848–1924), whose main problem as a casualty surgeon was to distinguish coma due to alcohol from that produced by conditions such as concussion, skull fracture, apoplexy, and opium use.

(iii) Incontinence of urine and feces

(iv) Reflexes abolished

(v) Respiratory depression

(vi) Temp [subnormal]

(vii) Unconsciousness

(7) **Death** [BAC = >500 mg%] - due to respiratory failure. Pupils dilate just before death just as in chloroform.

(8) Rare and unusual symptoms - allergic reactions [urticaria]. May result in death.

b. D/d

(1) Trauma

(2) Various illnesses causing confusion and delirium.

Table 4: (with **Memory Aid 4**) Alcohol intoxication in relation of blood concentration [7 stages]. Please co-relate signs and symptoms with text

S.No	Blood alcohol conc [BAC][mg%]	Stage of intoxication	Mnemonic [memory aid]
1.	0-50	Sobriety	Dry and decent
2.	50-100	Euphoria	delighted and devilish
3.	100-150	Excitement	dizzy and delirious
4.	150-200	Disorderly behavior	delinquent and disgusting
5.	200-300	Stupor	dazed and dejected
6.	300-500	Coma	dead drunk
7.	>500	Death	Dead as dodo

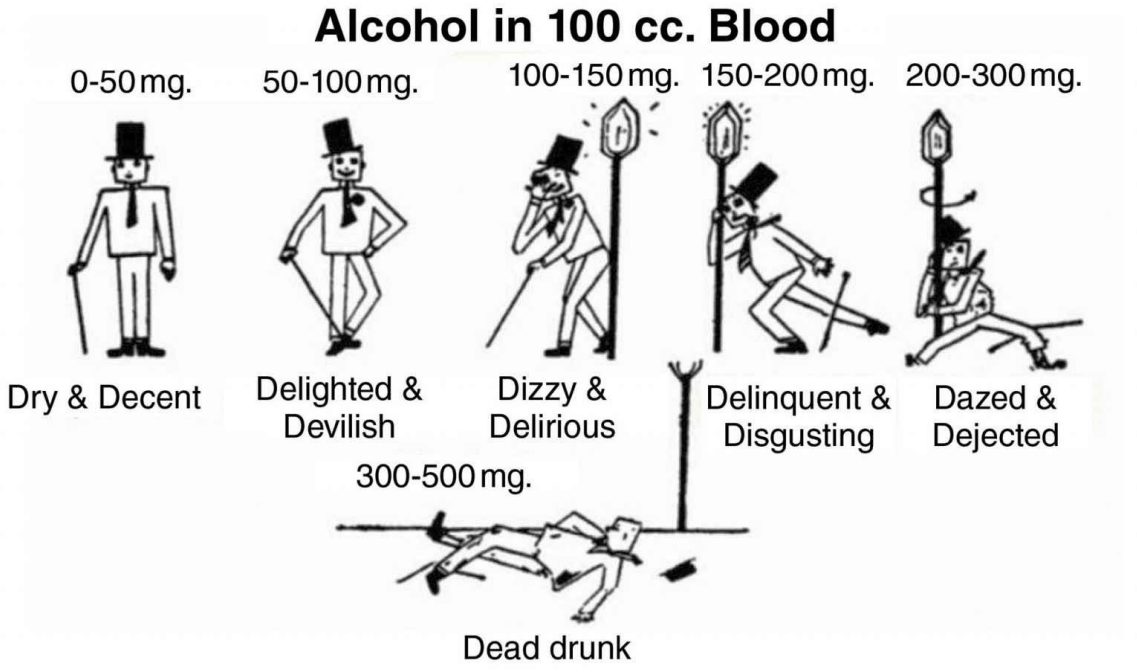


Fig. 40.2: Alcoholic intoxication in relation to blood alcohol. Please compare with **Table 4** above.

Memory Aid 5: Confirmation of ethyl alcohol poisoning
CONFIRMED
C - Confusion or confabulation or memory loss for recent events.
O - Odour of breath, stale alcohol / ingredients / metabolites.
N - Nystagmus.
F - Funny expressions, slurred speech and fuddled thinking.
I - Inco-ordination and postural instability.
R - Rapid, full and bounding pulse.
M - Maudlin. Half-drunk, tearfully affectionate or "familiar"
E - Eyes bloodshot. Pupils dilated and sluggish in reaction.
D - Demeanour. Deviations of behaviour, bearing and deportment.

c. Management

(1) ABCDE of Immediate resuscitative measures – **ch 31**.
(2) **Gastric lavage** – preferably with an alkaline sol^{n}.
(3) **Activated charcoal** – not useful
(4) **IV fluids** – 1 L normal saline + 10% glucose + 100 mg thiamine + 15 units insulin
(5) **Hemodialysis** – very useful.
(i) Eliminates ethanol 4 times faster than that metabolized by liver.

(ii) Not employed normally, but may have to be used if (a) Blood levels are excessive (b) Liver function is compromised [as in ch alcoholics] (c) Condition is deteriorating continuously

(6) Peritoneal dialysis – useful. Indications same as hemodialysis.

(7) **Drugs** – Not much useful. Following have been tried

(i) Caffeine

(ii) Flumazenil - May be considered for patients with hepatic encephalopathy. Reverses respiratory depression associated with ethanol.

(iii) Naloxone

(iv) Physostigmine.

d. Tests

(1) **Woodbury's test** [syn - **Dichromate test**] - 5 mL urine + 5 mL of 10% sodium dichromate in 50% H_2SO_4"Green color if ethanol is present.

(2) **Bedside test** - 5 mL urine + 1 mL acetic acid + 1 drop H_2SO_4 [heat gently in a test tube for 1 min]"A characteristic strong fruity odor [due to ethyl acetate] if ethanol is present.

e. Fatal dose

(1) **Ingestion** –

(i) Adults - 5-8 g/kg absolute ethanol [6-10 mL/kg].

(ii) Children - 3 g/kg (4 mL/kg). Paradoxically larger doses, especially taken quickly may not kill, because of violet emesis set up due to GIT irritation [**ch 31**"**Causes modifying action of poisons.** $CuSO_4$ behaves the same way **(ch 36)**].

(2) **Blood conc** – >500 mg% is fatal

(3) **Inhalation** -

(i) Does not cause problems unless concentrations are high.

(ii) Irritating to eyes and skin.

(iii) MAC in air – 1000 ppm.

f. Fatal period

12-24 h.

g. Cause of death

(1) Depression of brain stem"Depression of Respiratory Center"respiratory paralysis.
(2) Aspiration of vomit.

h. Postmortem appearances [acute poisoning]

(1) **Conjunctiva** - Congested
(2) **On opening the body** – Alcoholic odor from body cavities, stomach contents and from viscera
(3) **Stomach** – congested with a coating of mucus [due to irritation]
(4) **Internal organs** – Brain and meninges, liver, spleen, kidneys etc congested
(5) **Blood** – Fluid and dark
(6) **Microscopically** – Cloudy swelling of parenchymatous organs
(7) changes seen in chronic alcoholism, if subject is a ch. alcoholic. Please see below.

i. Collection of specimens [at PM]

(1) For collection of specimens **in the living**, please see "Clinical examination of a case of drunkenness" below.
(2) For **general guidelines** regarding collection of **specimens in the dead**, please see **ch 5**.
(3) Specimens helpful specifically in alcohol are (i) Blood (ii) Brain (iii) CSF (iv) Lung (v) SDH [in cases of long survival] (vi) synovial fluid (vii) Vitreous
(4) Specimens in embalmed bodies – **ch 9**.

9. Chronic poisoning

Syn - Alcohol addiction, chronic alcoholism, ethanolism.

a. Definitions

DSM-IV-TR, classifies "**Alcohol Use Disorders**" into 2 broad groups - **Alcohol Dependence** (Diagnostic Code - 303.90) and **Alcohol Abuse** (Diagnostic Code - 305.00). Definitions are same as for substance dependence and substance abuse [please see **ch 46** – Drug dependence and drug abuse]. ***Alcohol addicts*** or ***chronic alcoholics*** are people who cannot stop drinking for long periods or who experience withdrawal symptoms if they do so. Most of them have reached a state of irreversible somatic or brain changes induced by alcohol.

b. Tolerance to alcohol

Tolerance to alcohol means that after continued drinking, consumption of a constant amount of alcohol produces a lesser effect or increasing amounts of alcohol are necessary to produce the same effect.

Salient features:

(1) Tolerance may - with alcohol use, till liver gets damaged with . capacity to metabolize. Thus tolerance is restricted by liver damage

(2) **Relationship with barbiturates** – The signs and symptoms of alcohol and barbiturate intoxication are similar, as are the signs and symptoms of abstinence from these drugs and their routes of metabolism. Barbiturates will suppress alcohol abstinence phenomena, and alcohol will suppress, at least partially, the symptoms of barbiturate withdrawal. The two drugs are essentially additive and interchangeable in chronic intoxications. Consumption of alcohol along with barbiturates -es their duration of action. Because of these similarities one of the 7 types of drug dependence has been termed "barbiturate-alcohol type" [**ch 46**].

c. Signs and symptoms

(1) **GIT -** Nausea, vomiting, anorexia, diarrhea

(2) **Hepatic** – Jaundice

(3) **CNS** – tremors of tongue and hands, insomnia, loss of memory, impaired power of judgment. Peripheral neuritis and dementia occur in advanced stages

(4) **nutritional** – Hypoproteinemia, general anasarca.

d. Subtypes of chronic alcoholism

American physiologist **Elvin Jellinek** (1890-1963) identified five subtypes of alcoholism.

i. Alpha alcoholism

(1) **Psychological dependence** only
[**Memory Aid 6**: Al**P**ha - **P**sychological].

(2) **Syn - P**roblem drinking, esca**P**e drinking.

ii. Beta alcoholism

(1) Results in **physical disease states** (ulcers, cirrhosis, damage to the nerves, kidney problems).
(2) No physical dependence.

iii. Gamma alcoholism

(1) **Mild physical dependence** occurs.
(2) Tolerance to alcohol, withdrawal symptoms, craving, loss of control of drinking.
(3) Despite craving, addict can live without alcohol for a short period of time.
(4) **Syn** - "Anglo-Saxon" alcoholism, **malignant alcoholism**] [**Memory Aid 7-Gamma alcoholism - mild physical dependence**].

iv. Delta alcoholism

(1) **Strong physical dependence**.
(2) Addict cannot stay away from liquor even for 1-2 days without suffering from withdrawal symptoms.
(3) high blood alcohol level is maintained.
(4) Syn – alcoholization.

v. Epsilon alcoholism

(1) Individuals engage in **[Memory Aid 8 - Epsilon - periodic] periodic binges**, with long periods of abstaining in between.
(2) Syn – dipsomania, Paroxysmal or periodic alcoholism.

e. Specific conditions seen in chronic poisoning

i. Acute alcoholic hallucinosis

(1) Persistent hallucinations [auditory, visual] develop within 48 h of alcohol intake.
(2) Content is unusually disturbing and unpleasant
(3) May last for several weeks or months.

ii. Alcoholic Brain Syndrome

A general term for a range of disorders due to the effects of alcohol on the brain - amnesic syndrome, delirium tremens [DT], dementia, hallucinosis, psychotic

disorder. Specific terms are preferred.

iii. Alcoholic epilepsy

(1) 45% of alcoholics have seizures at some stage.

(2) Seizures [syn, **rum fits**, **withdrawal seizures**] are grand mal in type and classically occur as part of the withdrawal syndrome. Seen after about a day of termination of drinking session. But may occur rarely even while the patient is drinking.

(3) **Pathogenesis**

(i) Cerebral atrophy

(ii) Alcohol promotes Mg2+ excretion " resultant electrolyte disturbances lower convulsive threshold

(iii) Faulty carbohydrate handling mechanisms, as evidenced by peculiar oral GTT curves

(iv) Alcohol has a biphasic stimulant depressant action on CNS.

iv. Alcoholic paranoia

(1) Alcoholic paranoia - alcohol-induced psychotic disorder in which delusions of reference and persecution occur.

(2) Alcoholic jealousy [syn, amorous paranoia, conjugal paranoia, morbid jealousy] is a form of alcoholic paranoia characterized by delusions of infidelity.

(i) There is intense searching for evidence of infidelity.

(ii) Direct accusations lead to violent quarrels.

v. Alcoholic polyneuritis

Alcoholic polyneuritis [syn, alcoholic peripheral neuropathy] is a painful condition produced by ch alcoholic consumption.

(1) **Signs & symptoms** -

(i) Allodynia [Gk allos, other; dynia, pain], i.e. pain from stimuli which are not normally painful [bedsheets, air movement], or pain which occurs other than in the area stimulated [cf thermal allodynia, which occurs in ciguatera **- ch 39**]

(ii) Hyperalgesia

(iii) Loss of deep reflexes

(iv) Spontaneous burning pain
(v) Tenderness of muscles of arms and legs
(vi) Unsteady gait
(vii) Weakness
(viii) Wrist and foot drop
(2) **D/d** – ch arsenic poisoning [**ch 36**].

vi. Battered alcoholic syndrome

Defined as the presence of three or more fractures in different chronological stages of healing in an alcoholic. **Etiology** - (i) direct toxic effects of alcohol (ii) greater exposure to trauma [- incidence of falls, being involved in fights etc]. In some of these deaths, there may be a genuine concern of alcoholic having been strangulated because of finding of fractures of thyrohyoid complex. Death of chronic alcoholics generally occurs from liver failure, and not from fractures.

vii. Cardiac Conditions

[A] **Beriberi heart** - Due to deficiency of thiamine. Characterized by cardiac failure and edema. No CNS involvement. [B] **Dysrhythmias** – Alcohol withdrawal " -Adrenergic activity " tachyarrhythmias " Sudden death. [C] **Munich beer Heart** - First reported by the German pathologist Bollinger in **1884**. Dilated hearts were found in heavy beer drinkers on autopsy. He called it Münchener Bierherz (Munich beer heart. Now known as alcoholic cardiomyopathy. **Mechanisms** - (1) .ed myocardial protein synthesis, (2) calcium channel dysfunction (3) Disturbance of ion homeostasis (4) oxidative stress. [D] **Myocarditis** - (1) It is part of a bigger syndrome [alcoholic cardiomyopathy]. Not to be confused with beer drinker's cardiomyopathy. (2) **Etiology** - Direct toxic effect of alcohol and its metabolite acetaldehyde. Consumption - 80 g/d for > 5 years for cardiomyopathy to develop.

viii. Cirrhosis

(1) Alcoholic cirrhosis develops in 10-20% of individuals who drink heavily for a decade or more.
(2) **Mechanism** - Alcohol injures the liver by blocking the normal metabolism of protein, fats, and carbohydrates. There may be concurrent alcoholic hepatitis with fever, hepatomegaly, jaundice, and anorexia. Aspartate transaminase

[AST] and Alanine transaminase [ALT] are both -ed but < 300 IU/L with a AST:ALT ratio > 2.0, a value rarely seen in other liver diseases.

(3) **Liver biopsy** - hepatocyte necrosis, Mallory bodies, neutrophilic infiltration, perivenular inflammation.

ix. Delirium tremens

Delirium tremens [Latin, shaking frenzy] [syn, DTs, “the horrors”, “the shakes”] is an acute episode of delirium precipitated by alcohol withdrawal.

Salient features:

(1) First described in **1813** by Pearson. Sutton in same year called it delirium tremens.

(2) **Incidence** – occurs in 5% of patients with alcohol withdrawal.

(3) **Pathology** -

(i) Loss of the inhibitory influences of GABA receptor system

(ii) - in catecholamine release [hyperadrenergic state]

(iii) Hypocalcemia and hypomagnesemia - occur during alcohol withdrawal. Contribute to CNS hyperexcitability by altering neuronal-evoked potentials.

(iv) .ed oxygen use and ATP turnover and release" contributes on the cellular level, to the physiologic changes of alcohol withdrawal.

(4) **Precipitating factors** -

(i) Sudden withdrawal of alcohol [most common] – typically occurs **72-96 h** after last drink

(ii) Acute infections [erysipelas, influenza, pneumonia]

(iii) Shock – as occurs after an injury [eg fractures]

(iv) Temporary excess

(5) **Signs and symptoms** –

(i) CNS and psychiatric – Most significant (a) Agitation (b) Coarse muscular tremors of face, tongue and hands (c) Confusion (d) Delirium (e) Disorientation [in time and place] (f) Hallucinations [auditory, visual and tactile with a predominantly horrific content, eg patient may believe insects are crawling under the skin or snakes are crawling in his bed; syn, delirium of horrors] (g) Insomnia (h) Memory loss (i) Restlessness (j) Seizures (k) Tendency to commit suicide, homicide, violent assault, damage to property (l) Uncontrollable fear

(ii) General - (a) Pupils - Dilated (b) -Temp – due to seizures

(iii) GIT - Diarrhea (iv) Resp – Tachypnea (v) CVS - (a) Tachycardia (b) BP – Variable. May be - or .

(6) **Management** -

(i) Drugs - (a) Drug of choice: Benzodiazepines - Diazepam IV 5 mg every 5 m (b) Other drugs: Rectal paraldehyde, 10 ml every 30 m.,

(ii) Supportive - (a) large amounts of parenteral saline and (b) dextrose to control hypotension.

(7) **Death** – occurs in 5-15% cases.

(8) **Cause of death** –

(i) Cardiac arrhythmias associated with hypokalemia

(ii) Fat emboli

(iii) Hyperpyrexia, poor hydration and hypotension

(iv) Infections

(v) Respiratory failure

(vi) Heat stroke [rarely].

(9) **PM appearances** – Same as in ch alcoholic poisoning.

x. Gastric and peptic ulceration

Due to irritant effects of alcohol.

xi. Korsakoff's psychosis

Korsakoff's psychosis (Korsakoff's dementia, Korsakoff's psychosis, Korsakoff's syndrome, amnesic-confabulatory syndrome), is a neurological disorder caused by the lack of vitamin B_1 (thiamine).

Salient features:

(1) First described by Russian neuropsychiatrist **Sergei Korsakoff** [1854-1900]

(2) Late neuropsychiatric manifestation of Wernicke's encephalopathy. Also known as Wernicke-Korsakoff syndrome [WKS]

(3) **Clinical features** – 3 salient features

(i) Amnesia [anterograde (inability to form new memories) and retrograde (loss of existing memories)]

(ii) Confabulation [falsification of memory in an alert, responsive individual]. The memory problems associated with Korsakoff 's syndrome are irreversible.

(iii) Polyneuropathy [psychosis polyneuritica]

Memory Aid 9: Korsakoff's psychosis

Korsakoff's psychosis is a more **K**ronic condition than Wernicke's encephalopathy. **K**onfabulation is seen. All features are remembered by **PAK** – **P**olyneuropathy, **A**mnesia, **K**onfabulation

xii. Mallory-Weiss syndrome

Ruptured esophagus with mediastinitis

xiii. Malnutrition

Due to lack of food intake, calorie requirement being satisfied by alcohol.

xiv. Mania a potu

Mania a potu ["madness from drinking"] is a state of pathological intoxication, consisting of extreme excitement, sometimes with homicidal attacks and resulting from the ingestion of comparatively small amounts of alcohol by a susceptible individual.

xv. Marchiafava syndrome

(1) Also known as **Marchiafava-Bignami syndrome** [MBS] or disease [MBD].

(2) **Signs and symptoms** - Marked impairment in brain functioning. Severe global dementia.

(3) **Cause** - Necrosis and atrophy of corpus callosum.

xvi. Mental illness

Depression, suicidal tendencies.

xvii. Pancreatitis

Alcoholic pancreatitis is a major complication of alcohol abuse.

Salient features:

(1) Alcohol exerts dose-related toxic effects on the pancreas.

(2) Risk of developing pancreatitis -es with -ing doses of alcohol.

xviii. Tryptophan metabolism disturbance

In ch alcoholics, conversion of tryptophan to 5-hydroxyindoleacetic acid is depressed.

xix. Wernicke's encephalopathy

Wernicke encephalopathy is a syndrome, caused by lesions in the medial thalamic nuclei, mammillary bodies, periaqueductal and periventricular brainstem nuclei, and superior cerebellar vermis, caused by inadequate intake or absorption of vitamin B_1 (thiamine), especially in conjunction with carbohydrate ingestion.

Salient features:

(1) First described by German physician **Carl Wernicke** [1848 –1905]

(2) **Etiology** –

(i) Ch alcoholism - Alcoholic fulfils his caloric demands from alcohol [empty calories]; thus avoiding food

(ii) Thiamine deficiency arising from gastric disorders - (a) carcinoma (b) chronic gastritis, (c) Crohn's disease, (d) repetitive vomiting, particularly after bariatric surgery and

(iii) Starvation.

(3) **Signs and symptoms** – [A] Classical triad - (i) ***A***taxic gait (ii) ***E***ncephalopathy [agitation, amnesia (short term memory loss), indifference and inattentiveness, confusion, disorientation, disturbance of consciousness, delirium, drowsiness and stupor] (iii) ***O***culomotor disturbance [nystagmus, external ocular palsies (commonly lateral rectus palsy), ophthalmoplegia, sluggish pupillary reflexes, anisocoria (unequal size of pupils)].

Memory Aid 10: Wernicke's encephalopathy

Classical triad of Wernicke's encephalopathy *includes 3 vowels* - **A**; **E**; **O**

[B] Other imp signs - (i) Peripheral neuropathy. [C] Atypical symptoms - Dysphagia. (1) **If left untreated** – 80% cases progress to Korsakoff's psychosis [a more chronic condition]. (2) **Management** - (i) IV thiamine (ii) IV glucose [both of these are part of coma cocktail]. (3) **Mortality** – high. Can cause death within 48 h of onset.

f. Management

i. Drug treatment

(1) **Baclofen** (Kemstro, Lioresal, Liofen, and Gablofen) - Derivative of GABA, primarily used to treat spasticity but now found useful in alcoholism also.

(2) Benzodiazepines

(3) **Clomethiazole** [syn, chlormethiazole] – Sedative and hypnotic. Used in treating and preventing symptoms of acute alcohol withdrawal.

(4) **Chlorpromazine** – 25-50 mg every 4-6 h.

(5) **Citrated Calcium carbimide** [CCC, Temposil] -

(i) Works in the same way as disulfiram [please see below].

(ii) Dose – 50 mg od

(iii) Advantages - Adverse effects of disulfiram, hepatotoxicity and drowsiness, do not occur.

(6) **Clonidine** – 60-180 mg IV/hr. MOA same as that in opioid withdrawal.

(7) **Disulfiram** – [Antabuse, Antadict, Esperal] –

(i) Mechanism - It inhibits the metabolism of alcohol beyond acetaldehyde stage. When patient on disulfiram takes alcohol, he gets unpleasant effects of acetaldehyde [Alcohol flush reaction], forcing him to abstain from alcohol.

(ii) **Dose** - start with single daily dose of 250 mg. Dose is .ed till a daily dose of 0.125-0.25 mg is reached.

(8) **Ibogaine** [**ch 46**].

(9) **LSD** [**ch 46**].

(10) **Naltrexone** (Trexan) – Mechanism - (a) Alcohol causes the body to release endorphins, which in turn release dopamine " reward pathways are activated. This is one of the mechanisms of addiction. (b) Naltrexone is a competitive antagonist for opioid receptors; blocks effects of endorphins ".es craving for alcohol and encourage abstinence.

(11) **N-Methyl-D-aspartate (NMDA) receptor blockers** –

(i) Mechanism - (a) Prolonged use of alcohol upregulates NMDARs [please see above under “Mechanism of Action”] (b) NMDAR blockers are thus useful for pharmacotherapy of alcoholism. They attenuate not only the physical symptoms but also some affective and motivational components of alcohol withdrawal.

(ii) NMDAR blockers useful in alcohol dependence - (a) Acamprosate [Campral] (b) Memantine (c) Neramexane

(12) **Ondansetron** [Zofran] –

(i) $5HT_3$ antagonist.

(ii) .s cravings for alcohol, especially in early-onset alcoholics.

(iii) Combination of ondansetron and naltrexone is superior than either treatment alone.

(13) Topiramate (Topamax)
(14) **Vitamin supplements** - esp thiamine.

ii. Conditioned reflex treatment

(1) **Mechanism** – Conditioned reflex treatment [CDR] works on the principle of classical conditioning [syn, Pavlovian or respondent conditioning]. He observed that the dogs began to salivate in the presence of the lab technician who normally fed them. Pavlov called these **psychic secretions**. He then used a bell to call the dogs to their food and, after a few repetitions, the dogs started to salivate in response to the bell. Terminologies used in such experiments are

(i) Unconditioned stimulus (US) – food
(ii) Unconditioned response (UR) – Salivation
(iii) CS – Light
(iv) Conditioned response (CR) – Salivation in response to light

(2) **Procedure** -

(i) With a backdrop of bottles of various alcoholic beverages [CS] the patient is given various types of liquor [US] together with drugs that will cause immediate nausea and vomiting [UR].
(ii) After 5-8 daily treatments, unpleasant symptoms are brought on simply by the sight of a bottle [CR].

g. Cause of death

(1) Hepatotoxicity [most common]
(2) most of above mentioned complications.

h. Postmortem appearances [ch alcoholism]

(1) **Esophagus** - shows varices
(2) **Stomach mucosa** -
(i) Hypertrophied
(ii) Color – Deep reddish brown with patches of congestion and effusion.
(iii) Gastritis and peptic ulcer disease
(3) **Brain** -
(i) Central pontine myelinosis [CPM] - may be associated with a low-salt vitreous electrolyte pattern.
(ii) Cerebellar vermis atrophy

(iii) Mamillary body atrophy or necrosis [indicative of Wernicke–Korsakoff syndrome in association with thiamine deficiency]
(iv) Remote (old) brain contusions [consistent with falls, being involved in fights].

(4) **Lungs** - Pulmonary infections including pneumonia, aspiration pneumonia, and abscesses

(5) **Heart** -

(i) Alcoholic cardiomyopathy

(ii) Dilated

(iii) Shows fatty degeneration.

(6) **Peritoneum** - Bacterial peritonitis is commonly seen.

(7) **Liver** –

(i) Congested

(ii) Alcoholic hepatitis

(iii) May be greasy to touch, steatosis.

(iv) May show fatty changes or cirrhosis [contracted, nodular].

(8) **Kidneys** – Granular degeneration

(9) **Bones** – Chronic alcoholics show -ed incidence of incidental old healed fractures. Please see "Battered alcoholic syndrome" above.

10. Alcohol withdrawal syndrome

The ***alcohol withdrawal syndrome*** (***AWS***) is a syndrome seen in alcohol-dependent individuals, typically within 24–48 hours of consumption of their last drink.

11. Clinical Examination of a case of drunkenness

Drunkenness is a condition produced in a person, who has taken alcohol in a quantity sufficient to cause him to lose control of his faculties to such an extent, that he is unable to execute safely the occupation in which he was engaged at the particular time.

Salient features:

(1) **Clinical diagnosis** – Depends on a combination of symptoms and signs. No single sign or symptom is reliable, except odor of alcohol from breath.

(2) **Variability** – Different individuals would show different signs and symptoms with same amount of alcohol intake. Even same individual may show different

signs and symptoms at different times, depending upon his state of metabolism.

(3) **Individuals showing excessive reaction** –

(i) Epileptics (ii) Liver and kidney disease (iii) Mentally unstable subjects (iv) Past cerebral trauma

(4) **Situations needing examination** - Police officer would bring a case of alcoholic for examination most often when the person was driving or causing disturbance in public after taking alcohol. The following scheme of examination has been suggested by the Special Committee of the British Medical Association.

a. Consent

(1) Consent is tricky for examination of an alcoholic.

(2) **If consent not taken** "Legally it is application of criminal force [S.350, IPC; **ch 2**]

(3) **If consent taken, and person is later proved to be under the influence** - he may claim that his consent had no value

(4) **Examination u/s 53 CrPC** - Virtually in all cases police will bring an alcoholic for examination, after arrest [S.202 Motor Vehicle Act, 1988; S.510, IPC etc]. Doctor can examine all such cases without consent u/s 53, CrPC.

b. Preliminaries

Record name, age, sex, date of examination etc as in any MLC [**ch 11**]. Note specifically the time at the beginning and end of examination.

c. Exclusion of injuries and pathological states

Conditions which simulate intoxication should be excluded. These are

(1) CO poisoning

(2) Drugs [antihistamines, hyoscine, insulin, morphine, sedative hypnotics (eg barbiturates), tranquillizers]

(3) Head injury

(4) High fever

(5) Impending cerebrovascular accidents

(6) Metabolic disorders [Diabetic precoma, Hyperthyroidism, Hypoglycemia, Uremia]

(7) Neurological conditions associated with ataxia, drowsiness, dysarthria and tremors [acute aural vertigo, disseminated sclerosis, epilepsy, intracranial tumor, Parkinson's disease] and
(8) Psychiatric disorders [Hypomania, General paresis].

d. History

(1) Take history directly from the accused [even if he is accompanied by a relative] and observe him keenly as he speaks [note dysarthria, shifting gaze etc]
(2) ask the time of intake, amount of alcohol, brand [whisky, rum, wine, as it would indicate conc], mode of intake [**Table 2**]
(3) Ask history of above illnesses and whether he is under treatment for any disorder.

e. General physical examination

Please co-relate with signs and symptoms given earlier
(1) Pulse – at the beginning and end of examination.
(2) BP
(3) Temp
(4) skin – dry, moist, pale or flushed
(5) Mouth
(6) Ears –
(i) Examine for gross impairment of hearing
(ii) If there is impairment, is there any gross pathology accounting for it, eg rupture of TM
(7) Pulmonary – lung disorders, asthma, bronchitis, emphysema
(8) CVS – Pulse, BP etc. Alcoholic heart disease
(9) GIT – enlargement of liver, indicating ch alcoholism
(10) CNS – Whether alert and oriented to time, place and person [**ch 11**]. In presence of CNS diseases, tests like gait, stance, muscular co-ordination, reflexes etc become meaningless. Systemic examination is done to rule out any pathology or disease.

f. General behavior

(1) **General manners and behavior** –

(i) Whether excessively aggressive, resists examination, abusive, or overly submissive. Both situations indicate drunkenness

(ii) Drooling, driveling, dribbling, slobbering etc [saliva flowing outside the mouth].

(2) **State of dress** -

(i) Presence of nasal secretions, saliva, vomit etc on clothing. If yes, note their character, color, smell etc. Whether dried, drying or wet. Is there a wet layer over dried ones [indicates repeated vomiting etc over a long period of time]

(ii) Soiling of underwear or pants etc by urine, feces. Preserve clothing for examination

(3) **Speech** – Note type of speech [over-precise, slurred, thick etc]. Slight blurring of certain consonants is one of the earliest signs of incoordination of the muscles of tongue and lips. Certain test phrases or mild tongue twisters may be used to bring out this difficulty in speech. English speaking nations mostly use “British Constitution”, “Truly rural” or “West Register Street”, but local language tongue twisters should preferably be used. In Hindi, two most common phrases, the author has used with success are “Nandu ke Nana Ne Nandu ki Nani ko Nand Nagari me Nagin dikhaie” and “Pital ke patile mein papita pila pila”. A sober person might utter out the sentences or honestly admit that he is not good at such phrases. Intoxicated person would always insist on getting them correctly, and make repeated mistakes.

(4) **Self-control** – Whether able to control himself in response to demands made on him by the medical staff.

g. Memory and mental alertness

(1) Ask few questions about his movements during preceding few hours.

(2) Details of accidents if any

(3) If he drank at a party, details about who were with him; for what purpose was the party organized.

(4) If he drank at a pub, what was the cost of each drink, and how much did he pay in all

(5) few simple sums of addition and subtraction.

h. Handwriting

(1) Ask to copy a few lines from a book or newspaper. Give him a paper without

lines. Note (i) time taken (ii) repetition and omission of letters or words (iii) ability to read his own handwriting

(2) Retain both original and the copy for production in court.

(3) Ask subject to sign. Compare signature with those on I-card, or driving license etc.

i. Eyes

i. General appearance

(1) Eyelids (2) Conjunctivae.

ii. Visual acuity

Note any gross defect. Co-relate with any existing defect before the drinking episode.

iii. Intrinsic muscles

(1) **Pupils** – equal or unequal; contracted or dilated; any other abnormality

(2) **Reaction to light** – Show light in one pupil. Watch both pupils closely. Note

(i) If reaction is brisk, slow or absent [pupillary reflex time].

(ii) What is the reaction in unexposed pupil

(iii) If pupil contracts, how much time does it take to come back to normal

(iv) Does it dilate even if light is continued to be shown.

(v) Whether the pupils become unequal on exposure to light

(vi) Do they equalize immediately or after some time. All observations may need light to be shown 4-5 times.

iv. Extrinsic muscles

(1) **Convergence** –

(i) Show a finger to subject and ask him to follow its movement. Then take the finger in all directions, and see if subject can follow finger. Alcohol impaired subjects have difficulty smoothly tracking a moving object.

(ii) Next bring finger gradually nearer to the subject. Observe if he is able to converge his gaze.

(2) **Nystagmus** – HGN, VGN and PAN. please see above.

(3) **Strabismus** – note its presence or absence.

j. Gait

(1) Integrity of neuromuscular system is determined by balance and gait.

(2) **Ask subject to walk across room** – Observe (i) Whether he can walk at all or not (ii) Manner of walking – If walks, whether feet wide apart or close together and whether gait is irregular, overprecise, straight or unsteady. (iii) Reaction time to a direction to turn – does the subject turn at once, or takes a step or two before turning? (iv) Manner of turning – Does the subject keep his balance, lurch forward or reel to one side? Does he correct his mistakes in a normal or exaggerated way?

(3) **Walk-and-Turn (WAT) test** – One of the 3 tests which form part of the **Standardized Field Sobriety Test** (SFST). Usually conducted by police officers on the spot [in the US], but can be employed by the doctor also. The subject is directed to take nine steps, heel-to-toe, along a straight line. After taking the steps, the suspect must turn on one foot and return in the same manner in the opposite direction. The examiner looks for eight indicators of impairment:

(i) if the suspect cannot keep balance while listening to the instructions,

(ii) begins before the instructions are finished,

(iii) stops while walking to regain balance,

(iv) does not touch heel-to-toe,

(v) steps off the line,

(vi) uses arms to balance,

(vii) makes an improper turn, or

(viii) takes an incorrect number of steps. 80% of individuals who exhibit ≥2 indicators in the performance of the test will have a BAC ≥ 80 mg%.

k. Stance

(1) **Romberg's test** [syn, Romberg maneuver] – Ask the subject to stand erect with feet together and eyes closed and observe if he sways. Note presence of any tremors during standing. Stand close by as a precaution in order to stop the person from falling over. Romberg's test is +ve if the patient sways or falls [Romberg's positive, Rombergism, Romberg's sign].

(2) **One-Leg Stand test** [**OLS**]- One of the 3 tests which form part of the **Standardized Field Sobriety Test** (SFST). The suspect is instructed to stand

with one foot approx 6” off the ground and count aloud by thousands [One thousand-one, one thousand-two, etc] until told to put the foot down. The examiner times the subject for 30 seconds. Four indicators of impairment are observed

(i) swaying while balancing,

(ii) using arms to balance,

(iii) hopping to maintain balance, and

(iv) putting the foot down. Like the WAT test, 80% of individuals who exhibit ≥2 indicators in the performance of the test will have a BAC ≥ 80 mg%.

l. Muscular co-ordination

(1) **Following tests are performed** -

(i) Lifting 2 objects, eg water filled tumblers from the table and replacing them side by side in a different position.

(ii) Lighting a cigarette with match

(iii) Picking up medium sized objects from floor

(iv) Placing finger to finger

(v) Placing finger to nose

(vi) Unbuttoning or rebuttoning a coat

(2) **Precautions and interpretations** -

(i) Non-performance or inadequate performance may indicate alcohol consumption

(ii) In general no task should be asked to perform which a normal person cannot perform

(iii) Some persons may have difficulty performing above tasks even under normal conditions

(iv) A chronic alcoholic may not be able to perform above tasks even when he has not consumed alcohol.

(v) CNS diseases should have been ruled out.

m. Reflexes

(1) **Knee and ankle reflexes** – delayed or sluggish

(2) **Plantar reflex** – May be extensor or flexor.

n. Miscellaneous tests

(1) Color difference threshold
(2) Complex reaction time
(3) Delayed auditory feedback
(4) Flicker fusion test
(5) Glare recovery test
(6) Nerve conduction speed.

o. Field sobriety tests [FST]

(1) Administered by police officers on the spot during drunken driving rather than by doctors. May be usefully employed by doctors also
(2) The **Standardized Field Sobriety Test** (SFST) is a **battery of 3 tests** developed by the **National Highway Traffic Safety Administration** (NHTSA), USA.
(3) The tests are
(i) Horizontal Gaze Nystagmus (HGN)
(ii) Walk-and-Turn (WAT)
(iii) One-Leg Stand (OLS) [please see above for all].

p. Breath alcohol analyser

Also used by police officers on field rather than by doctors

(1) Commonly known as **breathalyzer**, one can detect alcohol in breath both qualitatively and quantitatively.
(2) **Principle** - Ratio of alcohol between blood and breath is 1:2100.
(3) **Advantages** – can be administered at the spot without requiring complicated apparatus
(4) **Disadvantages** - Not reliable, because for venous blood, this ratio keeps changes as ethanol metabolizes.

q. Bedside tests

Please see above under “acute poisoning”.

r. Collection of specimens

For collection of specimens in the dead, please see “Acute poisoning” above.

i. Blood

(1) **Collection procedure** "ch 31.
(2) To establish whether the blood alcohol level is rising or declining – two specimens must be taken at spaced intervals and their alcohol content compared
(3) **Preservatives** - [ch 5]. (i) Loss of alcohol etc is prevented through bacterial action, glycolysis etc. (ii) Samples with adequate preservatives can be kept at room temperature even for several weeks.
(4) Serum or plasma alcohol concentration is 12-20% higher than that of whole blood.

ii. Saliva

For collection, please see **ch 31**.

iii. Urine

(1) **Collection procedure** "ch 31.
(2) urine conc must be multiplied by 0.75 to get blood concentration, based on blood-urine alcohol ratio of 1:1.3 [**Table 3**]
(3) Extrapolation from this blood level back to the time of incident would indicate blood levels of subject at the critical moment when the accident occurred [please see rate of fall under “Metabolism and excretion”].

s. Lab tests for alcohol

i. Chemical oxidation methods

Utilize chemical oxidation of ethanol by potassium dichromate.

(a) Cavett’s test

(1) Described by **Cavett** in **1938**.
(2) **Micro method** - works well with smaller volumes of sample (eg blood, urine) eg 0.1 mL.
(3) **Principle** - alcohol is oxidized to acetic acid by oxidizing agents such as potassium dichromate and sulphuric acid. 1 ml of N/20 dichromate solution oxidizes 0.575 mg of alcohol.

(b) Kozelka and Hine Test

(1) Described by **Kozelka** and **Hine** in **1941**.

(2) **Macro method** - Requires larger volumes of sample, eg 1 mL.
(3) It is a color or spot test. Reagents used are same as above.
(4) Like all color tests the rate of false +ve and false –ve is high.

(c) Weaknesses of above methods

(1) Both are not specific for ethanol. Problems may arise if a dead body is decomposed or a living subject suffers from diabetes and ketoacidosis
(2) Neither method can be automated. Multiples samples difficult to analyze
(3) one needs to work with corrosive reagents - sulphuric acid and potassium dichromate in both methods.

ii. Gas liquid chromatography

Most reliable method.

t. Other lab tests

Osmolal gap - -ed. Ethanol is the most common cause of -osmolal gap [**ch 31**].

u. Opinion

(1) Opinion is given in 4 standard formats
(2) **Opinion** - After examination of the accused I am of the opinion that the person examined (i) did not consume alcohol (ii) has consumed alcohol, but is not under the influence (iii) has consumed alcohol and is under the influence [intoxicated] (iv) is under the influence of alcohol, and is/but is not a public nuisance if kept at large.
(3) **Samples** - The samples collected [blood, urine, saliva etc] must be mentioned in a separate box in the MLC and following line must be added after the opinion – Blood [urine, saliva etc] has been sent for chemical analysis to determine the conc of alcohol. These must be sealed and handed over to the police along with sample of seal and report.

12. ML importance (of ethyl alcohol)

(1) **Alcohol and traffic accidents** – (i) Progressive loss of driving ability as BAC -es. Reasons (a) Concentration impaired (b) Confidence levels -ed, so more risks are taken by -ing speed etc (c) Judgment dulled – they believe they are driving better than they are (d) Muscular co-ordination .ed (e) Reaction time -ed (f) Visual and auditory acuity .ed. Because of this drunken driving is

prohibited in most countries. [**Driving under influence of drugs**"**ch 46**].

(2) **BAC and driving ability** -

(i) <50 mg% - majority of drivers are not affected

(ii) 50 mg% - (a) tasks which are affected are those which require control of speed, [eg braking] and those which are required in keeping a vehicle on course [eg sensorimotor coordination] (b) Boldness -ed (c) Impulsiveness -ed (d) Tendency to drive faster and more erratically

(iii) 60 mg% - Driver is twice as likely to be involved in an accident as compared to a sober driver

(iv) 80 mg% - Risk of accident begins to - markedly

(v) 100 mg% - (a) Risk of accident is 12 fold (b) All drivers are affected and accidents are common

(vi) 150 mg% - (a) Risk of accident is 20 fold (b) driving is distinctly impaired

(3) **Drunken driving in India**– is prohibited by Motor Vehicles Act, 1988 [MVA, 1988]. (i) **S.185(a)** – (a) sets legal limit of blood alcohol levels for driving in India as 30 mg%. (b) Punishment for 1st offence – 6 months or `2000 fine or both. (c) 2$^{nd}$ or subsequent offence [if committed within 3 y]- 2 y or `3000 fine or both.

(4) **Alcohol and trauma, delayed deaths** – Drunk person "suffers trauma "Intracranial hemorrhages [**EDH**, **SDH**] are produced " dies after several days or weeks "alcohol may have disappeared from blood, but would be present in EDH and SDH [because it is sequestered from metabolic actions of liver]. Thus in delayed deaths due to alcohol, intracranial hemorrhages may be sent for chemical analysis [please also see **ch 5**]. In fact any sequestered hemorrhage anywhere in the body, eg retroperitoneal hematoma, perinephric hematoma, hematoma around fractured bones and joints etc may be used for chemical analysis.

(5) **Auto-brewery syndrome** –

(i) It is the natural production of alcohol from carbohydrates in the GIT by persons suffering from intestinal candidiasis [hence the term ‘auto-brewery’ syndrome].

(ii) Candida albicans readily produce ethanol both in-vitro. In-vivo levels may go up to 80 mg% in Asians who show a -er incidence of a defective allele of **ALDH2** [please see above under “metabolism and excretion”] involved in metabolism of alcohol.

(iii) Some drunken drivers caught driving under influence of alcohol have taken the defense that they never consumed any alcohol, but were suffering from intestinal candidiasis, which produced alcohol in the body. In rare cases this defense has been accepted.

(6) **Brompton's cocktail**– one of its constituents is ethyl alcohol [**ch 41**].

(7) **Drug facilitated sexual assault** – Alcohol is one of the drugs used in DFSA [**ch 25**].

(8) **Embalming fluids** – contain both ethanol and methanol. In an embalmed body determination of these compounds carries no legal value [**ch 9**].

(9) **Endogenous production of alcohol** - Endogenous alcohol [syn, endogenous ethanol, EnEth] is alcohol, which is not ingested but is produced within the body through metabolic processes [normal metabolism, bacterial activity in the GIT]. It can be produced both during life and after death

(i) During life - Traces of alcohol are found in all persons, because of endogenous production.

(ii) After death – due to putrefaction [may vitiate the interpretation of PM alcohol concentration – **ch 9**].

(10) **Fetal alcohol syndrome** [FAS]-

(i) Caused by alcohol consumption by pregnant women.

(ii) The children have a pattern of retarded growth and development, both mental and physical, with cranial, facial, 1imb, and CVS defects.

(iii) The commonest abnormalities are - (a) abnormal palmar creases (b) cardiac [especially septal] defects (c) developmental delay (d) mental retardation (e) microcephaly (f) prenatal and postnatal growth deficiency (g) short palpebral fissures (h) short upturned nose with sunken nasal bridge (i) thin upper lip

(11) **Forced and voluntary ingestions - S.85, S.86, IPC -** please see ch **31**.

(12) **Loss of alcohol after death** -

(i) by evaporation – insignificant

(13) **Manner of poisoning** -

(i) Accidental– Deaths far rarer than those due to methyl alcohol.

(ii) Suicidal– possible, but virtually unknown

(iii) Homicidal– virtually impossible, because of peculiar smell, taste and large fatal dose.

(14) **Misconduct in public by a drunken person** – Punishable **u/s 510, IPC** -

Punishment is simple imprisonment for up to **24 hours**, or **`10 fine**, or both.

(15) **Mixed ingestions** -

(i) Meptazinol - Meptazinol [Meptid] is an analgesic drug used for pain [postoperative, obstetrical, renal colic]. Death may occur with sublethal conc of alcohol if ingested concomitantly. A case has been reported where the victim died with BAC of just 232 mg%.

(16) **Mortality rates** – Among alcoholics are 2 fold -er. Reasons are same as those of -er mortality rates among drug abusers [**ch 46**].

(17) **Postmortem diffusion** - please see **ch 5**.

(18) **Sudden death** -

(i) Alcohol has a toxic effect on myocardium. Produces cardiac arrhythmias causing sudden death. Other poisons which cause arrhythmias "**ch 31**.

(19) **Widmark's formula** -

(i) Used for - calculating amount of alcohol ingested by a suspect if his blood alcohol level is known and vice-versa.

(ii) The formula for blood conc : **a=cpr**, where **'a'** is the alcohol ingested in gm, 'c' the concentration of alcohol in the blood in decigrams%, 'p' weight of the body in kg, and 'r' the Widmark constant.

(iii) The formula for urine conc : **a=3/4 x cpr**, since conc in urine is 4/3 of that of blood

(iv) Widmark constant - (a) represents V_d for alcohol, which is less for women, who have more fat compared to men. (b) Value: Males - 0.68; Females - 0.55.

(20) **Workplace drug testing** [WDT] – is done by many employers to check alcohol intake among employees during work [**ch 46**].

B. Methyl Alcohol (CH_3OH)

Methyl alcohol (Gk. methy "wine" + hyle "wood) is a colorless, volatile liquid with an odor similar to ethyl alcohol, and having a burning taste.

Salient features:

(1) Methyl alcohol (methanol, wood alcohol, wood naphtha, wood spirit) is produced by destructive distillation (heating under vacuum) of wood. What remains behind is activated charcoal.

(2) **Common household products** containing it are – antifreeze, paint removers,

varnish. Used as a solvent in these products

(3) **Slang names** - In India, it is known as bewada, dalda, French polish, hooch, khopadi and ladda.

1. Toxicokinetics

(1) Methanol is rapidly **absorbed** through stomach, intestines, lungs and skin.

(2) **Metabolized** - to formaldehyde [**33 times** more toxic than methanol] and formic acid [**6 times** more toxic than methanol] [**Fig 40.3**].

(3) **Rate of oxidation** – 1/5th of that of ethanol

Memory Aid 11: Toxicity of methanol metabolites

Formaldehyde is a larger word – thus it has a larger toxicity.

3+3=6 " Thus formaldehyde is 33 times more toxic, and formic acid is 6 times

(4) Takes about **3-4 days** to completely disappear from body

(5) With repeated doses, tends to **accumulate** in the blood

(6) **Distribution in the body** – According to water content of the tissues [Vd=0.6-0.7 l/kg]. High concentration is found in **vitreous** [vitreous should be collected in all cases of alcohol related deaths] and optic nerve.

(7) **Elimination** –

(i) 80% is excreted unchanged from lungs

(ii) 3-5% is excreted unchanged in the urine.

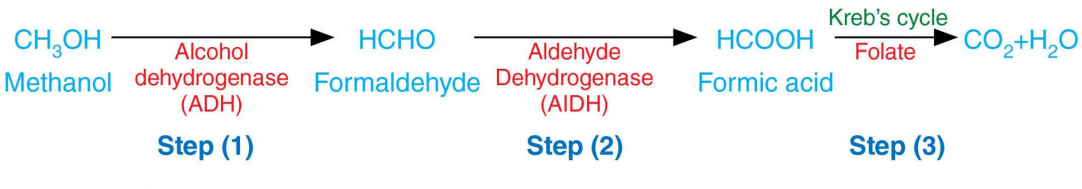


Fig. 40.3: Metabolism of methyl alcohol

2. Mechanism of action

(1) - **formaldehyde** and **formic acid** conc - formed through metabolism [**Fig 40.3**]. Both are highly toxic. Slow degradation of formic acid causes its accumulation in human body.

(2) The **selective neurotoxicity** of methanol is due to **histotoxic hypoxia** as a result of

(i) Inhibition of the cytochrome oxidase complex of the mitochondrial

respiratory chain

(ii) Inhibition of oxidative phosphorylation by .ing both the intracellular synthesis of ATP and the ratio of NAD/$NADH^+$.

(iii) -ed oxidative stress [**ch 31**], caused by **formic acid**.

(3) **Formic acid** [metabolite of methanol] **causes** -

(i) **Retinal toxicity** and (ii) **metabolic acidosis** [Formic acid"inhibits mitochondrial cytochrome oxidase"lactic acid accumulation]. Formic acid causes most of the characteristic signs and symptoms of methanol toxicity.

3. Signs and symptoms

(1) Toxicity may result by absorption through (i) GIT (ii) respiratory tract (iii) skin

(2) **Onset** - Symptoms appear after a latent period of 24 h [range 1-72 h]. The latent period may be longer if ethyl alcohol is a co-ingestant or shorter if the volume of methanol is large.

(3) **GIT** - (i) Nausea, vomiting (ii) Abdominal pain in the form of severe cramps (iii) Pancreatitis

(4) **Vision** – Most characteristic symptoms relate to vision

(i) Blurred, misty or dimmed vision [“flashes” or “snowstorm” vision, “snowfield” vision – as if seeing through a snowstorm]

(ii) Constricted visual fields

(iii) Photophobia

(iv) Nystagmus

(v) .ed light perception

(vi) Seeing spots

(vii) Central and peripheral scotomata [Partial degeneration of field of vision surrounded by normal vision]

(viii) Complete blindness [**Causes** - (a) formic acid inhibits cytochrome oxidase in the optic nerve (b) causes optic neuritis and optic atrophy]

(ix) Ocular examination - (a) pupillary dilatation, (b) loss of pupillary reflexes.

(x) Fundoscopy - (a) Hyperemia of optic disc (b) retinal edema

(5) **CNS** - (i) Confusion (ii) Dizziness, vertigo (iii) Headache with stiff neck [meningismus] (iv) Neck stiffness (v) Drunkenness – is not as prominent as that in ethyl alcohol. Effects are however more prolonged (vi) Delirium and coma may last 2-3 days [effect on CNS more intense and persistent than with

ethyl alcohol] (vii) Pupils - dilated and fixed in later stages

(6) **Muscular system** - Marked muscular weakness

(7) **Respiratory system** – Dyspnea, cyanosis, odor of alcohol from breath

(8) **CVS** - Depressed cardiac action leading to (a) Tachycardia (b) severe hypotension [Alcohol-induced vasodilation, combined with vomiting]

(9) **Urinary system** - (i) Acute tubular necrosis (ii) Urine – strongly acidic. Contains albumin traces and acetone

(10) **Temperature** – Hypothermia

(11) **Metabolic acidosis** - (i) is of high anion gap type. Causes (a) formation of formic acid (b) respiratory depression (ii) Blood pH <7.0 indicates poor prognosis

(12) **Elevated osmolal gap**

(13) **Permanent sequelae in survivors** –

(i) Blindness [most common]

(ii) Extrapyramidal movement disorders –

Parkinsonism like syndrome [due to putamen damage]

(iii) Toxic encephalopathy

(iv) Neuropathy

4. Fatal dose

(1) 60-200 ml. Paradoxically larger doses, especially taken quickly may not kill, because of violet emesis [same as ethyl alcohol above]

(2) Blood levels of **200 mg%** are fatal

(3) Hemodialysis must begin when blood levels are **20 mg%** or above.

5. Fatal period

(1) 24-36 hours

(2) in some cases may be delayed up to 4 days.

6. Management

(1) **Gastric lavage** – with 5% bicarbonate solution. 500 mL may be left in the stomach

(2) **Emesis** – not recommended, because of potential for CNS depression

(3) **Activated charcoal** – Not of much use because of limited adsorption of methyl alcohol. Yet it may be given because it adsorbs other co-ingested

poisons

(4) **Specific antidote** is ethanol

(i) Mechanism of action - It competes with methyl alcohol for ADH [step 1 in **Fig 40.3**], and prevents conversion of methanol to more toxic formaldehyde. Methanol is excreted unchanged in the urine.

(ii) Mode of administration - (a) IV route – (I) Dose - **10% ethanol** [available as vials of pyrogen free absolute alcohol] in a dose of **10mL/kg**. Serum concentration to be maintained is **100 mg%**. (I) Dose – 1ml/kg of 95% ethanol in fruit juice [200 mL], over 30 min. For maintenance 0.2mL/kg/hr as 50% ethanol in fruit juice.

(5) **4-methyl pyrazole** [4MP, **Fomepizole**] -

(i) Competitive antagonist of ADH.

(ii) Advantages over ethanol - (a) reliably inhibits ADH (b) conc do not need to be monitored as with an ethanol (c) does not cause inebriation (d) associated with fewer side effects (e) does not require ICU monitoring. For these reasons, it has become the preferred method of ADH blockade, despite being significantly more expensive than ethanol.

(iii) Dose - (a) **15 mg/kg** IV as an initial loading dose (b) followed by **10 mg/kg** every 12 h. (c) After 48 h of therapy, fomepizole induces its own metabolism, so the dose must be -ed to **15 mg/kg** every 12 h.

(6) **Abacavir**.

7. Cause of death

(1) Mainly due to **acidosis** from production of organic acids [mainly formic acid]

(2) **Respiratory failure**

(3) **CNS depression** [minor factor].

8. Postmortem appearances

(1) **Cyanosis** – marked. Blue color of skin may also be due to methyl violet, a blue dye often present in methylated spirits

(2) **PM clotting of blood** – absent

(3) **Lungs** – congested, edematous

(4) **GIT** –

(i) Mucus membranes - of stomach and duodenum is congested, inflamed and

shows hemorrhages

(ii) Small and large intestines - both contracted. Resemble a thick pipe of very narrow lumen.

(5) **Liver** – early necrosis, fatty change.

(6) **Kidneys** – Acute tubular degeneration.

(7) **Urinary bladder** – mucosa congested.

(8) **Brain** – congested, edematous, shows hemorrhages.

(9) **H/P** – Retinal ganglion cells and optic disc show degenerative changes.

9. ML importance (of methyl alcohol)

(1) **Manner of poisoning** -

(i) Accidental - Mostly. (a) Consumption of spurious liquor [**hooch tragedy**] (b) due to its similar smell to white wine. (c) Villagers consume it when ethyl alcohol is not available, as by ingesting methylated spirit, which is available as fuel.

(ii) Suicidal – rare.

(iii) Homicidal – rare because of taste and odor, but can be mixed in ethyl alcohol and offered. Homicidal poisoning in children is known.

(2) **Consumption of solox** – Solox is the trade name for a shellac and paint solvent consisting primarily of ethyl alcohol and methanol. Victim may consume solox as a cheap alternative to alcohol.

(3) **Illicit liquor** – Illicit liquor, which is made without license and with poor infrastructure is called hooch, moonshine [because it was produced in night or during moon light], mountain dew, Tennessee white whiskey or white lightning. It is mostly methyl alcohol, which can cause accidental poisoning. Other unintentional adulterants which have been recorded are: **Heavy metals, eg arsenic and lead** – Arsenic is a contaminant of lead or solder used in the stills [apparatus used to distill illegal alcohol]. Thus persons who regularly consume illicit liquor may slowly develop arsenic or lead poisoning. Secondly since alcohol is produced by grain and grain is stacked near sills, rodents are attracted to the area of stills. To kill them rat poison containing arsenic is frequently used in close proximity to the grain and mash stores. Careless use of arsenic results in contamination of alcohol.

IV. OPIUM AND ITS DERIVATIVES

A. Opium

Opium (Afim, Poppy tears, Lachryma papaveris) is the coagulated juice of the opium poppy (Papaver somniferum) or any mixture containing it [***S2***(xv) NDPSA 1985]. **Powdered Opium** is opium dried at a temperature not exceeding 70°C, and reduced to a very fine powder. Powdered Opium yields between 10.0-10.5% of anhydrous morphine. **Endogenous opioid peptides** [dynorphins, Enkephalins, endorphins, endomorphins and nociceptin] are the naturally occurring ligands for opioid receptors. The term **endorphin** is used synonymously with endogenous opioid peptides but also refers to a specific endogenous opioid, **β-endorphin**.

1. Plant

a. Cultivation

Because the lab synthesis of morphine is difficult, morphine is still obtained from opium. It is cultivated in India and other Eastern countries. In India, it is mainly grown in MP, UP, and Rajasthan.

b. Licensing

In most countries including India, it can be grown only by license from the govt. In India, legal cultivation is carried out only in MP, Rajasthan and UP. Licenses are issued by the **Central Bureau of Narcotics** [CBN].

c. Extraction of raw opium

(1) **Opium** - comes from the unripe capsules of Papaver somniferum (Gk papaver, poppy; Somniferum, sleep), which is an annual herb belonging to family Papaveraceae

(2) **Flowers** - are white, red or purplish, depending on variety

(3) **Capsules** - One plant bears 5-8 capsules. Capsules ripen to about 4 cm in diameter; on ripening, their color changes from bluish-green to yellow.

(4) **Extraction of opium** – The color change from bluish-green to yellow signals the optimum time for latex collection. while the capsules are still attached to the plant, very shallow incisions are made into their wall (lancing) with a special nushtar having three or four small blades, separated by spaces of about

3 mm.

(5) **Poppy tears** - The latex which collects on the capsule walls is known as 'poppy tears' (**Fig 40.4**). On drying up, it becomes raw opium [Gk opos, juice].

(6) **Poppy seeds** -

(i) The capsule has several chambers (loculi) which contain thousands of white, tiny, kidney-shaped seeds known as khus khus. They are less than **1mm** in length, and very light. One capsule contains more than 1000 seeds. 3500 poppy seeds weigh about 1 gram.

(ii) Poppy seeds are non poisonous, demulcent and nutritive and used for flavoring food. Poppy seed bagels are common in the West.

(iii) They yield 45–50% oil (poppyseed oil), which is used for cooking purposes. Also contain traces of morphine [7-60µg/g] and codeine.

(iv) Poppy straw – Empty poppy capsule without the seeds (also opium straw, poppy chaff, poppy head, poppy pod or post ka doda).

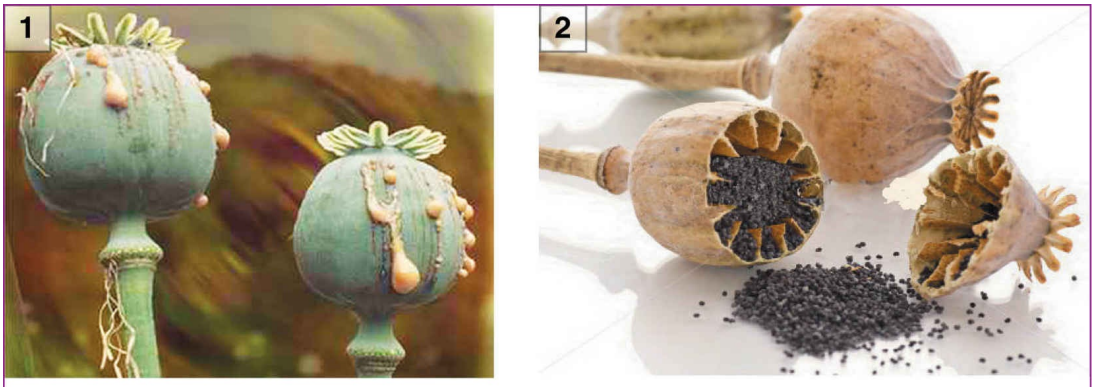


Fig. 40.4: (1) Poppy capsules with incisions over surface. Drops of latex (poppy tears) can be seen oozing out. On drying they would constitute raw opium **(2)** Poppy seeds (slate gray variety).

d. Raw opium

(1) **Physical characteristics** -

(i) Appearance - Opium appears as a more or less rounded, oval, brick-shaped or elongated, somewhat flattened mass, usually about 8-15 cm in diameter and weighing about 0.3-2 kg each.

(ii) Odor - Strong characteristic

(iii) Taste - bitter [due to alkaloids present]

(iv) Consistency - It tends to be plastic when fresh, but becomes more dense and tough on storage.

(2) **Alkaloids** – contains about **25 alkaloids**, combined with meconic, lactic and sulfuric acids. Chemically they form two groups (a) **Phenanthrenes** [morphine [Indian Opium yields approx 9.5-14%. The morphine content is standardized in the Govt opium factory at Ghazipur, to contain 10% morphine. This is known as **Standard** or **Standardized Opium**. The total alkaloids of this opium are 40%], codeine (0.5%) and thebaine (0.2%)] and (b) **Benzylisoquinolines** [noscapine (formerly called narcotine) (6%), papaverine (1%), narceine(0.2%)].

(3) **Terminology** – **Differences between opiates and opioids - Opiates** are drugs derived from opium, and include natural products morphine, codeine, thebaine and semisynthetic congeners derived from them eg heroin. **Opioi*d*** (Gk eidos, similar to) on the other hand – is a much broa***d***er term which refers to any agent that binds to opioid receptors. It is a more inclusive term applying to all agonists and antagonists with morphine-like activity. The term opioid would thus include completely synthetic products such as diphenoxylate, fentanyl, loperamide, methadone, pethidine (meperidine), propoxyphene, which are not included under opiates.

Memory Aid 12: How to differentiate opioids from opiates

Opioi **d** is a much broa**d**er term.

2. Classification of opioids

(1) **Natural** – morphine, codeine

(2) **Semi-synthetic** – Heroin, Pholcodeine. Many others like -Hydrocodone, Hydromorphone, Oxycodone and Oxymorphone are not used in India.

(3) **Synthetic** – Dextropropoxyphene, Diphenoxylate, Fentanyl, Methadone, Paregoric, pethidine [Meperidine], propoxyphene, Tramadol.

3. Absorption, distribution, fate and excretion

(1) **Opioids** -

(i) Absorbed from the GI tract and through rectal mucosa.

(ii) Placental barrier - Most opiates cross placenta, and appear in fetal circulation within 5 min following maternal IV injection.

(2) **Morphine** -

(i) There is significant first-pass metabolism in the liver, because of which oral

routes are less effective than parenteral.

(ii) Half life of morphine - in plasma is **2 hours**.

(iii) Elimination of morphine - (a) is by glomerular filtration. (b) **Enterohepatic circulation** of morphine and its glucuronides occurs, which accounts for the presence of small amounts of morphine in feces and urine for **several days** after the last dose.

(3) **Codeine** - and its analogs [levorphanol, oxycodone, methadone] have a lower first-pass metabolism in the liver.

(4) **Heroin** (diacetylmorphine) - is rapidly hydrolyzed to 6-monoacetylmorphine (6-MAM), which in turn is hydrolyzed to morphine.

4. Mechanism of action

Opium acts through opioid receptors, which are a group of **G protein-coupled receptors** (**GPCRs**). These are mainly located in the CNS.

5. Acute poisoning

a. Signs and symptoms

i. On contact

(1) If a person is sensitive to opium, he may experience

(2) Erythema

(3) Itching dermatitis

(4) Urticaria.

ii. Ingestion

Symptoms begin within half an hour upon oral ingestion. It is customary to study the symptoms as occurring in 3 stages.

(a) Stage of excitement [1st stage]

(1) **Duration** – Very short. This stage may even be absent, if a large does is taken

(2) **Euphoric** Symptoms - -ed mental activity, -ed sense of well-being, Euphoria, Freedom from anxiety, Talkativeness

(3) **Dysphoric Symptoms** – Seen sometimes. Excitement, Flushing of face, Hallucinations, Maniacal condition and Restlessness

(4) **Seizures** – Seen especially in neonates.
(i) May cause rhabdomyolysis, hyperkalemia and ARF.
(ii) Mechanism - Hypoxia.
(5) **Signs** –
(i) Odor of opium + [in all stages]
(ii) Pulse rate -ed
(6) **In children** – Convulsions may be seen.

(b) Stage of stupor [2nd stage]

(1) **Symptoms** –
(i) **Initially -** Drowsiness, feeling of heaviness in the limbs, giddiness, headache, incapacity for exertion, itching sensation all over the body [through release of histamine], nausea and vomiting [through stimulation of CTZ].
(ii) **Later** – Uncontrollable desire to sleep. The patient lies motionless with eyes closed. Initially he can be roused, but later goes into stupor.
(2) **Signs** -
(i) Pupils - contracted
(ii) Conjunctiva - congested
(iii) Face and lips - cyanosed
(iv) Pulse and respiration normal.

(c) Stage of narcosis or coma [3rd stage]

(1) **CNS depression** -
(i) Patient passes into coma from which he cannot be roused.
(ii) All reflexes lost
(2) **Signs** -
(i) BP., RR. [3-4/min], stertorous breathing [bronchospasm via histamine release], Gurgling [accumulation of pulmonary edema fluid], Temp.
(ii) Conjunctiva congested
(iii) Face - pale
(iv) Pupils - (a) Pin point [most characteristic]. (b) Do not react to light (c) During last agonal phase, they dilate. (d) The ***classic triad*** of opioid toxicity is miosis, respiratory depression, and CNS depression (coma)
(v) Pulse – slow
(vi) Secretions - abolished, except sweat
(vii) Skin - cold and clammy [covered with perspiration]

(viii) Muscles - flaccid and relaxed
(ix) Gastric emptying – delayed due to coma
(3) **In cases of fatality** –
(i) Lividity of the body -s.
(ii) Pulse becomes slow, irregular and imperceptible,
(iii) Respiration becomes Cheyne-Stokes in type.
(iv) Finally death occurs from asphyxia.

b. D/d

Look for following 7 characteristic signs in opium poisoning: (i) **Pupils** – pinpoint, not reacting to light (ii) **R/R.,** (iii) **pulse.,** (iv) **temp**. (v) **Odor** – characteristic (vi) **Skin** – moist, perspiring (vii) **Coma.** None of the other conditions will show all seven. **D/d** is as follows.

(1) **Alcoholic poisoning** [acute] – (i) breathing – slow, stertorous (ii) eyes - congested (iii) face - hyperemic (iv) pupils – dilated; reacting to light (v) odor - of alcohol (vi) Temp – subnormal

(2) **Barbiturate** – (i) BP - . (ii) Coma – deep (iii) Deep reflexes – depressed (iv) Pulse – thready (v) Pupils – dilated (vi) Respiration – shallow (vii) Response to painful stimuli – diminished or none (viii) Temp – subnormal

(3) **Carbolic acid** – (i) Lips and mouth – show white patches (ii) Odor – phenolic (iii) Urine – greenish black [carboluria]

(4) **Cerebral hemorrhage** - (i) Onset - sudden (ii) History - of hypertension (iii) Muscular – Paralysis, usually hemiplegia (iv) Old age (v) Plantars – bilateral extensor (vi) Pulse – slow, full (vii) Pupils – dilated (viii) Respiration – Cheyne-Stokes (ix) Temp -

(5) **Cerebral malaria** – (i) Fever with chills and rigors (ii) Hepatosplenomegaly (iii) MP+

(6) **Cerebral trauma** – (i) History – of head injury (ii) Bleeding – from nose, mouth, ears (iii) Cranial nerves – dysfunction (iv) Pulse – initially rapid; later slow (v) Pupils – contracted, unreactive to light, sometimes unequal (vi) Respiration – Cheyne-Stokes, irregular, rapid, (vii) Subconjunctival hemorrhages

(7) **CO poisoning** - (i) History – of exposure to gas [eg sleeping in a closed room with fire] (ii) Skin color – cherry red (iii) Convulsions (iv) Blood examination – shows COHb

(8) **Coma [Diabetic]** - (i) Onset – Gradual (ii) Breath – acetone odor (iii) Face-flushed (iv) Intraocular pressure - . (v) Respirations – slow and deep (vi) Temp – subnormal (vii) Urine – contains sugar and acetone

(9) **Coma [Epileptic]** - (i) Characteristic epileptiform seizures (ii) Face and lips – cyanosis (iii) Mouth and nostrils – show froth (iv) Tongue – bitten (v) Pupils – fixed and dilated (vi) Respiration – slow (vii) Skin – flushed (viii) Recovery from coma – rapid

(10) **Coma [Hysterical]** - (i) More common in – females (ii) Previous history – of convulsions (iii) Attitude – unusual (iv) Reflexes – normal (v) Usually occurs – in presence of audience

(11) **Coma [Uremic]** - (i) Onset – gradual (ii) General anasarca (iii) Face – pale (iv) Breathing – Cheyne-Stokes (v) Odor – ammoniacal (vi) Urine – shows albumin, blood casts

(12) **Encephalitis** - (i) Onset – acute (ii) Involuntary movements + (iii) Ocular palsies + (iv) Temp - - (v) CSF – specific changes

(13) **Heatstroke** - (i) History of – prolonged exposure to high temperature or sun (ii) Conjunctivae – congested (iii) Skin – dry (iv) Sweating – absent (v) Temp - - (vi) Circulatory collapse, convulsions

(14) **Meningitis** - (i) Onset – gradual (ii) Signs of – meningeal irritation (iii) Temp - - (iv) CSF – specific changes.

c. Tests

Marquis' test - Suspect residue + 3 cc of Marquis Reagent [prepared by dissolving 5 ml of 40% formaldehyde in 100 ml of conc H_2SO_4]"(i) If morphine present, it gives a fine purple-red color, changing to violet and then to blue. (ii) Amphetamine - gives a yellow–orange color.

d. Fatal dose

Table 5: Fatal doses of some common opioids [in comparison with their therapeutic doses]

S.No	Opiate	Usual therapeutic dose	Usual fatal dose
1.	Heroin	-	50 mg
2.	Methadone	5-10 mg	100 mg
3.	Morphine	10-15 mg	200 mg
4.	Codeine	10-60 mg	800 mg
5.	Pethidine	50-150 mg	1 g
6.	Raw Opium	-	2 g

e. Fatal period

6-12 hours.

f. Management

(1) **ABCDE of resuscitation** - Ventilation support, oxygenation[ch 32].

(2) **Decontamination** –

(i) Emesis - induced by ipecac is not recommended, because of potential for CNS depression and seizures

(ii) Gastric lavage – with $KMnO_4$. Useful even if opioids are taken parenterally because of enterohepatic circulation. For the same reason, leave about 250-300 cc of $KMnO_4$ after lavage is over [**ch 32** for details].

(3) **Evacuation of intestines** – to prevent reabsorption (i) sodium sulphate orally 30 g (ii) enema twice daily for 2 days

(4) Activated charcoal [**ch 32**]

(5) **Antidotes** –

(i) Naloxone - (a) Routes - (I) IV [most preferred]. May also be given (II) sublingually, (III) intramuscularly, (IV) intranasally or (V) instilled down an endotracheal tube. Since repeated doses are required, naloxone infusion is better. (b) Dose - 1.2 mg [adult]; 0.4 mg [child]. Repeated till a total of 75 mg in 24 hours is given. (c) Dangers - (I) Reversal of a narcotic coma can

precipitate sudden narcotic withdrawal and may cause the patient to behave dangerously towards himself and staff. (II) Neonates of addicted mothers may have severe withdrawal effects. (III) Anticholinergic crisis - if ingested narcotic was contaminated with scopolamine [please see below – "additives of Heroin"].

(ii) Naltrexone - Orally. Dose 50 mg/day. Also used to treat opiate addiction.

(iii) Nalmefene - Naltrexone derivative. Pure opiate antagonistic effects. (a) Route - IV most common. Also IM or S/c. (b) Dose - Begin with 0.1 mg. If withdrawal reaction does not occur, administer 0.5 mg, followed by 1 mg in 2-5 minutes if necessary.

(iv) Physostigmine – 0.04mg/kg IV for reversing respiratory depression, if regular opiate antidotes [naloxone] are not available. Mechanism – it -es Ach content of reticular formation of brainstem which is suppressed by opiates.

(v) Antidotes and other therapies no more recommended - (a) Amiphenazole (b) Analeptics – eg amphetamine, caffeine, ephedrine (c) Atropine – Once a favorite antidote because most pharmacological actions of the two drugs are antagonistic. Atropine stimulates respiration; morphine depresses it. Atropine dilates pupil; morphine contracts it. Atropine -es body temp, and reddens skin; morphine .es body temp and produces pallor. Atropine .es body secretions; morphine -es them. Thus it intuitively appeared to be a good antidote. However it is no more recommended because it can cause death by paralyzing the motor and sensory nerves just like morphine. (d) Levallorphan [Levorphanol] (e) Nalorphine (f) Once it was common to ask patients to walk about in the open air to help excretion. No more recommended.

(6) **Rhabdomyolysis induced ARF** –

(i) Aggressive fluid replacement

(ii) Diuretics – furosemide, mannitol to maintain urine output.

(7) **Coma cocktail** – only when identity of poison not known [**ch 32**].

(8) **Immunotherapy** - Vaccines that would generate antibodies to sequester the drug and prevent its access to the brain. Currently under development for 4 major drugs of abuse – cocaine, methamphetamine, morphine/heroin and nicotine.

(9) **Symptomatic treatment** -

(i) Convulsions – Benzodiazepines 5-10 mg stat; repeat every 5-10 min if needed. Generally not needed if naloxone is available.
(ii) Hypotension – **ch 32**.

g. Cause of death

Respiratory failure.

h. PM appearances

(1) PM Staining – well marked, cyanotic
(2) Smell – of opium from corpse. More noticeable as abdomen is opened. Not felt if body is putrefied
(3) Signs of asphyxia (i) Face and nails-cyanosed
(4) Mouth – Froth
(5) Trachea, bronchi – congested, full of froth, inhalation of vomit
(6) Lungs – edematous, congested
(7) Stomach – lumps of opium
(8) Brain, meninges - congested
(9) All viscera – congested
(10) Blood – dark and fluid.

6. Chronic poisoning

Chronic poisoning [morphinism, morphinomania] is seen in addicts after a long period of usage.

Salient features:

(1) Habit is acquired by young people as morphine is considered an aphrodisiac
(2) Addicts can tolerate 3-6 g/d.
(3) This section deals with chronic poisoning due to oral [classical] use [opium eaters]. More modern use is to take injections of heroin [please see below].

a. Signs and symptoms

(1) **General** – (i) Emaciated (ii) disturbed sleep, insomnia (iii) Restlessness and irritability [with periods of disinterest and depression in between] (iv) weak
(2) **Pupils** - contracted
(3) **Tongue** – dry, furred
(4) **GIT** – Anorexia, nausea, marked constipation

(5) **Immune system** – is compromised.

(i) Innate immunity and adaptive immunity - both depressed. (a) .es proliferative capacity of macrophage progenitor cells and lymphocytes. (b) functioning of immune cells is compromised

(ii) Mechanism - (a) direct action of opioids on immune cells. immune cells differentially express classical opioid receptors. These binding sites mediate the antiproliferative effects of morphine. (b) indirect centrally mediated pathways.

(6) **CNS** – (i) Dementia or mania (ii) hallucinations (iii) intellectual and moral deterioration (iv) Loss of memory (v) mental fatigue (vi) neurasthenia

(7) **Sexual**- impotence.

(8) **Skin** –(i) pigmentation around mouth and eyelids (ii) generalized pruritus.

b. Management

Please see under Heroin.

c. PM appearances

Same as that of heroin [please see below]. Only appearances related to IV injection would be missing.

7. ML importance (of opium)

(1) **Ideal suicidal poison** – because death is painless. In order to commit suicide, opium is usually mixed with mustard oil or asafetida [Hing in Hindi] under the mistaken notion that these substances - the absorptive power of opium. However their presence does make it more difficult to remove opium by gastric lavage.

(2) **Homicidal poison** – Used only rarely, because of bitter taste, characteristic smell and dark brown color.

(3) **Euthanasia** – Morphine is one of the favored drugs for euthanasia [**ch 2**] [also barbiturates; please see below]. Many believe that the British general practitioner **John Bodkin Adams** [1899 –1983] euthanized his patients with morphine [see case studies below].

(4) **Accidental poisoning** –

(i) Occurs in addicts

(ii) Children – can be accidentally poisoned (a) when they accidentally swallow

crude opium or opium pills meant for their addict parents (b) Parents – generally of the labor class – usually drug their children intentionally so they can sleep while they go to work (c) In olden times, Ayahs [ladies hired for baby-sitting] were known to smear their nipples with opium mixed with honey, and allow child to suckle their breasts, so the child could go to sleep.

(5) **Persons extraordinarily prone to opium poisoning** – children, adults suffering from

(i) Addison's disease

(ii) Asthma [morphine causes histamine release]

(iii) Ch liver disease

(iv) Myxedema.

(6) **Cattle poison** – rarely

(7) For **doping race horses**

(8) -ed Arsenic levels are found in opium eaters [please see **ch 36** – MLI of arsenic].

(9) Consumed before undertaking **acts of courage**

(10) Opium frequently smuggled in condoms which are swallowed [please see **ch 46** – Body packer syndrome]. May cause death if condoms rupture in GIT.

(11) **Opium habit** – Opium is usually taken by mouth but may be taken in some unconventional ways

(i) Kasoomba [poppy tea] – A decoction made by boiling opium poppies in water, and consumed on festive occasions. Has mild euphoriant properties. Used for psychoactive effects, but also as a herbal remedy for minor ailments. [please see ch 41 for "cannabis tea"]

(ii) Bhujri - A preparation made by frying green ripe capsules in butter or ghee (clarified butter) is eaten by addicts

(iii) Halwa - A sweet prepared from the juice of green poppy capsules is also consumed by addicts.

(iv) Chandu, Madak or Opium dross – Smokable form of opium

B. Heroin (Diacetylmorphine)

Heroin (3,6-diacetylmorphine, diamorphine, morphine diacetate), is a semi-synthetic opioid drug synthesized from morphine.

Salient features:

(1) **Chemistry** - It is the 3,6-diacetyl ester of morphine [di-acetyl-morphine] and a morphine prodrug. Can be prepared by mixing morphine with acetic anhydride [$(CH_3CO)_2O$].

(2) **Potency** - Administered orally, heroin is 1.5 times more potent than morphine. Parenterally it is 2-4 times more potent. The enhanced euphorigenic effect is called "**rush**".

(3) **Metabolism** – Metabolized rapidly to 6-monoacetylmorphine, and then to morphine. Chemical analysis will reveal morphine and 6-monoacetylmorphine but not heroin. Half life of heroin is **38 min**.

(4) **Reason for higher potency** - Heroin has a lower affinity for the μ receptor than does morphine, yet is more potent, because

(i) Its metabolite 6-monoacetylmorphine, is a more potent μ agonist than morphine.

(ii) Enhanced blood-brain barrier penetration.

1. Forms of heroin

Heroin is available in **2** distinct chemical forms: salt [hydrochloride] or base [pure alkaloid].

(1) The hydrochloride salt – is typically a white powder. It was the common form of heroin available prior to the 1980s. Its high water solubility allows IV and IM administration.

(i) IV administration - is called mainlining, and users are known as mainliners. Because the needles used are contaminated, the user gets needle track ulcers.

(ii) IM or SC administration - If the user cannot locate a vein, he would lift the skin and inject subcutaneously or IM [Skin popping]. Such users get map shaped ulcers [geographical ulcers]

(2) Alkaloidal base – is currently the more prevalent form of heroin. Its color is brown [brown sugar] or black [black tar heroin].

(i) Brown sugar - acquires a brown color because heroin may be mixed with coffee powder, coco, jaggery [gur] or even brick powder by some dealers.

(ii) Black tar - refers to an impure South American variety. It is black, heat stable, insoluble in water, rubber-like. Usually produced in provisional factories in the vicinity of poppy fields and is thus often contaminated by spores. Drug abusers avoid to inject it IV because it causes sclerosis. S/c

injection causes spores to gain access to the s/c tissues"They multiply there due to the anaerobic environment and produce neurotoxins which inhibit the release of Ach at the NMJ"respiratory paralysis.

2. Additives [of street drugs]

Additives of heroin

Heroin mixed with additives is known as dope, junk or smack. Common additives are

(1) Quinine -

(i) Mimics the bitter taste of heroin [misleads clients].

(ii) Supposed to prevent malarial transmission due to shared needle use. First used in 1930s in New York City when an epidemic of malaria among IV heroin users was common.

(iii) Quinine adulteration currently is much less important than it was in the past.

(2) Scopolamine – Was commonly used in the northeastern US in 1995. Normally symptoms of scopolamine remain unmasked due to heroin. Administration of naloxone causes reversal of the opioid effects " unmasking of the effects of scopolamine " anticholinergic crisis.

(3) Strychnine -

(i) Mimics the bitter taste [as quinine]

(ii) May cause sudden death if quantity of strychnine is too much. The needle may still be in the vein [**accidental hot shot**]. Also seen when the addict gets a purer sample than the one he has been using. Another situation is when an addict is given a deliberate overdose, and then either smothered, strangled or drowned in the bath [**homicidal hot shot**].

(4) Others - Acetaminophen, amphetamines, caffeine, cocaine, lactose, lead, mannitol, methaqualone, phenobarbital and thallium. Thallium is a common component of traditional depilatory powders which are used to - the wt of heroin.

3. Combinations

Speedball - Refers to the hazardous IV use of **heroin** and **cocaine** [or **amphetamine**] together. The combination when smoked is known as **moonrocks**.

4. Administration

a. Hydrochloride salt

This is usually injected.

(1) **The works** – refers to the IV apparatus used by narcotic addicts [please see **ch 46** for diagram].

(2) **Typical heroin bag** – contains 100 mg of white or brownish powder [1-15 mg heroin + 0-40 mg quinine + various other additives mentioned above]. If the addict has been using a bag containing, say, 1 mg of heroin, and on one occasion he gets a purer brand, say containing 15 mg, he may die suddenly [hot shot; please see above also].

(3) **Cooker** – Bottle top or spoon.

(i) Contents of envelop are emptied in it, water is added and heated by a match till the powder dissolves.

(ii) Solution is drawn through a piece of cotton [sometimes obtained from the tongue of a shoe].

(iii) The belief is that cotton absorbs impurities and germs, making the solution sterile. [it actually causes "**cotton fever**". Please see below]

(iv) Scene of crime examination [**ch 46**] often reveals cooker with recent soot underneath its surface [due to heating].

(4) **Syringe** – The homemade eyedropper syringe is preferred to traditional medicinal syringe, because it is more easily manipulated with one hand.

(5) **Tourniquet** – belts, elastic bands or shoe laces are used as tourniquets. The needle is inserted into the vein [mainlining]. Skin poppers do not use tourniquet.

(6) **Booting** – Once the needle is in the vein, the addict pushes the drug and then draws the blood up. The process is repeated several times in order to fully inject the drug. This is known as booting.

b. Alkaloidal base

(1) Chasing the dragon - Because of its heat stability it can be smoked. Addicts inhale a thick, white **pyrolysate** that is generated by heating heroin base on an aluminum foil using a hand-held flame. Although there is some wastage [some of the heroin effluvium is dissipated into the surroundings], it is still preferred

because it does away with injections, which can cause infections and leave tell-tale signs on arms. Sometimes it is mixed with caffeine or barbiturates; this improves the sublimation of heroin and enhances the yield.

(2) IV usage – is difficult, because of alkaloid's insolubility in water. Possible in 2 ways - heating the heroin until it liquefies or mixing it with acid.

5. Metabolism

Please see above under the heading "Opium".

6. Signs and symptoms

Heroin is rarely – if ever – taken by mouth. Signs and symptoms are same as in opium, but due to IV administration, all stages seem to merge in one.

a. Acute

(1) Intense euphoria, which lasts for several minutes

(2) followed by sedation for about 1 hour

(3) Effects completely lost in 3-6 hours.

(4) There may be systemic reaction with fever and chills after injection ('**dirty hits**').

(5) **spongiform leukoencephalopathy** - caused by inhalation of heroin pyrolysate.

(6) **Allergic skin reactions** – due to allergens in the drug sample. Angioedema, bullous drug reaction, Eczemas, maculopapular exanthema, urticaria, vasculitides.

b. Chronic

(1) **General** – Emaciation [lack of nutrition]

(2) **GIT** - (i) Anorexia (ii) Constipation

(3) **Sexual** - Impotency

(4) **Signs associated with use of infected needles** -

(i) Systemic infections - hepatitis, HIV, malaria.

(ii) Muscles – (a) myopathy. (b) Partly due to chronic infection, but is exacerbated by an auto-immune response to damaged muscle. (c) H/p – affected areas show fibre necrosis, replacement fibrosis and infiltration by lymphocytes and polymorphs, which extends far beyond the area of

injection.

(iii) Region lymph nodes - enlarged

(iv) Cutaneous infections - (a) abscesses, erysipelas, phlegmons [walled off inflammatory mass with or without pus that is palpable on physical examination]. Pathogens involved belong to body's own flora [S. aureus, streptococci species]; often multibacterial infections; rarely gram-negative bacteria, anaerobes. (b) Anthrax - Rare after i.v. injections; caused by Bacillus anthracis. (c) Necrotizing fasciitis - Rare after "skin popping" (d) Nodes, pustules caused by Candida albicans (e) Wound botulism - after "skin popping", -ed risk with black tar heroin. Caused by Clostridium botulinum.

(5) **Skin** –

(i) Injection marks – Most common injection site is the antecubital fossa followed by forearms and back of hands. As these sites get scleroses other sites are chosen eg neck, groin, ankles and penis

(ii) Some fingers and toes may have been lost due to vascular damage

(iii) Dermal abscesses

(iv) Dry, postinflammatory hyperpigmentation along sclerosed vessels.

(v) Ulceration - (a) geographical ulcers, needle track ulcers, skin tracks, track marks. (b) Site - on scalp, neck, sublingual areas, shoulder, inguinal region, penis, vagina, popliteal area, ankle and foot. May be on unusual sites eg inner surface of lip, beneath the tongue or beneath penile foreskin. (c) Healing by fibrosis – may produce hyperpigmented macules or circumscribed, retracted scars which resemble those from smallpox vaccinations. May be atrophic, sunken, translucent [tissue paper scars]. Microscopic examination of scar tissue shows – foreign material eg fragments of cloth, cotton, talc or unidentifiable matter with surrounding foreign body giant cell reactions.

(vi) Signs due to addict's attempts to hide needle puncture marks from law enforcement authorities - (a) Elaborate tattoos (b) cigarette burns (c) damage to skin caused by abrading it with pumice stone or sandpaper or by using escharotic chemicals

(vii) Prurigo nodules, excoriations.

(viii) Hematoma, necroses - due to wound healing disturbances

(ix) Punctures, petechiae - Below the tourniquet.

(x) Shooting tattoo – Punctate areas of black discoloration [soot tattooing]. Due to foreign material introduced through injection.

(xi) Scabies, pediculosis capitis/corporis, tinea, impetigo contagiosa - Due to secondary neglect, homelessness.

(6) **Hands** - Chronic edema, secondary to occlusive thrombophlebitis in the forearms, lymphatic insufficiency and direct toxicity of injected drugs [**Puffy hand syndrome**]. Arm and forearm edema are voluminous and may cause functional disturbances.

(7) **Pulmonary hypertension** – due to excessive microcrystalline pulmonary emboli.

7. Tolerance

Occurs very rapidly (within days). Dose may have to be increased more than 100 times the original.

8. Complications

These are general complications of drug dependence, where drug is injected through unsterile equipment

(1) Abscess formation

(2) endocarditis

(3) Infections transmitted through needles [AIDS, hepatitis, malaria]

(4) Pigmented scars at sites of injection

(5) **Veins** – in arms, hands, legs etc are damaged and thrombosed. Show phlebitis. Become dark in color, hard and cord like due to thrombosis and fibrosis, and may ulcerate. Upon healing, show white or silvery linear scars

(6) septicemia

(7) **Due to general apathy and self neglect** - (i) Dental decay (ii) Malnutrition (iii) Infections – pneumonia, tuberculosis

(8) **Renal** –

(i) Amyloidosis - associated with s/c drug injection, and chronic soft tissue infection [**'skin poppers' amyloidosis**]. It particularly affects African-Americans

(ii) Heroin associated nephropathy - characterized by nephrotic syndrome and rapid progression to renal failure, proteinuria. H/P - focal segmental glomerulosclerosis.

(iii) Renal disease related to HIV, HBV and HCV

(iv) hepatitis-C-related membranoproliferative glomerulonephritis (MPGN).

(9) **Cotton fever** -

(i) It is a febrile condition due to inadvertent injection of cotton fibers in their veins by drug addicts.

(ii) Etiology - (a) It is due to the practice of addicts putting cotton in their drug solutions to remove germs [please see above under the heading "method of injection"]. (b) Also because many addicts use heroin reclaimed from previously used cotton filters. Enterobacter agglomerans [a bacterium] thrives in cotton fibers; enters bloodstream " fever.

(iii) Symptoms - (a) fever, (b) chills [violent shaking or shivering – thus cotton fever also known as "the shakes"], and (c) shortness of breath. Occur immediately after injection or within an hour.

(iv) Prognosis - good.

(10) **Drug abuser's elbow** - Myositis ossificans develops in drug abusers, caused by repeated, inept needle punctures. The muscle around the elbow is replaced with scar tissue or with a calcific mass (extraosseous metaplasia).

(11) **Foreign-body embolism** - Talcum powder embolism.

(12) Psychiatric complications

(13) **Involvement in accidents** – is common due to .ed alertness

(14) **Need for money** – leads to crime, theft, prostitution, living in squalor

(15) Personal violence and murder

(16) **Clostridial Infections** – Uncommon. Present as wound botulism or tetanus.

(17) **Death** – due to hot shot [please see above], hypersensitivity to heroin or any of its adulterants.

9. Management

a. Acute intoxication

(1) **Decontamination** – Gastric lavage [as in opium above] even if drug was administered IV, because of enterohepatic circulation of all opioids coupled with duodenogastric reflux. In fact in IV administration, stomach concentration of drug is often ***higher*** than that in blood.

(2) **Coenzyme Q** – 300 mg qds, for spongiform leukoencephalopathy

(3) Rest same as in opium.

b. Chronic intoxication

Detoxification is the primary aim. This is a mode of treatment in which the dependent person is "taken off" heroin. Detoxification should only the done under expert guidance of a specialist. Paradoxically **two contrasting approaches** are followed – one in which agonists are given [substitution drugs, eg methadone], and the other in which antagonists are used [eg naltrexone]. Both are so-called **conventional detoxification** methods and both require between 3-21 days for completion of treatment. In contrast **anesthesia-assisted opiate detoxification** is completed within 4-6-hours.

i. Conventional detoxification methods

(a) Use of substitution drugs [*Opioid replacement therapy* (*ORT*)]

(1) **Principle** -

(i) Replaces an illegal opioid drug [eg heroin] with a longer acting but less euphoric opioid taken under medical supervision. As the treatment completes, the addict does not experience withdrawal symptoms and drug cravings.

(ii) Results - (a) while receiving opioid maintenance therapy - 40-65% of patients are able to maintain complete abstinence from opioids. 70% to 95% are able to reduce their use significantly. (b) After discontinuing maintenance therapy - About 7% patients remain abstinent from opioids for 90 days after discontinuing maintenance therapy. <2.5% patients remain abstinent from opioids for 1 year.

(2) **Drugs** –

(i) Methadone – Synthetic μ receptor agonist (a) Dose - 40 mg daily. Prevents withdrawal symptoms. 80 mg in chronic addict (b) dose must be .ed gradually by 20% daily. If signs of withdrawal reappear, dose reduction is slowed down [Methadone Maintenance Treatment Program (**MMTP**)]. There are concerns that by this therapy heroin addict will ultimately become methadone addict, but this is not entirely true. Reasons are (1st)Methadone is less addicting (2nd)Has a longer half-life (3rd).s possible criminal behavior [which is generally associated with procurement of drug] (4th) Has a much milder withdrawal syndrome. (c) Although often successful, some methadone users continue to use heroin or other illicit

substances. (d) It was not available in India for a long time, but since **2012**, it has become available.

(ii) Buprenorphine (Subutex) - Partial μ agonist. Produces a "generalized feeling of contentment" rather than a heroin-like rush and euphoria. Withdrawal syndrome from buprenorphine is milder. It is a gentle agent for use in rapid opioid detoxification. (a) **Dose** – 8-16 mg sublingually [because of its poor oral bioavailability]. (b) **Initial dose** - Because it competes with the extant opioid for the μ receptor, it may produce acute opioid withdrawal. For this reason the initial dose must be administered in the presence of a physician. (c) **Subsequent doses** - Following the initial doses of buprenorphine, tablets containing both buprenorphine and naloxone (Suboxone) are prescribed.

(iii) LAAM [**L**evo-**α**-**a**cetyl**m**ethadol] [syn levomethadyl acetate] [trade name, **Orlaam**] - It is a synthetic opioid similar in structure to methadone. Approved in 1993 by the U.S. Food and Drug Administration for use in the treatment of opioid dependence. It is indicated as a second-line treatment for the management of opioid dependence if patient fails to respond to drugs like methadone or buprenorphine. Not available in India.

Fig. 40.5: A bottle of Methadone

(b) Opioid antagonists

Most preferred is

(1) Naltrexone – Orally effective, potent. Dose – 100 mg on Mondays and Wednesdays. 150 mg on Fridays. Combined with clonidine, it is a very effective method of treatment. Less preferred are

(2) Nalorphine – partial antagonist

(3) Naloxone – has to be administered parenterally.

ii. Other methods

Clonidine [Catapres]- (i) Dose - 0.3-1.2 mg/d orally. Tapered off in 10-14 days. Can be started after the stoppage of opioid itself or the substitution drugs (methadone etc).

10. Fatal dose

50 mg.

11. PM appearances

Autopsy must be performed with full precautions as the body may be having hepatitis B and AIDS.

a. External

(1) **General appearances:** signs of wasting and self-neglect - May be dirty, emaciated, unkempt, unwashed for days

(2) Needle, syringe and tourniquet may be found on the body if death occurred due to hot shot.

(3) **Hidden drugs** - They may be stuffed in rectum or vagina, or may be taped to be body under the breasts, between buttocks and toes, or attached to a string tied around a tooth and then swallowed.

(4) **Findings associated with use of infected needles** – please see above under Heroin"signs and symptoms of ch poisoning.

(5) **Findings related to attempts at revival** – If an addict has “passed out” during or after drug injection, his colleagues try to revive him by peculiar and specialized methods popular only within the drug community.

(i) Pouring milk in mouth – milk may be found in oral cavity, esophagus etc

(ii) Injection of milk, saline or water into the arm, back of hands or buttocks – needle marks may be found at these places with traces of milk etc in s/c tissues

(iii) Immersion - in cold shower or tub of water

(iv) Putting ice - on genitalia

(6) **Mouth and nostrils:** Froth [due to pulm edema]

(7) **Skin, Hands** – Same findings as seen in ch poisoning with heroin [please see above]

(8) **Needle puncture mark** – on the skin must be searched for. May be seen as a

tiny hemorrhagic focus. Incision through the skin may show a perivenous hemorrhagic track.

b. Internal

(1) **Upper respiratory tract** – contains froth

(2) **Examination of upper limb veins** - for phlebitis, phlebosclerosis, thrombosis and recent and resolving perivenous hemorrhage [**ch 5**]. Preserve vein and surrounding tissues for chemical analysis.

(3) **Lungs** –

(i) Heavy, congested, edematous with focal hemorrhages

(ii) Texture – gritty, multinodular

(iii) Pleurae – may show petechial hemorrhages

(iv) Non specific pulmonary triad of (a) Severe pulmonary edema [due to sudden ventricular dysrhythmia] (b) bronchopneumonia and (c) aspiration of gastric contents

(v) Microscopically – Foreign body [eg talcum powder, other adulterants] granulomas. They may erode capillary walls and unite forming larger granulomas. Under polarized light large quantities of cellulose, starch and talc can be seen.

(4) **Heart** -

(i) Myocardial damage

(ii) Valvular diseases

(iii) Right ventricular hypertrophy – due to pulmonary hypertension, in turn caused by extensive pulmonary emboli.

(iv) Microscopically – Due to prolonged hypoxic coma following heroin intoxication (a) Degenerated, necrotic myocardial fibres (b) small mononuclear inflammatory cells

(5) **Effusions** – in all major body cavities – pericardial, pleural, peritoneal

(6) **Liver** –

(i) Congested, enlarged, may show evidence of cirrhosis

(ii) Enlargement of hepatic lymph nodes – may measure up to 3-4 cm each

(iii) Chemical analysis of LN reveals its metabolites 6-MAM and morphine

(iv) H/P – Portal LN show nonspecific hyperplasia. chronic Triaditis, Dense lymphocytic and mononuclear infiltrates involve all portal triads with or without parenchymal pathologic stigmas of viral hepatitis

(7) **Reticuloendothelial system** – hyperplastic changes, lymph nodes show large germinal centers [most common finding]
(8) **Spleen** –
(i) Enlarged due to malaria [transmitted through infected needles],
(ii) large germinal centers [most common finding],
(iii) presence of birefringent material
(iv) Shows lysozyme containing cells [indicate bacterial contamination]
(v) Shows IgE and IgM containing cells [indicates antigen stimulation]
(9) **Brain** -
(i) Cerebral edema
(ii) Focal areas of necrosis, involving globus pallidus and hippocampus due to hypoxia
(iii) Spongiform degeneration of white matter
(iv) Fluid accumulation in myelin sheaths
(10) **Associated findings** – infections transmitted through contaminated needles, eg hepatitis B, AIDS.

12. Samples to be preserved

(1) Tissue from inj site. Can demonstrate morphine as well as additives eg talc or starch
(2) **Blood** -
(i) Bilirubin level may be - [Hepatitis].
(ii) Tests for Australia antigen, HIV may be +ve
(iii) Blood film may show malarial parasites.
(3) Samples of urine, bile, liver, kidneys, lungs and brain
(4) Hair, bone and bone marrow can show opioids in completely putrefied bodies or exhumation after several months.
(5) Empty syringes and other paraphernalia. Drug packets full as well as empty.
(6) Common methods of detection are – GLC, TLC and spectrofluorophotometry.

13. ML aspects [of heroin]

(1) Same as ML aspects of drug abuse [**ch 46**].
(2) **Determination of route of administration** – may have to be determined in many cases.

V. SEDATIVE-HYPNOTICS

A. Barbiturates

Barbiturates are white, crystalline, odorless powders, with a faintly bitter taste.

Salient features:

(1) Barbiturates are derivatives of barbituric acid, which itself has no CNS depressant properties.

(2) **Barbital** became the first commercially available barbiturate in 1903.

(3) They were popular sedatives and hypnotics up to 1960s, which led to their widespread abuse.

1. Classification

Please see [Table 6].

Table 6: Classification of Barbiturates

S.No	Class of Barbiturate	Onset of action	Duration of action	Major Examples
1.	Long acting	2 h	6-12 h	Barbitone, Diallylbarbituric acid, Mephobarbitone, Methyl phenobarbitone, Phenobarbitone, Phenytoin
2.	Intermediate acting	0.5-1 h	3-6 h	allobarbitone, Amobarbital [syn, Amobarbitone, Amylobarbital, Amylobarbitone, Pentymalum, Sodium amytal][Proprietary names Amal; Amytal; Eunoctal; Neur-Amyl; Stadadorm], Aprobarbital, Butobarbitone, Probarbitone, Vinbarbital
3.	Short acting	10-15 m	<3h	Cyclobarbitone, Ortal, Pentobarbital, quinalbarbitone, Seconal
4.	Ultrashort acting	immediate	5-10 m	Kemithal sodium, Pentothal sodium, Thiamylal sodium

2. Mechanism of action

(1) Barbiturates are **CNS depressants**. They depress neuronal activity in the midbrain reticular formation

(2) They facilitate and prolong the inhibitory effects of GABA and glycine.

(3) Their actions are not antagonized by flumazenil [Flumazenil reverses the CNS effects of benzodiazepines and is classified as an antagonist at BZ receptors].

3. Toxicokinetics

(1) Barbiturates are rapidly absorbed from GIT including the rectum

(2) After ingestion, barbiturates are preferentially absorbed from the small

intestine

(3) They are concentrated in the liver for a short time, and then evenly distributed in the body fluids

(4) Longer-acting barbiturates tend to be more lipid soluble and more protein bound, a more rapid onset and shorter duration of action, and are metabolized almost completely in the liver.

(5) Barbiturates are eliminated by hepatic and renal systems

(6) Urinary excretion of unchanged drug is significant for phenobarbital.

4. Acute poisoning

May result from a single large dose or with repeated small doses.

a. Signs and symptoms

(1) **CNS** – CNS depressant effect mimics that of ethanol. [A] **Symptoms** (i) drowsiness [usually the earliest symptom] (ii) Ataxia (iii) Confusion (iv) Delirium (v) Excitement (vi) Hallucinations (vii) Headache (viii) nystagmus [lateral and vertical] (ix) Paresthesias (x) Slurred speech (xi) Vertigo (xii) Visual disturbances (xiii) Finally stupor progressing to deep coma. (a) Coma may persist for a few hrs to a few days, and then the patient makes a gradual recovery. (b) Temporary failure of accommodation, leading to diplopia may occur during recovery. (xiv) Rapid anesthesia – seen in lipophilic barbiturates [eg thiopental] because penetrate brain tissue quickly. (xv) Psychological – (a) .ed anxiety (b) euphoria (c) loss of inhibitions (d) relaxed contentment. These symptoms make them a popular drug of addiction [B] **Signs** -(i) Babinski toe sign +ve (ii) Loss of response to painful stimuli (iii) Pupils – (a) slightly contracted (b) react to light (c) Dilate [may be unequal] during terminal stages [due to hypoxia] (iv) Superficial and deep reflexes - inhibited or completely lost.

(2) **Respiratory** –

(i) Barbiturates cause a depression of the medullary respiratory center " induce respiratory depression " leads to (a) bronchopneumonia (b) respiratory acidosis [contributes to CVS depression]

(ii) COPD patients more susceptible [even at therapeutic doses].

(iii) Respiration (a) either rapid and shallow or (b) slow and labored

(iv) Minute volume .ed in all cases

(v) In terminal stages "irregular "Cheyne Stokes "stops completely ["death].

(3) **CVS** -

(i) Depression of medullary vasomotor centers " Cardiovascular depression

(ii) Cardiac output.. Patients with CHF more susceptible to these effects

(iii) -capillary permeability [because of compromised vascular tone]"-ECF

(iv) Signs of cardiovascular collapse [hypotensive shock] – (a) Cold, clammy skin (b) Cyanosis (c) Hypotension (d) Pulse – weak and rapid. Shock and respiratory failure are more common and more serious with medium and short acting barbiturates than with long acting barbiturates.

(4) **GIT** -

(i) Bowel sounds absent [due to .peristalsis; bad prognostic sign]. Occurs in deeply comatosed patients

(ii) As condition improves " peristalsis returns to normal " -drug absorption " worsening of condition again " fluctuating levels of consciousness

(iii) Incontinence of feces

(5) **Renal** -

(i) Urine (a) Scanty and suppressed (b) contains albumen, hematoporphyrin, sugar (c) Incontinence

(6) **Dermatological** -

(i) **Cutaneous bullae** [barb burns, barbiturate blisters, coma blisters, friction blisters] - (a) Seen most commonly in (I) barbiturates, but in almost any other condition which produces coma. They have been reported in poisoning by (II) benzodiazepines, (III) CO, (IV) glutethimide, (V) hydrocodone, (VI) imipramine, (VII) meprobamate, (VIII) methadone, (IX) methaqualone [Mandrax], (X) tricyclics, valproic acid and in (XI) prolonged contact with cantharidin, paraffin and petrol. Seen also in pemphigus [but they are non tense and larger]. Thermal blisters will show associated signs of burns eg singeing of hair. (b) Frequency - seen in 6-10% cases (c) site – where friction or pressure naturally occurs, eg interdigital clefts, buttocks, back of thighs, calves and forearms, inner aspects of knees and ankles. Occasionally entire side of a forearm or thigh is blistered. Have been reported in non pressure points, eg around IV site. (d) Characteristics - (I) Tense subepidermal blisters which develop within 48–72 h of the onset of unconsciousness (II) Self-limiting lesions; heal spontaneously in 1-2 wks. (III) Inflammation usually absent or minimal. (IV) contain clear serous

fluid. May be hemorrhagic. Rupture of blister leaves a red raw surface, which dries to a brown parchment like area. May be mistaken for burns. (e) Mechanism - (I) External uninterrupted pressure + arterial hypotension due to shock"hypoxia, local ischemia " metabolically most active cells are affected earliest [eccrine sweat glands]"sweat gland necrosis"necrotic bullae. (II) direct toxic action on epidermis (III) Immune mechanisms. (IV) Barbiturate poisoning"muscle flaccidity"cessation of venous return"cutaneous edema.

(7) **Body temperature** -

(i) .ed

(ii) May be -ed in late stages"indicates bron chopneumonia [please co-relate with respiratory symp].

(8) **Uncommon symptoms** [adverse effects] –

(i) Paradoxical excitement [especially in the elderly]

(ii) Hypersensitivity reactions [erythematous or exfoliative dermatitis, localized swelling of eyelids, cheeks, lips].

(9) **Death** -

(i) In early stages due to – (a) respiratory failure [commonly], (b) ventricular fibrillation. Death is rapid if alcohol or antihistamines are ingested simultaneously [synergistic action]. [please also see above – Alcohol"Relationship with barbiturates]

(ii) In late stages due to –(a) bronchopneumonia (b) pulmonary edema (c) acute renal failure (d) cerebral edema.

Memory Aid 13: Major symptoms of barbiturate poisoning

BARBITURATES

Babinski +ve, **B**P **.**

Ataxia

Rresponse to painful stimuli lost, **R**eflexes **.**, **R**/r **.**

Bowel sounds absent

Incontinence of urine and feces

Temperature **.**ed

ver**T**igo

Urine **.**; contains albumen, hematoporphyrin, sugar

pa**R**esthesias

Reflexes lost

Abnormal senses [hallucinations]

nys**T**agmus

Eye can not see properly [visual disturbance]

Small pupils [contracted], **S**kin cold and clammy and shows bullae, **S**leepiness, **S**lurred speech, **S**tupor progressing to deep coma [final stage]

b. Fatal dose

(1) **Fatal dose** - (i) Short acting:1-2 g (ii) Medium acting:2-3 g (iii) Long acting:3-5 g

(2) **Fatal blood levels** - (i) Short acting:3 mg% (ii) Medium acting:7 mg% (iii) Long acting:10 mg%.

c. Fatal period

1-2 days.

d. Management

(1) **Gastric lavage** –

(i) With warm water mixed with $KMnO_4$ and suspension of activated charcoal or tannic acid

(ii) Leave a concentrated sol^n of $MgSO_4$ in the stomach "(a) produces purgation (b) minimizes intestinal absorption

(iii) Effective up to 24 h post ingestion

(2) **Emesis** – contraindicated, because (i) possibility of impending coma (ii) Ipecac can - potential for CNS depression.

(3) **Activated charcoal** – usual dose. MDAC [**ch 32**].

(4) **Enema** – to evacuate bowels

(5) **Specific antidote** – (i) None. (ii) Analeptics – Once recommended. Contraindicated now [**ch 32**].

(6) **Urine alkalinization** – **ch 32**.

(7) **Scandinavian method** – **ch 32**.

(8) **Hemodynamic support**– for hypotension [**ch 32**].

(9) **Hemodialysis** or **hemoperfusion** – **ch 32**.

(10) **Exchange transfusion** – **ch 32**.

(11) **Hemoperfusion** – **ch 32**.

(12) **Management of prolonged coma** – deep vein thrombosis, orthostatic

pneumonia etc [**ch 32**].

(13) **Immunotherapy -** Barbiturates are conjugated to Bovine Gamma Globulins [BGG] and injected in rabbits to produce active immunization. Serum from these rabbits is then injected in other rabbits to produce passive immunization. Both actively and passively immunized rabbits show a .ed pharmacological response to barbiturates indicating the presence of an antibarbiturate gamma globulin. [please see **ch 36**"Arsenic also].

(14) **Intravenous Lipid Emulsion (ILE)** – [**Ch 32**""“General principles of treatment”].

(15) **THAM** [trometamol; tris-hydroxymethyl aminomethane] – Useful as an alternative to $NaHCO_3$ in the treatment of acidosis.

(16) Symptomatic treatment.

e. PM appearances

(1) **Signs of asphyxia –** congestion of all internal organs, cyanosis, petechial hgs in lungs and on pleura and pericardium

(2) **Nose and mouth** - Froth

(3) **Skin** - Barbiturate blisters

(4) **Lungs** –

(i) Intensely congested. May even be turned completely black [full of dark deoxygenated blood]

(ii) Show edema and bronchopneumonia in late deaths

(5) **Esophagus** – lower end may show erosion from regurgitation

(6) **Stomach** – (i) congested. (ii) erosions. (iii) White particles on ingested barbiturates seen (iv) Fundus – thickened, granular, hemorrhagic

(7) **Kidneys** – degeneration of convoluted tubules

(8) **Brain** – edematous, especially in late deaths. In delayed deaths there is symmetrical necrosis of globus pallidus and corpus callosum, focal areas of necrosis in cerebrum and cerebellum and a variety of vascular lesions.

f. Laboratory detection

(1) **Dille-Koppanyi Test** - Two solutions are used.

(i) Sol^n 1 - 1% cobalt acetate in methanol $[Co(C_2H_3O_2)_2]$.

(ii) Sol^n 2 - 5% isopropylamine in methanol $[CH_3CH(CH_3)NH_2]$.

(iii) Add two drops of Sol^{n} 1 to the drug, followed by one drop of Sol^{n} 2"If barbiturates are present a lavender-blue color will develop.

(2) Calorimetric methods

(3) GC-HPLC.

5. Chronic poisoning

(1) **Etiology** -

(i) Prolonged therapeutic use [as in epilepsy and psychoneurotic patients]. Unintended addiction may occur. Dependence is both psychic and physical

(ii) Prolonged recreational use.

(2) **Signs and symptoms** - Similar to those of chronic alcoholism [please also see above – Alcohol" Relationship with barbiturates].

(i) Physical deterioration - (a) Ataxia (b) cerebral function impaired (c) depression (d) Dysarthria (e) hypertonia and tremors of Parkinsonian type (f) Tendon reflexes depressed

(ii) Social deterioration – Impairment of mood, behavior and intellectual functions.

(3) **Barbiturate abstinence syndrome** [syn, Barbiturate withdrawal syndrome] -

(i) Barbiturates can produce psychological and physical dependence and produce a withdrawal syndrome on the 2nd to 4th day after the drug is suspended.

(ii) Symptoms (a) anxiety (b) Disturbances of vision (c) dizziness (d) Hypotension (e) insomnia (f) Nausea and vomiting (g) psychosis (h) restlessness (i) rhythmic intention tremor (j) Tremors, convulsions, seizures and (k) Weakness. May persist for 2 wks. If the syndrome is not recognized and correctly treated, hyperthermia, circulatory failure, and death may ensue. Treatment - Administration of barbiturates.

6. ML importance

(1) **Popular drug as an agent of suicide** – Famous people who committed suicide with an overdose of barbiturates include

(i) Cesare Pavese [1908 –1950] Italian author

(ii) Dalida [1933 –1987] Italian singer

(iii) Margaux Hemingway [1954 –1996] American fashion model and actress

(iv) **Marilyn Monroe** [1926 –1962] American actress and

(v) Robert Hayward Barlow [1918 –1951] American author.

(2) **Euthanasia -** Barbiturates are a popular drug for euthanasia. In Switzerland, **Exit Association** [an organization providing assistance to individuals wishing to end their lives] utilizes oral pentobarbitone for the purpose.

(3) **Barbiturate automatism – ch 28**.

(4) **Drug of abuse, recreational use –**

(i) Popular drug of abuse because of psychological symptoms it produces. Short-acting and intermediate-acting barbiturates are preferred [amobarbital (Amytal), pentobarbital (Nembutal), secobarbital (Seconal), combination of amobarbital and secobarbital (Tuinal)]. Known in drug world as barbs, bluebirds, blues, dolls, downers, goofballs and tooties.

(ii) Physical and psychological dependence develops with repeated use.

(iii) Sometimes used to alleviate adverse or withdrawal effects of other illicit drugs.

(5) **Used in narcoanalysis** – As truth serum [**ch 30**].

(6) **Judicial Execution** - Used in judicial execution by injection. A sequential drug combination of **sodium thiopental** [CNS depression], **pancuronium bromide** [paralysis of respiratory muscles], and **potassium chloride** [cardiac arrest] is used. The first lethal inj execution however took place 5 y later in 1982 in Texas, with the execution of **Charles Brooks, Jr**. [1942 – Dec 7, 1982]. (i) Currently major methods of State execution (capital punishment) around the world are (a) electrocution [**ch 16**] (b) firing squad (c) gassing [**ch 44**] (d) hanging [**ch 19**] (e) lethal injection and (f) strangulation [garroting, **ch 19**]. Oklahoma and Texas became the first US states to introduce lethal injections in the spring of 1977.

(7) **Secondary poisoning** [please see ch 31]– has been described in dogs, who have eaten carcass of a horse euthanized with pentobarbital, up to 2 y previously. Barbiturates resist putrefaction. There is a necessity of proper disposal of animals euthanized with barbiturates. Can occur in humans also.

B. Chloral Hydrate

Chloral hydrate (CH) was first synthesized in **1832** by **Justus von Liebig**. First used as a sedative and hypnotic in **1869**; before this time the only sleep inducing drugs [soporifics] were alcohol and opium. It is often used as a **recreational drug**. Its structure is quite similar to that of ethyl alcohol.

1. General

Chloral hydrate is a colorless, crystalline substance with a **pungent pear like odor** and **bitter taste**. Several formulations containing CH are still marketed. Some are gargles, lotions, or solutions used as mouth bath owing to CH analgesic and disinfectant properties. Also used in syrup forms prescribed as **sedatives** in the **elderly population;** for short term **sedation in children** (during non painful procedures eg CT scan, MRI of brain, and detailed echocardiography); in the treatment of **alcoholism** and **strychnine** poisoning. CH exhibits a significant risk of mutagenesis and carcinogenicity. **Therapeutic oral dose** in adults -> 0.5 to 2.0 g.

2. Mechanism of action

Chloral hydrate is metabolized by hepatic alcohol dehydrogenase. **Trichloroethanol** (TCE) –its first active metabolite- is lipid soluble (high affinity for neurons) and is responsible for chloral hydrate's hypnotic effects.

3. Absorption, metabolism and excretion

(1) **Absorption** - Well absorbed from entire GIT including rectum [may be administered rectally]

(2) **Metabolism** – Metabolized rapidly in liver mainly to TCE

(3) **Excretion** – TCE has a plasma half life of 4-12 hours; it is metabolized to inactive trichloroacetic acid by aldehyde dehydrogenase. Part of it is conjugated with glucuronide and excreted in urine as **urochloralic acid**.

4. Acute poisoning

The clinical picture of acute CH poisoning resembles acute barbiturate poisoning in many respects

(1) Nausea, vomiting, retrosternal burning sensation, hemorrhagic gastritis. Rarely gastric and intestinal perforation or esophagitis with stricture formation.

(2) **Odor** - The peculiar pear-like odor of CH may help distinguish it from other sedative-hypnotics.

(3) **Pupils** - may be miotic initially and then dilate as the stage of coma deepens.

(4) **Skin** – Scarlatinal or urticarial rash [due to idiosyncrasy]

(5) **CNS** - Ataxia and lethargy. Deep coma within 1 to 2 hours.

(6) **CVS** - atrial fibrillation, supraventricular tachycardia, ventricular tachycardia, multi focal premature ventricular contractions, torsade de pointes, ventricular fibrillation, asystole and hypotension. The combination of ***deep coma*** and ***dysrhythmias*** in a patient who is ***not hypoxic*** is typical of CH poisoning.

(7) **Hepatorenal** - Dysfunction of liver [jaundice] and kidney [albuminuria]

(8) **Death** – Occurs from CVS disorders.

5. Chronic poisoning

Occurs after prolonged therapeutic use.

(1) **Skin** - Erythematous and urticarial eruptions

(2) **GIT** irritation

(3) **CNS** – convulsions, tremors

(4) **Respiratory system** – Dyspnea

(5) **Hepatic** – Liver damage

(6) **Dependency syndrome** with withdrawal symptoms when drug is withdrawn. Delirium tremens, seizures and psychosis may result.

6. Fatal dose

5-10 g.

7. Fatal period

8-12 hours.

8. Management

(1) **Stomach wash** - with alkaline solution, if patient is seen early. Chloral hydrate is rapidly absorbed, particularly after ingestion of liquid forms

(2) **Treatment of cardiac arrhythmias** – This should be the main concern.

(i) Propranolol

(ii) Esmolol – Short acting ß-blocker. Preferable to propranolol because of its rapid action.

(iii) Bretylium – Adrenergic neuron blocking drug

(3) **Treatment of hypotension** – Infuse 10-20 mL/kg of isotonic fluid. Catecholamines are contraindicated because they precipitate ventricular arrhythmias in chloral hydrate overdose.

(4) **Flumazenil** - 200µg by infusion followed by 100µg at 1 min intervals, up to 3 times. Total 500µg.
(5) Hemodialysis and hemoperfusion.

9. Tests

Fujiwara test - 1 mL Urine + 1 mL NaOH + 1 ml pyridine. Heat in a boiling water-bath for 2 minutes. An intense red/purple colour indicates the presence of trichloro compounds [chloral hydrate, chloroform, trichloroethylene].

10. Postmortem appearances

(1) Stomach mucosa congested, eroded and softened. Smells of chloral hydrate
(2) Congestion of all internal organs
(3) Necrosis of liver and kidney.

11. ML importance

(1) **Used for rape and robbery** - Chloral hydrate is soluble in both water and alcohol. A solution of chloral hydrate in alcohol called "**knockout drops**" or "**Mickey Finn**". It renders a person helpless immediately. Used for **robbery** [by giving it to watchmen] or **date rape** [date rape drug]. Chloral hydrate is sometimes referred to as **dry wine**
(2) Used to knock out **private detectives** in movies. However it takes a lot longer to put people to sleep with this drug, than the movies would show.

C. Hydrocarbons

Hydrocarbons are organic compounds consisting entirely of hydrogen and carbon. Signs, symptoms and treatment etc of all hydrocarbons are the same as that in kerosene poisoning.

1. Kerosene poisoning

Kerosene is a thin, clear liquid consisting of a ***mixture of hydrocarbons*** that boils between 150°C and 275°C.

Salient features:

(1) Kerosene is derived from **refined petroleum**, and is commonly used as a fuel or solvent.
(2) It is primarily used in **oil lamps**, as a **heating oil**, as **fuel in jet engines**, and

as a **solvent for insecticide sprays**.

a. Signs and symptoms

(1) Kerosene has a strong smell and taste. Thus it can not be used as a homicidal poisoning.
(2) Suicidal poisoning is rare.
(3) Most poisoning cases are accidental in nature, when a child ingests kerosene.

Acute poisoning

(a) Ingestion

(1) **Kerosene taste**, Burning sensation in the throat, nausea, vomiting, colicky pain, diarrhea.
(2) **Breath, vomit and urine smell of kerosene**.
(3) Fever
(4) **Lungs – Main organ system** affected. (i) **Bronchospasm,** (ii) **chemical pneumonitis,** (iii) **pulmonary edema,** (iv) **atelectasis**. (v) dyspnea, tachypnea, gasping, coughing and choking indicate aspiration (vi) cyanosis (vii) intercostal retraction(viii) Nasal flaring
(5) **Heart –Dysrhythmias**. Kerosene **sensitizes the myocardium to endogenous catecholamines** producing **dysrhythmias**. **Myocardial function may also be depressed** resulting in a poor left ventricular ejection fraction.
(6) **CNS** - Neural tissue is rich in myelin (lipid). Myelin is dissolved by kerosene. This causes **depression** of **CNS** and of **ventilatory drive**.
(i) **Depression of CNS** leads to " **Giddiness, weakness, drowsiness**.
(ii) **Depression of ventilatory drive** leads to " **cyanosis**.
(7) **Skin lesions** (**bullae**, **blisters** and **burns**) – Kerosene (like allo hydrocarbons) is **lipophilic** in nature. Hence it dissolves the lipids in the stratum corneum making the skin more vulnerable to drying. This in turn leads to skin lesions varying from (i) **bullae**, (ii) **blisters,** (iii) **maculopapular rash** and (iv) **first degree burns** (v) chronic **eczematoid dermatitis** with redness, itching and inflammation
(8) **Kidneys** - Renal damage results in **type 2 renal tubular acidosis**.
(9) **Autonomic dysfunction** - Hypotension, excessive sweating.

(b) Inhalation

Kerosene is sometimes “sniffed” by addicts [inhalant abuse - please see also **ch 46**]. Inhalation of fumes causes (1) **nausea**, (2) **vomiting**, (3) **headache** (4) **vertigo** (5) **Lung complications** and (6) **convulsions**. Sometimes (7) elation may occur.

b. Diagnosis

(1) **X-ray** -

(i) Changes may be evident as early as 30 min after exposure, and sometimes may precede clinical symptoms.

(ii) Chest – (a) perihilar densities (b) Bronchovascular markings (c) Pneumonic consolidation

(iii) X-ray of abdomen gives the classical **double bubble sign** [two liquid densities in the stomach – **air:kerosene** and **kerosene:fluid**. Air is lightest, then kerosene and finally fluids]

(2) **Arterial blood gases** – Hypoxemia

(3) **Blood** – Leucocytosis during first 48 hours.

c. Treatment

(1) **Decontamination -**

(i) **Contact poisoning** – Remove all clothings; copious washing of skin with soap and water.

(ii) **Gastric lavage and emetics** – ***avoided*** because of high risk of aspiration

(2) **Antidote** - No specific antidote

(3) **Supportive treatment -**

(i) **Oxygen administration** (a) **High Frequency Jet Ventilation (HFJV) – ch 32** (b) **Extracorporeal Membrane Oxygenation (ECMO) – ch 32**

(ii) **Antibiotics** - to prevent secondary pulmonary infection

(iii) **Corticosteroid therapy** – Was given once for severe bronchospasm. May increase bacterial superinfection though, and thus no more recommended now.

d. Fatal dose

30-100 ml. Fatality rate is low. Children younger than four years old are more often affected.

e. Fatal period
One day.

f. ML significance
If a parent has stored kerosene oil negligently resulting in accidental intake by a child, he is liable to be prosecuted u/s **284 of IPC** (negligent conduct with respect to poisonous substance; 6 months and/or $ 1000).

Chronic poisoning
Chronic exposure to kerosene causes (1) **acne** (2) **bone marrow depression** (3) **liver damage** (4) **polyneuritis** and (5) **skin eruptions**.

41. Deliriant Poisons

I. DATURA

Datura [from Sanskrit Dhurta, cheat] is a genus of vespertine [flowering in the evening] flowering plants belonging to the family ***Solanaceae***.

Salient features:

(1) Hyoscyamus and Atropa are related genera.

(2) **Habitat** – Grows on waste places all over India

(3) **Species -**

(i) D. ceratocaula

(ii) D. discolor – Desert Thorn-apple

(iii) D. ferox – Long Spined Thorn-apple ***D. Metel*** and ***D. Stramonium*** are important. D. Metel has two varieties (a) White flowered plant [sometimes loosely referred to as D. alba] (b) Deep purple flowered plant [called D. niger]. **Table 1** gives botanical and common names of some common deliriant plants.

(4) **Plant** – Characteristics of D. fastuosa

(i) Plant -Leafy herb with a woody stalk that grows up to five feet tall.

(ii) Flowers – bell shaped. Have five petals, which open after dusk and close by mid-morning the next day [Datura is a nocturnal plant]. They exude a pleasant, narcotic scent, especially at night.

(iii) Fruits – spherical, have sharp spines [**Fig 41.1**]. Give rise to the plant's English name 'Thornapple'. Contain up to 500 yellowish brown seeds [**Fig 41.1**]. They look very much like chili seeds and must be differentiated from them [**Table 2**].

(iv) Poisonous parts – All parts are poisonous [seeds, fruit, nectar (honey)].

Fig. 41.1: Fruit of Datura, showing spines

Table 1: Botanical and common names of some common deliriant plants

S.No.	Botanical name	Common name
1.	*Atropa belladonna*	Deadly nightshade
2.	*Datura alba*	Safed Datura
3.	*Datura fastuosa*	Thorn apple
4.	*Datura niger*	Kala Datura
5.	*Datura stramonium*	Jimson weed, Thorn apple
6.	*Hyoscyamus niger*	Henbane, Khorasani ajwayan

Table 2: Differences between Datura and Chili seeds. Please correlate with **Fig 41.2**.

S.No	Criteria	Datura seeds	Chili seeds
1.	Size	Large and thick	Small and thin
2.	Shape	Kidney shaped	Rounded
3.	Color	Dark or yellow brown	Pale yellow
4.	Margins	Double edged at the convex border	Single Sharp edge
5.	Surface	Numerous small depressions	Smooth
6.	Smell	Odorless	Pungent
7.	Taste	Bitter	Pungent
8.	Embryo [**Fig 41.2**]	On longitudinal section, curved outwards at hilum	Curved inwards like numeral 6

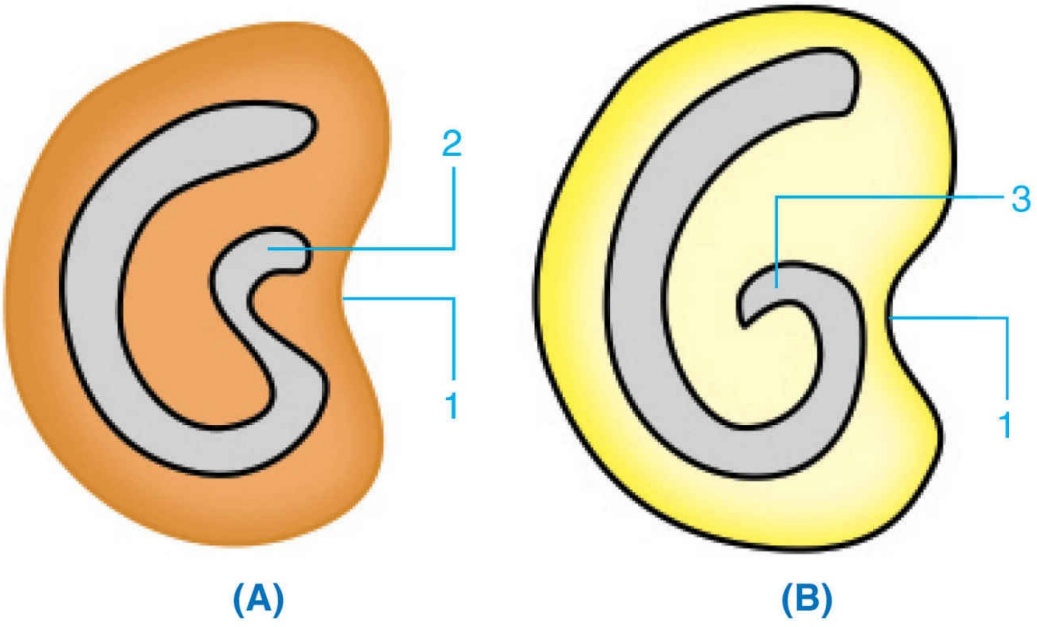


Fig. 41.2: Longitudinal section of **datura seeds (A)** and chili seeds **(B)**. (1) Hilum (2) Embryo curved outwards (3) Embryo curved inwards. (2 & 3) are visible on microscopic examination.

A. Active Principles and MOA

(1) Datura plant contains about 0.2%-1.4% of alkaloids (i) **scopolamine** [l-hyoscine], (ii) **atropine** [d,l-hyoscyamine, older name Daturine] and (iii) **hyoscyamine** [older name Duboisine]. Atropine and hyoscyamine are isomers, the latter being levo-isomer of atropine. During extraction hyoscyamine may be converted to atropine. These alkaloids competitively inhibit the muscarinic effects of Ach.

(2) There are also traces of glycoside **scopolin**.

B. Signs and Symptoms

1. Contact

Contact with leaves or flowers "Dermatitis in sensitive persons.

2. Ingestion

(1) **Appearance of symptoms** -

(i) Ingestion of seeds - appear within 30 min

(ii) Decoction of seeds – within 5 min

(iii) Ingestion of alkaloids – within few seconds. It results in the appearance of **anticholinergic toxidrome** [**ch 31**], with all its typical signs and symptoms [remember “blind as a bat” mnemonic].

(2) **Symptoms** - (i) Initially - Bitter taste, vomiting, burning pain in the stomach. Later "(ii) Face – flushed (iii) Conjunctiva – congested (iv) Loss of accommodation for near vision – results in (a) temporary blindness (b) photophobia (c) diplopia (d) Pupillary reflex – at first sluggish; later absent (v) Voice – hoarse (vi) Skin – Scarlatinal rash, or exfoliation of skin over most of the body (vii) CNS and psychiatric– (a) agitation (b) confusion (c) giddiness (d) restlessness (e) failure to recognize relatives and friends (f) Delusions (g) Hallucinations of sight and hearing (h) tonic convulsive seizures (viii) **15 D’s** [**Memory Aid 1**]. Additional points – (a) dysuria may progress to complete urinary retention. (b) skin is dry [due to loss of sweating (**hypohidrosis**)] and also hot. (c) Drunken gait is ataxia (d) Delirium – patient is restless, makes purposeless movements [eg pulling imaginary threads, threading imaginary needles, picking at bed clothes - Carphologia or floccilation] [also **ch 28**"delirium]. Pt is initially excited, incoherent, noisy, talkative and tries to run away from bed (ix) Excitement remains for 1-2 h after which patient goes in deep sleep [coma] (x) Lastly – resp depression, resp paralysis and death [also see **ch 31**"anticholinergic toxidrome].

Memory Aid 1: Datura’s signs and symptoms

Remember 15 D’s (1) Decreased muscle coordination (2) Delirium (3) Delusions (4) Dermatitis (5) Dilated pupils (6) Dreadful hallucinations (7) Drowsiness (8) Drunken gait (9) Dry hot skin (10) Dry membranes (11) Dryness of mouth and throat (12) Dysarthria (13) Dysphagia (14) Dysuria (15) Death due to respiratory failure

(3) **Signs** –

(i) Pulse – (a) - [120-150/min]. (b) Volume – Initially- full and bounding; later – weak and irregular [arrhythmia].

(ii) BP- [systolic BP may be up to 180]

(iii) R/R- [later resp failure]

(iv) Temp -ed by 3-4°C [other poisons causing hyperpyrexia"**ch 31**].

(v) Muscle tone and reflexes "-ed [hyperreflexia] with positive Babinski's sign.

C. Fatal Dose

(1) Atropine or hyoscyamine – 120 mg
(2) Scopolamine – 30 mg
(3) Datura seeds – 50-100.

D. Fatal Period

24 hours.

E. Diagnosis

(1) **Mydriatic test** [**animal testing**]– Instill patient's urine [or a sol^{n} of suspect seeds in water or alcohol] in cat's or rabbit's eye"immediate mydriasis.
(2) **Pilocarpine test** – instill 2-3 drops of 1% pilocarpine [or physostigmine] in patient's eye"no constriction within half an hour"Datura [or other anticholinergic] poisoning
(3) **Blood** - Neutrophil leucocytosis.
(4) **Hair analysis by MS** – Atropine and scopolamine can be identified in hair up to 3 weeks after exposure. Timing of exposure can be determined by segmental hair analysis [**ch 31**].

F. Management

(1) Shift patient to a quiet and dark environment [**ch 32**"gives all poisons in which pt is shifted to dark].
(2) **Monitor and correct**
(i) Vitals – ECG, Pulse, R/r, temp.
(ii) For sinus tachycardia – Requires treatment only if hemodynamic compromise develops. If required, give short acting cardioselective agent eg esmolol.
(3) **Respiratory assistance** – If seen late, patient may be in respiratory failure. Endotracheal intubation, assisted ventilation.
(4) **Decontamination** -
(i) Emetics
(ii) Gastric lavage – with a weak sol^{n} of tannic acid. Even late gastric lavage is

useful, because gut motility is .ed. Datura seeds may be recovered from stomach even 36 h after ingestion.

(iii) WBI

(iv) Purgatives – to continue for 3-4 days to remove seeds. Light diet during this period.

(5) Activated charcoal

(6) IV fluids. Monitor input, output, renal function

(7) Catheterize bladder

(8) Dialysis and hemoperfusion – not effective

(9) **Antidotes** -

(i) Physostigmine – Antidote of choice. It is a tertiary amine that easily passes BBB [reverses central effects too]. Dose – (I) Adult - 2 mg IV slowly; repeated if required in 20-60 min [Duration of action – 20-60 min]. (II) Pediatric – 0.5 mg IV slowly; repeated if required in 5 min, up to a maximum of 2 mg.

(ii) Neostigmine, pilocarpine and pyridostigmine – do not pass BBB because they are quaternary amines. Central effects are not countered.

(iii) Contraindicated drugs - Antihistamines, Disopyramide, morphine [depresses respiratory center], Phenothiazines, Procainamide, Quinidine and Tricyclics.

(10) **Symptomatic** -

(i) Agitation – diazepam IV. Generally antihistamines and phenothiazines are to be avoided because they -anticholinergic effects. Cyamemazine [**antipsychotic**] is however recommended.

(ii) Delirium – bromides, chloroform, ether, short acting barbiturates

(iii) Hyperthermia – cooling measures, hydration.

G. PM Appearances

(1) No characteristic findings

(2) Findings of asphyxia

(3) **Stomach and intestines** –

(i) Walls - inflamed

(ii) Fragments of seeds and plant may be found. Seeds resist putrefaction for a long time

(4) **Lungs** – edematous.

H. ML Importance

(1) **Abortifacient**

(2) **Aphrodisiac -** Believed to be an aphrodisiac. Used as a **love philter**.

(3) **Autumnal High** – If youth are seen with hallucinogenic symptoms during autumn months, it could be a case of Datura poisoning. Seeds of Datura appear around autumn, and youth may consume its seeds for their mild hallucinogenic effects.

(4) **Beer** - Solanaceous plants have been used to fortify beer since time immemorial in order to potentiate the effect of the alcohol. **Mandrake** was used in Egypt and **thornapples** in Russia for the same purpose.

(5) **Cornpicker's pupil** - (a) Corn fields are usually infested with Datura plants. Harvesting of corn invariably exposes the cornpickers to Datura dust. This results in persistent mydriasis (**cornpicker's pupil**). (b) A similar condition is **gardener's mydriasis**, where gardeners dealing with Datura plants [grown as a hobby in gardens] get persistent mydriasis. (c) Laboratory technicians, who weigh out atropine powder for experiments may show same sign. This has led doctors to wrongly diagnose cranial lesions, and has even led to lumbar punctures and MRIs. In all cases of persistent dilatation of pupil, the occupation of person must be asked.

(6) **Criminal responsibility** – If Datura is administered to a victim without his knowledge [as for stupefying him before robbery], he is not responsible for any criminal act done under its influence [**S.85, IPC (ch. 31)**]. If the person took Datura himself [eg as a drug of abuse], he is responsible for his criminal actions.

(7) **Drug of abuse**

(i) Seeds and leaves are mixed with tobacco or ganja and smoked in a pipe.

(ii) Decoction of seeds is added to liquor or toddy to -their intoxicating property.

(iii) Use of Datura is more lucrative to addicts because of absence of legislation regarding it.

(8) **Manner of poisoning** -

(i) Suicide and homicide - rare.

(ii) Accidental poisoning - may occur (a) in children who eat its fruits by mistake (b) When used as a medicine - (I) after instillation as eye drops. (II) When

used as antiinflammatory - In some regions it is believed that it acts as an antiinflammatory for conditions such as local trauma, erysipelas and gingivitis. (c) When used topically as a magic charm - Medieval Europe witches supposedly obtained hallucinatory effects by rubbing their bodies and genitals with ointments containing Datura, and Yaqui Indians in Mexico used crushed Datura leaves on their genitals, legs and feet for similar purposes. Still used as a magic charm in many places.

(9) **Poisoning to poultry** – D. stramonium is toxic to livestock, including poultry. Its seeds can contaminate grains and is the most common cause of poisoning in chickens.

(10) **Relief of Asthma** - During the 19th century, Datura was marketed as "Spanish Herbal Cigarettes." Advantage was taken of its antihistaminic and bronchiodilating properties; used as a smoke, it relieved the symptoms of asthma.

(11) **Stupefying poison** –

(i) Ingestion - Criminals mix crushed or powdered seeds in food and drink [chapattis, curry, sweets, tea, liquor] to stupefy a victim [usually travelers in railways] before robbery, rape or kidnapping [**Road poison**]

(ii) Inhalation – Seeds may be mixed with incense wood and victim exposed to fumes.

(12) **Toxic honey** – If during their nectar collecting activity, bees collect nectar from Datura plants, the honey may become contaminated with Datura alkaloids. This may give rise to unintentional poisoning [toxic honey. Please see more in **ch 39** – "Food poisoning"]. Some hummingbirds, eg sword-billed hummingbird (Ensifera ensifera), can feed on the nectar from Datura flowers without ill-effects.

II. ATROPA BELLADONNA

Atropa belladonna [syn. Belladonna, Devil's Berries, Death Cherries, Deadly Nightshade], is a perennial herbaceous plant of the family Solanaceae.

Salient features:

(1) Active principles are hyoscyamine [82-97%], atropine [3-15%] and scopolamine [2.5%].

(2) Signs, symptoms, treatment and pm appearances are same as that of Datura.

(3) Because it produced senselessness, it was used in the Middle Ages as an anesthetic for surgery.
(4) The plant has reddish-purple flowers.

ML Importance

(1) Arrow poison.
(2) **Manner of poisoning** - Homicide - The ancient Arabs used it as a poison. The wife of Emperor Augustus and the wife of Claudius both used it to murder their enemies.

III. CANNABIS

Cannabis (Indian hemp, pot, dope, grass) is a genus of flowering plants that includes **three** species, Cannabis sativa, Cannabis indica, and Cannabis ruderalis. All three are found in India, Central Asia, and South Asia (**Fig 41.3**).

Fig. 41.3: Cannabis plant

Salient features:

(1) **Plant** - (i) Cannabis is an annual, dioecious (male and female plants occurring separately), flowering herb. (ii) The leaves are digitate with serrate leaflets

(2) **Frequency of use** - Currently it is the most commonly used illicit drug of abuse. If socially acceptable drugs are also taken into account, then it is 4th most common drug of abuse - (i) Caffeine (ii) Nicotine (iii) Alcohol (iv) Cannabis.

A. Active Principles

(1) Active principles are **cannabinoids [**syn**,** phyto-cannabinoids**]**, secreted by trichomes. At least 90 plant Cannabinoids have been isolated from cannabis.

(2) Cannabinoids of importance are

(i) **Tetrahydro-cannabinols** or **THC**. Most important THCs are **Δ^8-tetrahydrocannabinol** and **Δ^9- tetrahydro-cannabinol**(pronounced as delta-8 and delta-9 tetrahydrocannabinol)

(ii) **Cannabinol** [CBN]

(iii) **Cannabidiol** [CBD].

B. Mechanism of Action

(1) **Endogenous cannabinoid receptors** -

(i) Cannabinoids exert their effects by interaction with specific endogenous cannabinoid receptors [**CB_1, CB_2, GPR18**, **GPR55** and **GPR119**].

(ii) Discovered in 1990s.

(iii) All are **G protein-coupled cannabinoid receptors** [GPRs].

(iv) Location - CB_1 is located in the brain, and CB_2 in peripheral tissues of the immune system.

(2) **Endogenous cannabinoids - Anandamide** [from Sanskrit word “Anand”, happiness], an endogenous cannabinoid, attaches to these receptors. Produces natural pain relief and controls immunity.

(3) **THC** – Additionally acts on CB_1 and enteric nervous system to suppress emetogenic stimuli communicating between them. Acts as an effective antiemetic.

C. Forms of Cannabis

1. Bhang

Bhang [Patti, Siddhi, Sabji] consists of dried mature leaves and stems of cannabis.

Salient features:

(1) **Potency** - It is the mildest form of Cannabis and contains **15%** of active principle. Fresh Bhang is more potent; it loses its potency on storage. Bhang stored for 2-3 years has very mild euphoriant properties.

(2) **Forms in which consumed** - (i) As an infusion – Seeped in water, strained through a muslin cloth and used as a beverage (ii) Cannabis tea – leaves boiled in water just like tea leaves [please see ch 40 for "poppy tea"]. The resulting infusion is supposed to have more **Δ^9-THC** [main bioactive component of cannabis], because heat converts tetrahydrocannabinolic acid (THCA) to **Δ^9-THC**. (iii) As a pill or bolus – Leaved rubbed with black pepper on a stone. Sugar is added to make a pill or bolus [Bhang golis or balls] (iv) Mixed with milk – Almonds and spices (mainly black pepper) are also added and served on festive occassions such as Holi in March and Vaisakhi in April, especially in places like Mathura and Varanasi [Bhang Ki Thandai, Shardai] (v) Mixed with fruit juice

(3) **Commonly used** - (i) to produce a cheerful mood before singing and dancing (ii) as an appetizer (iii) for enhancing sexual pleasure (iv) part of many Ayurvedic medicinal preparation.

(4) **Duration of action** - Intoxication lasts for about 3 hours, ending with sleep.

2. Majoon

Majoon is a sweet prepared with Bhang.

Salient features:

(1) Made by mixing Bhang with honey, and ghee. Sometimes henbane, datura seeds and poppy seeds [for flavoring] are also added

(2) Sold in the market as lozenge shaped pieces [barfi]

(3) Used as an appetizer and aphrodisiac.

3. Ganja (Marijuana)

Ganja is is a rusty-green powder with a characteristic odor, which is prepared from the flowers of female plants.

Salient features:

(1) **Potency** - It is a stronger form of Cannabis and contains **15-25%** of active principle.

(2) **Method of use** – (i) Smoked in a pipe [Chillum or hukka] – About 4-5 g of ganja is taken with little water on palm and rubbed till it becomes sticky. It is then mixed with tobacco and smoked. Sadhus, faqirs and poor laborers in India use this method. (ii) in the form of cigarettes [reefers or joints] – This method more common in the West, and among the affluent in India. A good

quality reefer may contain up to 500 mg of ganja.

(3) **Other names** - (i) Known as Marijuana in the West.

4. Charas (Hashish)

Charas (syn hashish) refers to the dried resin of cannabis.

Salient features:

(1) Contains **25-40%** of the active principle.

(2) **Slang names** – dope, shit

(3) Dark green or brown in color

(4) Mixed with tobacco and smoked just like ganja.

(5) The best charas grown in India comes from the mountains [6-8000 ft above sea-level].

D. Acute Poisoning

1. Signs and symptoms

(1) Signs and symptoms may differ in different persons. They depend upon

(i) life experiences,

(ii) Personality styles and

(iii) Environmental [eg in familiar or unfamiliar environment] and

(iv) social setting [eg in a group of friends or strangers] in which drug is consumed.

(2) **Time of appearance** –

(i) **Smoking** – Appear soon after smoking and last for 1-2 h. Characteristic odor [of burnt rope] is apparent on smoking, but not on ingestion

(ii) **Ingestion** - Appear within ½ h and last for 2-3 h.

a. Psychiatric

(1) **When small doses are taken** –

(i) **General euphoria** - -ed well-being, -ed self confidence, feelings of clarity, cleverness, detachment, disinhibition, dreaminess, elation, jocularity, laughing, relaxation, sometimes silliness

(ii) **Emotions** – labile. change rapidly [laughing followed by weeping and back to laughing]

(2) **When Moderate doses are taken** –

(i) **Sensory** – (a) -ed awareness of stimuli (b) Illusions and hallucinations (c) Sensory novelty (d) Vivid images

(ii) **Memory, judgment and thought processes** – are affected with moderate dosages. (a) .ed attention span [lapses of attention] (b) .ed concentration (c) Altered reality testing (d) Disorders of perception [person may feel superhuman, and may feel he can stop buses with one hand, or could fly out from high windows. Can actually jump leading to death] (e) Altered sense of identity (f) Disorientation [Altered concepts of time and space] (g) Impaired judgment (h) Irresponsible behaviour, Failure to meet responsibilities [eg of home, children etc] (i) Maladaptive behavioral effects (j) Disturbed and irrelevant thoughts (k) Impaired memory (l) Subjective feeling of unfamiliarity. Symptoms mentioned below are seen with -ing dosages.

(iii) **Speech changes** [speech is rapid, impaired. Person becomes talkative] Flight of ideas.

(iv) **Sexual feelings** – May be heightened. That is why, sometimes used as an aphrodisiac

(v) **Somnolence**

(3) **When high doses are taken** -

(i) Changes in body image,

(ii) Depersonalization,

(iii) Marked sensory distortion,

(iv) Sensitive individuals,

(v) Particularly persons recovering from mental illness may become paranoid even after a relatively low dose.

b. Physical

(1) -ed appetite and thirst (2) ataxia (3) congestion of conjunctiva (4) dizziness (5) dry mouth (6) dysesthesias and paresthesias (7) Skin - Acute generalized exanthematous pustulosis (AGEP). (8) precordial distress (9) restlessness (10) Slight nausea (11) heaviness and pressure in the head (12) tachycardia (13) tightness in chest (14) tremors and (15) urinary frequency. (16) Victim becomes drowsy and passes into deep sleep. Wakes with exhaustion and impaired mental function (17) Recovery occurs in 6 h.

c. Intravenous marijuana syndrome

First described in **1968** by Henderson and Pugsley. Seen with IV injections. Usual practice is to boil marijuana, strain it through cotton and inject into a vein.

(1) **Symptoms** – appear within 1 h. (i) nausea, vomiting and diarrhea (ii) myalgia (iii) abdominal pain (iv) weakness and (v) hypotension.

(2) **Signs** – (i) fever, (ii) hepatomegaly, (iii) hypotension [due to hypovolemia resulting from emesis and diarrhea; vasodilation due to circulating endotoxins, due to unsterile equipment etc] (iv) muscle tenderness, (v) Renal insufficiency (vi) tachycardia, (vii) tachypnea.

(3) **Lab findings** -

(i) Pronounced leukocytosis [>20,000/µl]

(ii) Platelets <100,000/µl [thrombocytopenia]

(iii) -Partial thromboplastin time

(iv) Azotemia [BUN >75 mg/dl; creatinine >5.8 mg/dl]

(v) -bilirubin in serum [> 4.1 mg/dl]

(vi) -creatine kinase [CK] in serum

(vii) -myoglobin in both serum and urine

(4) **Pathophysiology** – Vasodilation is the major mechanism.

(5) **Management** - IV administration of fluids, mannitol, vasopressors.

2. Fatal dose

(1) **THC** – 30mg/kg; (2) **Charas** - 2g/kg; (3) **Ganja** - 8g/kg; (4) **Bhang** - 10g/kg

(5) **Some pointers** - (i) Due to **high FD** [eg 700 g of bhang for a 70 kg person], death occurs very rarely. If occurs, it is due to respiratory failure. (ii) In few recorded deaths due to cannabis, there were either multiple drug ingestions, or his health was already compromised [eg heart surgeries, pulmonary diseases etc].

3. Fatal period

Several days.

4. Management

(1) Assurance

(2) **Decontamination** – Stomach wash or emesis following by activated charcoal. Cathartics

(3) If patient is aggressive or violent – 5-10 diazepam IV
(4) If flashbacks occur – antianxiety agents
(5) antipsychotics, eg haloperidol
(3) psychotherapy.

5. PM appearances

(1) Not characteristic (2) Findings of asphyxia.

E. Chronic Poisoning

1. Signs and symptoms

(1) **General** - (i) Facial expressions – sleepy, vacant (ii) Loss of appetite (iii) Suffused eyes (iv) Tremors (v) Wasting (vi) Weakness.

(2) **Amotivational syndrome** - A constellation of effects associated with substance use [especially cannabis], including apathy, loss of effectiveness, diminished capacity to carry out complex or long-term plans, low tolerance for frustration, impaired concentration, and difficulty in following routines.

(3) **Medical conditions** –

(i) Cancers - of mouth and lung [cannabis is a potential carcinogen]

(ii) Cannabis [or cannabinoid] Hyperemesis Syndrome [CHS] – First described in **2004**. Had been described earlier as psychogenic vomiting, but known to be associated with cannabis. Occurs after several years of moderate to heavy use of cannabis in susceptible individuals. (a) Characterized by (I) recurrent episodes of vomiting (II) symptomatic relief with a hot bath and showers (b) Treatment - The only effective treatment is cannabis cessation. (c) Mechanism – It is paradoxical that despite THC being antiemetic, CHS is produced on prolonged use. Appears to be through dysregulation of endo-cannabinoid receptors and episodic gastroparesis [THC delays gastric emptying]. Relief from hot baths and showers appears to be due to temporary improvements in autonomic dysfunction. (d) A history of cannabis use should be enquired in all patients presenting with unexplained recurrent or cyclical vomiting.

(iii) Chronic lung disease - airway narrowing

(iv) Dermatology - Striae on body. Etiology not clear.

(v) Digital clubbing

(vi) Heart - Bradycardia, CCF, Non-specific ST wave changes

(vii) CNS – Degeneration of neurons, poor cognition

(viii) Hormonal and sexual – in males [.fertility, .serum testosterone, .sperm count, Gynecomastia, Impotence]

(ix) Infarctions – (a) May cause infarctions [eg cerebral infarction, MI] (b) Mechanisms -(I) cardio embolism [because of -ed risk of arrhythmia] (II) coagulopathy (III) disruption of atherosclerotic plaque in response to stress (IV) hypotension or hypertension (V) reversible cerebral vasoconstriction syndrome (VI) vasculitis (VII) vasospasm.

(x) URI - Chronic sore throat, Rhinitis, Bronchitis.

(4) **Psychiatric conditions** - Hashish insanity [syn, Charas insanity] may develop

(i) Existing schizophrenia – (a) exacerbation of condition (b) behavioral problems (c) criminal behavior (d) Delusions [of persecution] (e) Hallucinations [auditory, visual] (f) Moral and mental deterioration (g) Culture bound syndromes [CBS]–Conditions occurring in certain cultures only. Two main CBS are seen in cannabis (I) Amok - Running amok [Malay mengamuk, to make a furious and desperate charge] is a psychiatric condition in which a person runs about deliriously killing anyone who comes in way [killing spree]. **Salient features:** [1] **Etiology** - Consumption of cannabis. [2] **Sequence** - Period of depression"followed by sudden running around and killing people with whatever comes in his hand [knife, gun] " depression once again, during which he either commits suicide or surrenders himself to police. (II) Koro [Malay koro, turtle’s head] – Male person has a belief that his penis is shrinking and may disappear in to his abdominal wall and then he may die. Females are affected similarly with a corresponding belief that their breasts [and/or vulva] are shrinking.

(ii) Normal people – (a) may become psychotic, and show same symptoms as above. However it is possible that latent schizophrenia already exists in people who abuse cannabis chronically. (b) Manic or paranoid psychosis may develop.

(iii) Normal people with no latent schizophrenia – no authentic proof of development of psychosis.

(5) **Diseases due to contamination** of street marijuana. Long term use of such contaminated drug use may cause diseases. Some common contaminants are

(i) Animal feces (ii) Aspergillus spores [Aspergillosis] (iii) Formaldehyde (iv) Fungi [Histoplasmosis] (v) Heavy metals [lead, mercury] (vi) Herbicides [paraquat] (vii) Insects (viii) Microorganisms [Salmonella] (ix) Other drugs [PCP etc].

2. Withdrawal

Known as "**Marijuana Withdrawal Syndrome**". (1) Insomnia (2) Irritability (3) Mild agitation (4) Nausea, cramping (5) Restlessness (6) Sleep EEG disturbance.

F. Tests

(1) **Colour tests** -

(i) **Duquenois-Levine test** - Suspected material + Duquenois-Levine reagent [2% vanillin + 1% ethanol] + Conc HC*l* + chloroform " **Purple colour** in the chloroform layer indicates a +ve result. This is a presumptive test for THC and other cannabinoids. Lacks specificity, and is thus good only as a screening test.

(ii) **Fast blue B salt test - Wine red colour** indicates presence of cannabis. Also known as **KN test**.

(iii) **p-dimethylaminobenzaldehyde (p-DMAB) test - Red colour changing to violet** indicates presence of cannabis.

(2) Thin Layer chromatography.

G. ML Importance

(1) **Aphrodisiac** – cannabis is consumed because it is falsely thought to - sexual desire and duration of coitus.

(2) **Driving under influence of cannabis** – Cannabis is the 2nd most common drug of abuse found in drivers [after alcohol]. It does not affect driving performance significantly when used alone, but when taken with alcohol, the effects are pronounced. It is possible for a driver to be driving within safe legal alcohol limit [BAC <30 mg%], but driving may still be impaired if cannabis is ingested concomitantly. Police officer can arrest **u/s185(b)** of Motor Vehicles Act 1988,and produce the accused for medical examination **u/s202** MVA. Laboratory tests can be ordered **u/s204** of MVA [**ch 40**].

(3) **Heavy metal poisoning** – Lead and mercury poisoning have been described

in marijuana abusers.

(4) **Manner of poisoning-**

(i) Abuse - Most cases of poisoning are due to abuse,

(ii) Accidental ingestion or inhalation - occurs also.

(iii) Suicidal – extremely rare. Virtually not known

(iv) Homicidal – Not possible, because of large fatal dose and peculiar odor

(5) **Physiological dependence or addiction** – is not seen with cannabis

(6) **Run amok** - Please see above

(7) **Reasons for which cannabis is taken** – (i) To produce euphoria [most common] (ii) to overcome feeling of tiredness (iii) to overcome feelings of hunger and thirst (iv) to achieve union with God [most often by ascetics, fakirs and religious mendicants] (v) to improve concentration

(8) **Second Hand Marijuana Smoke** – A person may passively inhale marijuana smoke from the environment if an addict is smoking nearby. In random workplace testing [**ch 46**] such an innocent person may be falsely implicated. To avoid such complications, most labs set a higher threshold before reporting THC in the system.

(9) **Stupefying poison** – Unsuspecting strangers are given majoon and charas to commit (i) rape (ii) robbery (iii) Other criminal acts, which are committed as soon as stupefying effects begin to take over. Majoon and charas are offered by road poisoners to unsuspecting travelers often in trains and buses. After stupefaction sets in, they are robbed of valuables.

(10) Sometimes taken by criminals themselves to gather courage before committing a criminal act.

IV. COCAINE

Cocaine [Blow, “C”, Charlie, Coke, Devil’s Dandruff, lady or “she” (as opposed to “he”-heroin), Snow] is a colorless, odorless, crystalline substance with a bitter taste.

Salient features:

(1) The alkaloid cocaine comes from the leaves of Erythroxylon coca, which grows in South America, India and Java (**Fig 41.4**).

(2) A **coca leaf** typically contains less than **2% cocaine**.

(3) By treating the leaves with sulfuric acid, **cocaine sulfate** is produced, which

in turn is converted to **cocaine hydrochloride** by treating it with HCl. Cocaine HCl contains 90% cocaine and is either snorted or injected. It **cannot be smoked**, as its vaporization point is high at which cocaine disintegrates. **Smokeable form of cocaine** (**free cocaine, free base** or **crack** – makes a "crackling" sound when heated) is produced by treating **cocaine HC*l*** with **baking soda**. Act of snorting free base is referred to as **freebasing**.

(4) Second most commonly used illicit drug of abuse after cannabis. [please see ch 46 for full list].

Fig. 41.4: Coca plant showing Leaves and berries

A. Mechanism of Action

(1) Blocks **reuptake of biogenic amines** [dopamine, norepinephrine, epinephrine, serotonin]. -epinephrine" tachycardia; -norepinephrine" hypertension; -dopamine, -se**r**otonin" **r**eward" cocaine addiction. -**s**erotonin

also causes **s**eizures.

(2) **-es excitatory amino acid concentrations** in the brain (psychomotor agitation, hyperthermia, seizures). **Excitatory amino acid antagonists** prevent seizures and death in experimental animals.

Memory Aid 2: Reuptake of biogenic amines blocked by cocaine

SEND – **S**erotonin; **E**epinephrine; **N**orepinephrine; **D**opamine

Memory Aid 3: Main side-effect of cocaine

-Serotonin at **S**ynapses causes **S**eizures

B. Toxicokinetics

(1) **Absorption** - Rapidly absorbed following all routes of exposure. Marginally slower when applied to mucus membranes (nasal, urethra, vagina, rectum) or when ingested because of its **vasoconstrictive** action [limits absorption]. Smoking cocaine produces immediate effects; intranasal administration takes 5-10 min. 90% bound to plasma proteins. **Vd is 2.7L/kg**.

(2) **Metabolism** - occurs through **three** routes (i) 50% is hydrolyzed to **benzoylecgonine** (**BE**) (ii) 45% metabolized by **plasma cholinesterase** (**PChE**) to **ecgonine methyl ester** (**EME**). Persons with low PChE activity demonstrate increased sensitivity to cocaine (iii) 5% undergoes N-demethylation in the liver to form **norcocaine**.

C. Acute Cocaine Poisoning

1. Symptoms and signs

[A] **Modes of administration** - (1) On **smoking** and **IV administration** onset of action is **within 1 minute**, peak action in **3-5 minutes**; (2) **nasal insufflation** " onset of action in **1-5 min**, peak action in **20-30 min**; (3) **Oral ingestion** " onset of action in **30-60 min**, peak action in 6**0-90 min**. Duration of action varies from half hour to 2 hours. Since the duration of action is short, cocaine has to be repeated frequently to maintain a high. [B] Cocaine produces **organ toxicity** in every organ due to (a) its vasospastic action (b) -hemorrhagic tendencies secondary to vasospasm and (c) enhanced **co**agulation. [C] **Symptoms and signs** - (1) **Initially** - there is bitter taste, dryness in the mouth, dysphagia, euphoria. **Headache** may occur due to vasospasm. (2) **Sympathomimetic toxidrome** - (i) Arrhythmias, (ii) diaphoresis, (iii) hypertension, (iv) hyperthermia [**cocaine**

fever], (v) mydriasis, (vi) tachycardia, (vii) tachypnea – [Please also see **ch 31** under the heading “toxidromes”]. (viii) -ed inotropic and chronotropic activity of heart + - in peripheral vascular resistance"(a) acute MI (b) CAD (c) coronary vasoconstriction (d) SAH (e) stroke (f) thoracic aortic dissection. (3) **Neuropsychiatric manifestations** – Hypomanic picture with -d psychomotor activity, -d speech output, agitated delirium, grandiosity, elation of mood, hypervigilance [being constantly tense and “on guard”], seizures. (4) **Eyes** – acute angle closure glaucoma (due to mydriasis). Loss of vision (vasospasms of retinal vessels). **Madarosis** (loss of eyebrow and eyelash hair) occurs from **thermal injury** associated with smoking crack cocaine. (5) **Chest pain** – due to ischemia to heart. (6) **Asthma like symptoms** – Smoked cocaine (not IV cocaine) produces asthma like symptoms (cough, shortness of breath, wheezing). This is because of (a) **thermal insult** to bronchi (b) **AEME** (**AnhydroEcgonine Methyl Ester**) – the unique pyrolytic metabolite of cocaine produces bronchospasm. (7) **Abdomen** – **abdominal pain** due to local ischemia of GIT, -acid production associated with -sympathetic activity. Bloody stools. Intestinal infarction. (8) **Renal** – **Renal failure**. Mechanism is agitated delirium"Seizures"rhabdomyolysis"release of myoglobin. (9) **Paralysis** - **Vasospasm** in the spinal cord can produce paralysis from an **anterior spinal artery syndrome**. (10) **Crack dancing** - Choreoathetoid movements.

2. Fatal dose

1 g IV. Toxic symptoms occur after **200 mg**. Regular users can take up to **10 g**.

3. Fatal period

Few minutes to few hours.

4. Management

a. Decontamination

(1) **Gastric lavage** with warm water containing $KMnO_4$, if taken orally

(2) If snuffed, **nasal irrigation** with NaCl

(3) in cocaine body packer, **surgical removal of cocaine packets**.

b. General supportive care

(1) Maintain **a**irway, **b**reathing and **c**irculation. **Tracheal intubation** may be required. **Succinylcholine** which is usually given prior to tracheal intubation is contraindicated in cocaine toxicity. **Reasons** (a) cocaine seizures"Rhabdomyolysis"hyperkalemia"exacerbation of **hyperkalemia** by succinylcholine"dysrhythmia. (b) **PChE** metabolizes both cocaine and succinylcholine. Their simultaneous use could cause either prolonged cocaine toxicity, or paralysis or both.

(2) For **hyperthermia** – rapid cooling with **ice water immersion**. Avoid antipyretics (acetaminophen or salicylates), drugs that prevent shivering (chlorpromazine, meperidine) and dantrolene. Reason"potential for adverse drug reactions.

(3) For **agitation** – sedation with **benzodiazepines (midazolam**, **diazepam**). If benzodiazepines fail, give short acting **barbiturate** or **propofol**. Avoid phenothiazines and butyrophenones (enhance cocaine toxicity, interfere with heat dissipation, exacerbate tachycardia, prolong QTc interval).

(4) For **hypoglycemia** – Hypertonic dextrose.

(5) For **hypertension** – Use direct acting vasodilators (**nitroglycerin** or **nitroprusside**), or **α adrenergic antagonists** (e.g. **phentolamine**). **β** adrenergic antagonists are absolutely contraindicated (resultant unopposed **α** adrenergic effect produces life threatening vasospasm and hypertension).

c. Specific management

(1) For **acute coronary syndrome** (cocaine induced coronary vasoconstriction, and MI) – **high-flow oxygen therapy**, **aspirin** (reduces platelet aggregation), **morphine** (reduces cocaine-induced vasoconstriction, preload reduction, reduction of catecholamine release), **nitroglycerin**, diltiazem (for tachycardia, atrial fibrillation). In patients with underlying atherosclerotic heart disease, consider revascularization (**cardiac catheterization**). If cardiac catheterization can not be done, consider **thrombolytic therapy**.

(2) For **wide-complex dysrhythmias** – hypertonic sodium bicarbonate, lidocaine.

5. Additives of cocaine

(1) **Levamisole** - Used to adulterate cocaine because

(i) adds bulk and weight to powdered cocaine [similar appearance to cocaine]

(ii) makes it appear more pure
(iii) possible stimulant effects, and
(iv) an ability to pass street purity tests. It thus acts as a diluent and synergistic adulterant both [**ch 40**].
(2) **Others** - (i) Amphetamine, (ii) local anesthetics, (iii) phencyclidine, (iv) strychnine, (v) thallium. Cases of thallium poisoning after cocaine use have been reported. (vi) Thiamine.

6. Tests for cocaine

(1) **Gold Chloride test** –
(i) Reagent - **Gold Chloride in Acetic Acid**.
(ii) Suspected material + one drop of reagent" characteristic crystals form immediately.
(2) **Scott test** - Suspected material + 5 drops of Cobalt thiocyanate [shake]"Blue colour develops at once.

7. PM appearances

a. External

(1) **perforated nasal septum** – only if the victim was a chronic cocaine addict [For details – please see below under ch cocaine poisoning].
(2) **Nasal swabs** and **injection sites** would reveal cocaine.
(3) **Oropharynx** - **burns** from inhaling superheated fumes of crack cocaine
(4) **Injection site** - Toxic contaminants (particulate matter, strychnine) may be demonstrated.

b. Internal

(1) Nasal passages and nasopharynx - congestion
(2) **GIT** – Mucosal edema, congestion
(3) **Brain** - Subarachnoid, intraventricular and intraparenchymal hemorrhages. These hemorrhages are second to intense vasospasm produced by cocaine.
(4) **Lungs** -
(i) Crack lung – diffuse alveolar hemorrhage [due to pulmonary vasoconstriction " damage to epithelial and endothelial cells], hemorrhagic alveolitis
(ii) Pulmonary infarction – due to vasospasm and thrombosis of pulmonary

artery

(iii) Pneumothorax and pneumomediastinum are sometimes seen. These are not due to cocaine per se, but due to **Valsalva maneuver** that the addict performs after inhalation of smoke (bearing down against a closed epiglottis). This -es intrathoracic pressure. An alveolar bleb may rupture against the pleural or mediastinal surface

(iv) Toxic contaminants (particulate matter, strychnine) may be demonstrated in the lungs.

c. Viscera

Cocaine may be recovered from liver and brain.

D. Chronic Cocaine Poisoning

[syn. **Cocainism**, **cocainophagia**, **cocainomania**] Sniffers generally use **0.5 g** daily.

1. Signs and symptoms

(1) **Magnan's symptom** (**cocaine bugs, formication**) - illusory sensation of ants crawling under the skin. The patient scratches his skin as a result, causing irregular scratches, excoriation and ulcers.

(2) **Nasal septum** -

(i) Perforation – due to necrosis which in turn occurs due to vasospasm and enhanced coagulation. (a) size – varies from a pinhead to several mm (b) Shape – may be round, oval or irregular (c) Seen in 5% of cocaine snorters. (d) Seen also in ch arsenic poisoning [**ch 36**]. (e) Perforation of the nasal septum reduces the nasal support and results in a broad, flat nose (**saddle nose**).

(ii) Granulomas of the nasal septum.

(3) **Perforation of the palate** - Seen more in females. Addicts with a palatal perforation suffer from serious speech impairment. Their speech becomes hypernasal and articulation may be so poor that they can not communicate effectively. In addition, eating and drinking is difficult due to oronasal reflux of both solids and liquids. Some addicts resort to obturating the defect by inserting chewing gum, a piece of white bread or a plug of tissue paper into the palatal defect.

(4) **Tongue** – black.

(5) **Bruxism** (grinding of the teeth accompanied by clenching of the jaw)- occurs frequently in cocaine addicts. Gives rise to pain in the temporomandibular joint and masticatory muscles. Attrition of teeth is seen.

(6) **Crack eye** - **Corneal defects** are seen.

2. Cocaine withdrawal

(1) Bradycardia

(2) Cocaine craving

(3) Dysphoria, depression

(4) Sleepiness, fatigue. Cocaine is probably the best example of a substance to which neither tolerance nor physical dependence develops and with which simply psychic dependence can lead to profound and dangerous levels. Out of 7 types of drug dependence, one is **cocaine type** [**ch 46**].

3. Management [of chronic cocaine poisoning]

Management for cocaine abuse is quite ineffective, especially in comparison with those for other abused substances.

(1) **Drugs for reducing craving** - (a) Amantadine [antiparkinsonian] (b) Bromocriptine [dopaminergic agonist] (c) Aripiprazole – It is a second generation antipsychotic. Blocks cocaine-seeking behaviour, due to its action on dopamine receptors.

(2) **Drugs for both reducing craving and for antidepressant effect** - (a) Desipramine (b) Imipramine (c) Trazodone

(3) **Recent drugs** -

(i) Agonist replacement therapy [sustained-release methylphenidate, d-amphetamine]

(ii) Dopamine agents (disulfiram).

(iii) GABAergic agents [vigabatrin]

(iv) Glutamatergic agents [N-Acetylcysteine, modafinil, topiramate]

(v) Ibogaine [**ch 46**].

(4) **Psychosocial management techniques** – (a) Contingent behavior therapy and (b) Supportive psychotherapy are useful in the post-withdrawal treatment and in prevention of relapse.

(5) **Vaccination** - **TA-CD** is a vaccine against cocaine. It is a cocaine derivative

coupled to the recombinant cholera toxin B (rCTB) carrier protein. It inactivates the cocaine molecule and also inhibits its passage to the brain.

4. PM appearances

As above in acute poisoning.

E. Medicolegal Importance [of Cocaine]

(1) **Brompton's cocktail** is a mixture of cocaine, morphine (or heroin), chlorpromazine and alcohol. It is sometimes provided to patients suffering from pain associated with terminal illness such as cancers. Named after the **Royal Brompton Hospital** in London, England, where it was invented in the late **1920**s for patients with tuberculosis.

(2) **Contaminated currency - ch 46**.

(3) **Prostitutes** sometimes inject cocaine into vagina to produce local constriction.

(4) Rubbed on glans penis sometimes by addicts, as it is wrongly considered to be an **aphrodisiac**.

(5) **Control of illegal coca plants with herbicides** – In several countries [eg Colombia], large tracts of lands where illegal coca plants are grown are spray aerially with herbicides [eg glyphosate]. This may cause off-target spraying [about 1% of the total area sprayed]. Exposures to humans and wildlife is also a concern.

42. Spinal and Peripheral Nerve Poisons

I. SPINAL POISONS

A. Strychnos Nux Vomica

(1) **Active principles:** The **whole tree**, including the seeds, is poisonous. The active principles are alkaloids **strychnine** and **brucine**. Strychnine is about

10-20 times more poisonous than brucine. The alkaloidal content of the seeds ranges from 1.8 to 5.3%. They also contain a glucoside **loganin**.

(2) **Use** - (i) used as **rodenticides**, and for **killing stray dogs**. The commercial baits available contain < 0.5% strychnine and are dyed red or green to make them distinctive. (ii) Once used as a **tonic** and a reflex stimulant of gastric secretion (because of its bitter taste) (iii) **Analeptic and Respiratory stimulant** – But only in toxic doses.

Strychnine – It occurs as colorless, odorless rhombic prisms, and has an intensely bitter taste. Strychnine is one of the **most bitter substance known**; its taste is detectable in a dilution of **1:70,000**.

Fig. 42.1: Seeds of Nux vomica

1. Absorption and excretion

(1) Strychnine is rapidly absorbed from the GIT, nasal mucosa, and all parenteral sites.

(2) It is rapidly metabolized in the liver (up up to 80%) by microsomal enzymes.

(3) The highest concentrations of strychnine are found in the liver, kidneys and blood.

(4) Strychnine taken up by liver and muscles may be released into the blood stream, as its concentration falls. This may produce convulsions on 2nd or 3rd day of poisoning after sedation is discontinued.
(5) About 15% appears unchanged in the urine within 24 hours.
(6) Traces appear in bile, milk and saliva.
(7) **Elimination half life** of strychnine is **10h**.
(8) Strychnine may be found in the cadaver up to **4 y**.

2. Mechanism of action

Strychnine mainly acts at the anterior (ventral) horn cells of the spinal cord, where **glycine** is a major post-synaptic inhibitory neurotransmitter, and prevents its effects. By inhibiting the inhibitory transmitter, it brings about excitation (convulsions).

3. Signs and symptoms

[A] **Uncrushed seeds** - swallowed as such do not cause poisoning, as the hard coat is not dissolved by GIT juices. They are passed as such in the feces. [B] When **crushed seeds** are taken, the symptoms start **after one hour**. [C] When **pure alkaloid** is swallowed, symptoms appear **within 5 minutes.** [D] **Dermal exposure** - Tingling sensation locally. Other symptoms as in ingestion. [E] **Ingestion - Symptoms** are (1) Bitter taste in mouth, a sense of restlessness and uneasiness, anxiety, fear and a feeling of suffocation (2) **difficulty in swallowing** (3) **Prodromal symptoms** – appear before onset of convulsions: (i) -acuity of perception (ii) -rigidity of muscles and (iii) muscular twitchings (4) **Convulsions** - First clonic, eventually become tonic. During convulsions (i) patient has an **anxious look** (ii) **Eyes** - are prominent and staring (iii) **Pupils** - are **dilated** (iv) **Mouth** – is covered with froth, which is frequently bloodstained (v) **Face** - is **cyanosed** (vi) **Duration of convulsions** – ½ to 2 minutes. Breathing stops (spasm of diaphragm, and other accessory muscles of respiration) (vii) **Period between convulsions** – Muscles are completely relaxed. Patient looks relatively better, although severely exhausted. Breathing resumes. Cyanosis .s. Cold perspiration covers the skin. Dilated pupils may contract (viii) **Resumption of convulsions** – Occurs after 5-15 minutes, or earlier if there is slightest sensory stimulus (eg touching the patient, a current of air, lights, noise etc) (ix) **Opisthotonus** - (a) Most common convulsive symptom

(b) Spasms are most marked in antigravity muscles. Results in hyperextension of the body. In supine position, the body may be supported only by the heels and head. The body assumes a ***bow like form***. The arms are flexed over the chest or rigidly extended; hands are tightly clenched and the legs are abducted and extended. (x) **Emprosthotonus** - Sometimes spasms of abdominal muscles are more prominent. This is the "mirror image" of opisthotonus, in which the body is bent forwards (xi) **Pleurosthotonus** - bending of the body sideways, if lateral muscles are in greatest spasm (xii) **Risus sardonicus** - (L. risus, laughter; Gk. sardonius, mocking) Highly characteristic, abnormal, sustained spasm of the facial muscles (appears as if patient is grinning). Results from contraction of jaw muscles. Corners of mouth are drawn back. Spasms may have to be differentiated from tetanus [**Table 1**]. (xiii) **In non fatal cases** – Interval between convulsions -s. Spasms .. Recovery occurs in 1-2 days (xiv) **In fatal cases** - Convulsions rapidly succeed one another. Severity and duration -s. Death occurs usually after **4-5 cycles** of convulsions and relaxations (5) **Effects of Convulsions:** (i) Temp - -ed; up to 43°C (ii) Lactic acidosis – Blood pH may fall to 6.6 "leads to death (iii) Rhabdomyolysis - Myoglobin released in blood "Renal tubular blockage "Renal failure. (6) **Consciousness** – is not lost. Mind remains clear till death (strychnine does not act at cerebral level). Patient is conscious of his impending death, and suffers greatly. (7) **Cause of death:** (i) Medullary hypoxia (ii) spasm of diaphragm leading to respiratory arrest (iii) fall of blood pH (iv) Renal failure due to myoglobinuria.

Table 1: Differences between Strychnine poisoning and Tetanus

S.No	Feature	Strychnine poisoning	Tetanus
1.	History	History of ingesting some substance. No h/o injury	H/o injury present
2.	Onset	Sudden	Gradual
3.	Convulsions	All muscles affected simultaneously	All muscles are not affected simultaneously
4.	Nature of convulsions	First clonic, eventually become tonic	tonic
5.	Lower jaw	Neither starts in, nor particularly affects lower jaw	Usually starts in and especially affects lower jaw
6.	Muscular condition between convulsions	Completely relaxed	Slightly rigid
7.	Fatal period	1-2 h	>24h
8.	Chemical analysis of blood, urine etc	Strychnine detected	Strychnine not detected
9.	Microscopic and culture examination of wound/blood	There is no wound. C. tetani not recovered from blood	C. tetani recovered from the wound in about 30% of cases

4. Fatal dose

(1) One crushed seed (2) Strychnine - 30mg/kg.

5. Fatal period

1-2 hours.

6. Management

(1) **Control of convulsions** - (i) **Room** – Patient should be kept in a dark, noise free room, free of all disturbances [any sensory stimulus can start convulsions] (ii) **Drugs** – (a) **Diazepam** 0.1-0.5 mg/kg slow IV, followed by (b) **Barbiturates** - Phenobarbital IV. Short acting barbiturates [pentobarbital sodium, sodium Amytal in doses of 0.3-0.6 g IV] (c) **Muscle relaxants** (eg. Curare, Gallamine, Mephenesin, Pancuronium bromide, Succinylcholine) (d) **General anesthesia** – if all measures fail to control convulsions. Inhalation anesthetics would fail during convulsions (because respiration stops). During relaxation phase administer any inhalation anesthetic (eg **ether**)

(2) **Gastric lavage and emesis** – Not recommended because of -risk of convulsions.

(3) **Activated charcoal** – to minimize absorption

(4) **After control of convulsions** – Correct fluid, electrolyte and acid-base abnormalities. These would have been caused by repeated convulsions.

(5) **Diuresis** - Opinion is divided regarding forced acidic or alkaline diuresis. Forced acidic diuresis is advantageous in strychnine [**ch 32**]. However urine alkalinization is useful as prophylaxis against renal failure secondary to rhabdomyolysis, which in turn is secondary to convulsions. Physician's judgment is important.

7. PM appearances

(1) Signs of **asphyxia**

(2) **Rigor mortis** – starts early. Prolonged.

(3) **Stomach, duodenum** – congested. Show patches of ecchymoses

(4) All **internal organs** including **spinal cord** - Congested

(5) **Peritoneum** – may show hemorrhages

(6) **Muscles** – show microscopic tears [due to violent convulsions], extravasated blood.

8. Tests

(1) **Sonnenschein's test** - Suspected material + 1 drop of H_2SO_4 + cerosoceric oxide"stir with a glass rod"a deep blue color is formed, changing to violet, and finally to cherry red.

(2) **Wenzell's test** - Suspected material + [$KMnO_4$ – 1 part + H_2SO_4 – 2000 parts]"Color reaction [after **William Wenzell** (1829–1913), an American physician].

(3) **Physiological test** – Aqueous sol^n of Suspected material"Inject into the dorsal lymph sac of a frog"produce tetanic convulsions in a few min.

9. ML importance

(1) Used for **homicide** – in the form of (i) alkaloid (ii) Powdered Nux vomica seeds. It has a very strong bitter taste; thus usually given in alcohol [a pungent drug] to mask its taste.

(2) **Suicide** – Rare, because of painful death

(3) **Accident** – More common (i) Administration by mistake (ii) Eating seeds [children] (iii) Medicinal preparation overdose

(4) Cattle poison - Seeds

(5) Arrow poison

(6) Used as an aphrodisiac.

(7) Used as an **adulterant** in street drugs [eg amphetamines, cocaine and heroin].

(8) **Secondary poisoning** [please see **ch 31**] – Strychnine is used as a rodenticide. Secondary poisoning have been recorded in many non target domestic and wildlife species, who eat rodents killed with strychnine.

(9) **Used as a stimulant and performance enhancing drug** - In low dosages has been used by athletes to enhance their performance.

(i) 1904 St. Louis Olympics - British marathon winner Thomas Hicks took it. There were no restrictions on performance enhancing drugs then.

(ii) 1992 Barcelona Olympics - Wu Dan, female Chinese volleyball player took it.

(10) Strychnine both delays and resists putrefaction [please also see **ch 9**].

(11) Used for extortion – Paracetamol laced with strychnine.

II. PERIPHERAL NERVE POISONS

A. Curare

Curare is a common name for various arrow poisons originating from South America.

Salient features:

(1) Curare is not a single chemical, but a mixture of several chemicals

(2) **Source:** (i) Chondodendron sp [C. platyphyllum; C. tomentosum] (ii) Strychnos sp [S. curare, S. toxifera]

(3) **Types:** (i) Tubocurare [syn, tube or bamboo curare, because it is packed in hollow bamboo tubes]. (a)Active principle: D-tubocurarine [a mono-quaternary alkaloid; isoquinoline derivative]. It is the best known of all active principles of curare (ii) Calabash curare [syn, gourd curare, because it is packed into hollow gourds]. (a) Active principles: Alloferine and toxiferine (iii) Pot curare [because packed in terra cotta pots]. (a) Active principles: protocurarine, protocurine, and protocuridine. Calabash curare is most toxic.

(4) **Mechanism of Action:** (i) Curare is a non-depolarizing muscle relaxant. Occupies nicotinic acetylcholine receptors (nAChR) with greater affinity than Ach, eliciting no response [competitive antagonist]"flaccid paralysis of skeletal muscles. (ii) Action is entirely peripheral, at myoneural junction.

(5) **Toxicokinetics:** (i) Not absorbed through oral or GIT mucosa [molecules are too large and highly charged to pass through GIT lining]. Thus harmless when swallowed. Animals killed by curare tipped arrows are thus safe to eat (ii) Absorbed through wounds or abrasions.

1. Signs and symptoms

The drug has little or no effect if taken by mouth, unless swallowed in very large doses. Symptoms after arrow poisoning are

(1) **Muscular:**

(i) The muscles of the jaw, neck, and head are the first to become weak and relaxed.

(ii) Then the arms and legs begin to feel heavy and difficult to move. Soon it is impossible to move at all.

(2) **Eyes:** (i) Pupils – dilated (ii) Vision – Blurred

(3) **Hearing** - Low tones are heard better because small muscles in the middle ear relax.

(4) **Respiration:**

(i) Becomes harder.
(ii) Victim experiences “shortness of breath,” even with artificial respiration.
(iii) Paralysis of respiratory muscles
(iv) Feels like he is choking because saliva accumulates in the throat.
(5) **Swallowing** – becomes difficult
(6) **CNS** – Headache, dizziness, vertigo. No loss of consciousness and mental faculties are clear till the end. In large doses, short phase of excitation with muscular movements, convulsions and loss of consciousness
(7) **Hypotension** [due to liberated histamine]
(8) **Death** - from respiratory failure and asphyxia.

2. Fatal dose
60 mg.

3. Fatal period
1-2 h.

4. Management
(1) **Artificial respiration** – Effects of curare do not last long, and if artificial respiration is provided, effects begin to wear off after about 20 minutes.
(2) **Antidotes:**
(i) AChE inhibitors - (a) **Physostigmine** [3 ml of 1:200 sol^{n} IV] (b)**Neostigmine** [5-10 mg IV].
(ii) Aminopyridine - Acts by blocking K^{+} channels, prolonging action potentials and thereby -ing neurotransmitter release at the NMJ [please see **ch 39** also].

5. PM appearances
Similar to those of asphyxia.

6. MLI
(1) Curare was introduced for general anesthesia by **Harold Griffith** in 1942.
(2) Used as an arrow poison
(3) **Homicide** – Possible, but has to be administered parenterally. **Case study** - In 1917 **Alice Wheeldon** [1866-1919] of UK and her family were convicted

for planning to murder British Prime Minister **David Lloyd George** by administering curare to him through a spiked nail in his boots [Other imp forensic case involving British Prime Minister - **ch 28**].

B. Conium Maculatum (Poison Hemlock)

(1) Conium maculatum (Poison hemlock, spotted hemlock, poison parsley, spotted cowbane) is a member of the parsley family [Umbelliferae] and is often mistaken for it's edible relatives - fennel, anise and chervil.
(2) It grows to 8 to 10 feet.
(3) It has white, umbrella-shaped flower clusters, which appear in spring and strongly resemble those of Queen Anne's lace [wild carrot].
(4) Stems are hollow, ribbed and purple-spotted.
(5) The leaves emit a strong musty odor when crushed
(6) All parts of the plant are highly toxic.
(7) **Active principles** – Alkaloids present in Hemlock are **coniine** and some congeners.
(8) Coniine content is highest in the (i) leaves [especially at flowering time] (ii) roots [especially during summer] (iii) seeds and (iv) unripe fruit.

1. Signs and symptoms

(1) Burning in the mouth and throat
(2) Nauseating taste and unpleasant mousy odor
(3) Vomiting, diarrhea
(4) R/R.
(5) Pulse-. Later.
(6) **CNS:** (i) Ataxia (ii) Blindness [sometimes] (iii) Mental confusion (iv) Progressive motor paralysis extending upwards from the extremities (v) Tremors
(7) Death from respiratory failure.

2. Fatal dose

60 mg of coniine.

3. ML importance

Socrates, the Greek philosopher was executed in 399 B.C. by forcing him to

drink a cup of poison hemlock. The plants stems show purple mottling, which are sometimes called the “blood of Socrates”.

43. Cardiac Poisons

I. ACONITE

Aconitum [aconite, blue rocket. Devil’s helmet, helmet flower, leopard’s bane, Meetha Bikh, Meetha Bish, Meetha zeher, monkshood, wolfsbane, women’s bane] is a genus of flowering plant belonging to the buttercup family (Ranunculaceae).

Table 1: Differences between root of aconite and root of horseradish

S.No	Feature	Root of aconite	Root of horseradish
1.	Size	Short and conical	Long and cylindrical
2.	Color [external]	Dark brown	Yellowish white
3.	Color [on cutting]	White; becomes pink on exposure to air	White; does not change on exposure to air
4.	Taste	Sweet initially; later acrid	Pungent

A. Active Principles

(1) **Main** - C_{19}-diterpenoid-ester alkaloids - (i) aconitine, (ii) mesaconitine, (iii) hypaconitine, (iv) jesaconitine, (v) Yunaconitine.

(2) **Other alkaloids** - (i) aconine, (ii) bikhaconitine, (iii) indaconitine (iv) lappaconitine (v) picraconitine (vi) pseudoaconitine.

B. Mechanism of Action

Na^+ channel activation - Aconite alkaloids opens up Na^+ channels [by binding at **site 2**] "- Na^+ influx through them "- inotropy while delaying the final repolarization phase of action potential " promotes premature excitation of myocardial muscle. [Tetrodotoxin (**ch 39**) has an exactly opposite action. That is why mixing up the two can delay death]. As a consequence of repeated depolarization, the Na^+ channels become refractory to excitation. In older books this was often described as initial stimulation followed by depression of CNS.

C. Toxicokinetics

Absorbed through all routes. Eliminated mainly through urine, but through other routes as well.

D. Signs and Symptoms

1. Routes other than ingestion

(1) **Eyes exposed to Pollen** " pain and swelling

(2) **Touching** – Leaves or roots handled for a long time or rubbed on the skin "numbness, tingling

(3) **Inhalation** – Root inhaled for a long time has a narcotic effect.

2. Ingestion

Symptoms appear immediately or within a few minutes.

(1) **GIT** -

(i) Burning sensation from the mouth to stomach

(ii) Numbness, tingling in the mouth tongue and pharynx

(iii) Salivation, nausea, vomiting, diarrhea

(iv) Later – Mouth is dry, thirst, dysphagia.

(2) **CVS** –

(i) ECG and heart rhythm changes - (a) Atrial and ventricular arrhythmias, resistant to electric defibrillator. (b) AV block (c) Bundle branch block (d)

Ectopic beats (e) Tachycardia followed by bradycardia (f) Torsades de pointes [Fr “twisting of the points] (g) Ventricular ectopics

(ii) BP.

(iii) Pulse – slow, feeble, irregular.

(3) **Respiratory** –

(i) Feeling of constriction in the chest

(ii) Respirations slow, labored and shallow.

(4) **CNS** –

(i) Consciousness clear. Sometimes there may be hallucinations

(ii) Headache and giddiness

(iii) Numbness and tingling over entire body

(iv) Restlessness

(v) Vertigo.

(5) **Muscular** –

(i) Marked weakness of muscles with spasms and twitchings

(ii) Shooting pain in muscles

(iii) Limbs become weak

(iv) Patient unable to stand or walk

(v) Cramps and convulsions.

(6) **Skin** – (i) Pallor (ii) profuse sweating (iii) cold and clammy skin.

(7) **Other** – temp..

(8) **Eyes** - (i) Diplopia (ii) Impaired vision (iii) Hippus - Pupils contract and dilate alternately. Fluctuations in pupillary diameter [about 1-2 mm] occur with a marked periodicity [5s]. Remain dilated in later stages.

E. Cause of Death

Ventricular fibrillation mainly. May be by respiratory failure.

F. Fatal Dose

(1) **Root** – 1-2 g (2) **ild plant** – 1 g. (3) **Aconitine** – 2 mg. (4) **Extract** – 250 mg

(5) **Tincture** – 5 mL.

G. Fatal Period

2-6 hours.

H. Management

(1) **Gastric lavage** – with warm water and weak sol^{n} of tannic acid, or I_2 in KI. Precipitates alkaloids
(2) **Activated charcoal**
(3) **Atropine** – 1 mg
(4) **Cardiac monitoring**
(5) **Symptomatic**

I. PM Appearances

GIT mucosa – congested, ecchymosed. Stomach may contain root pieces **Viscera** – congested **Toxicological analysis** clinches the diagnosis. Aconitine is extremely unstable and is destroyed by putrefaction. Quick analysis is most important.

J. ML Importance

(1) **Arrow poison** – The name aconite itself derives from Gk akon, dart or javelin.
(2) **Cattle poison**
(3) **Suicide** – Used as a suicidal poison.
(4) **Homicide** –
(i) Used alone - Once very popular with killers, but not very commonly used now. In India, traditionally it was given with betel leaves to conceal its taste. Occasional cases still occur.
(ii) Mixed with other poisons – When mixed with **tetrodotoxin**, the death may be greatly delayed.
(iii) Reasons why aconite was once a popular homicidal poison - (a) Cheap and easily available (b) Lethal dose small (c) Fatal period short (d) Taste is somewhat sweetish. Can be made more acceptable by mixing it in paan (e) Destroyed by putrefaction, hence cannot be detected
(5) **Accidental poisoning** – Caused under following circumstances (a) Usually mistaken for horseradish root, which is edible. (b) Mixed with liquor to - intoxication (c) Used as abortifacient. (d) Used in traditional medicine - Roots [“bushi”] are used in Chinese herbal medical practice to treat rheumatic pain,

paralysis due to stroke, carbuncle and furuncle. May cause accidental poisoning. (e) Used by quacks in India.

II. CINCHONA ALKALOIDS

Cinchona (after a Countess of Chinchon, the wife of a viceroy of Peru) genus belongs to family Rubiaceae, and contains between 30-40 species.

Salient features:

(1) The plant is native to tropical South America. They were transported for cultivation in India by the British in nineteenth century.

(2) The bark of the tree [Jesuit's bark, Peruvian bark] contains **quinine**, **a levorotatory alkaloid**. Its d-isomer is **quinidine**, which is used as antiarrhythmic [and as an antimalarial in some countries].

(3) **Active principles** – The bark contains more than 20 alkaloids, most importantly (i) **quinine**. Other important alkaloids are (ii) dihydroquinine, (iii) quinidine, (iv) dihydroquinidine, (v) cinchonine and (vi) cinchonidine.

(5) **Mechanism of action** – Quinidine blocks myocardial Na^+ channels.

A. Signs and Symptoms

Signs and symptoms after a single large dose or high therapeutic doses taken for a few days are grouped under the term **cinchonism**. The symptoms are:

(1) **Ophthalmologic** - blurred vision, Visual field loss, color vision defects, optic nerve edema, optic atrophy, blindness

(2) **Vestibuloauditory symptoms** – hearing loss, tinnitus, and vertigo

(3) **CNS** – Headache, giddiness, confusion, delirium, seizures, coma, and encephalopathy

(4) **GIT** – abdominal pain, nausea, vomiting, and diarrhea.

(5) **CVS** - arrhythmias, conduction defects, and hypotension

(6) **Hematologic** – hemolysis, anemia

(7) **Skin –** diaphoresis, flushing, erythematous or urticarial rash.

B. Fatal Dose

(1) **Quinine** 2-8 grams; (2) **quinidine** 4-6 grams.

C. Fatal Period

6 hours.

D. Treatment

(1) **Gastric lavage** - of doubtful value because vomiting usually occurs before admission and that quinine is rapidly absorbed
(2) **Activated charcoal**
(3) **Forced acidic diuresis**
(4) **Stellate ganglion block** – causes return of vision immediately
(5) **IV hydrocortisone** – if signs of hemolysis appear
(6) Hemodialysis and peritoneal dialysis – are **useless** as cinchona alkaloids are largely protein bound.

E. PM Appearances

(1) **Organs** - congested
(2) **red cell hemolysis**
(3) **Kidneys**
(i) Renal tubules blocked by hemoglobin.
(ii) Papillae swollen
(iii) Engorgement of the peritubular capillaries
(4) **Liver** – fatty change.

F. ML Importance

(1) **Accidental** - Poisoning is usual accidental due to overdose
(2) **suicide** – rare
(3) **Homicide** – very difficult because of bitter taste. May be mixed with alcohol and given
(4) **Abortifacient**. Please see **ch 26** for its use as an ecbolic
(5) **Urine** - Quinine is often found in the urine of patients who drink **gin and tonic**. Malaria was endemic in India in the 19th century. The British stationed in India needed **quinine** to prevent malaria. Since quinine is soluble in very small quantities of alcohol, they would dissolve it in the gin they drank and to mask the bitter taste, they would use lemon or lime. This was the “gin and tonic” (tonic because quinine preserved health by preventing malaria). By

tradition, tonic water still contains small amounts of **quinine**, although the intention now is never to prevent malaria – it is simply preservation of tradition. Quinine fluoresces under ultraviolet light. Gin containing tonic water glows blue in bars having UV lights.

III. DIGITALIS PURPUREA

(1) English physician **William Withering** (1741 –1799) first learned of the use of ***Digitalis purpurea*** [**Common Foxglove, Purple Foxglove, Lady's Glove],** in congestive heart failure from an old woman who practiced as a folk herbalist in Shropshire. He studied it scientifically in **1775** demonstrating its value in dropsy [edema]

(2) In **1905 James Mackenzie** showed its special value was in heart failure with atrial fibrillation. Digitalis prepared from it is widely used in cardiology today.

(3) In **1930 Sydney Smith** isolated a new glycoside, which he called digoxin, from the Balkan or woolly foxglove Digitalis lanata. Digoxin is extracted directly from the plant, and is not synthesized.

(4) It is thought that the Dutch painter **Vincent Van Gogh** may have been poisoned by digitalis (used at that time as a treatment for epilepsy). His art shows prevalent use of yellow color, which could be because he was suffering from Xanthopsia (yellow vision) due to foxglove poisoning. He also painted his physician, Dr Gachet twice showing him holding a foxglove.

(5) **Etymology** – The word **digitalis** comes from the finger shaped flowers [L, digitus, a finger]. The word **foxglove** comes from an old tradition according to which fairies gave the flowers to foxes to wear on their feet so that they could move in magic silence up to hens or away from men.

A. Active Principles

(1) **Leaves** - (i) Digitoxin**,** (ii) Gitoxin

(2) **Seeds** - (i) Digitalin. It does not contain digoxin, which is present only in the leaves of its cousin Digitalis lanata.

B. Signs and Symptoms

Cardiac – Almost every type of **dysrhythmia** is produced. Most frequent dysrhythmia is **ectopic ventricular rhythm**. **Bidirectional Ventricular Tachycardia** (activity arising alternately from the left anterior and posterior

fascicle, giving rise to “bidirectional” appearance) is virtually diagnostic of poisoning. **Extracardiac** – **GIT** – Nausea, vomiting (due to gastric irritation and CTZ stimulation) Abdominal pain (due to mesenteric vasoconstriction) **CNS** – anxiety confusion delirium depression disorientation drowsiness Fatigue, no desire to walk or lift an arm, mental confusion, restlessness hallucinations headache muscle weakness trigeminal neuralgia **Endocrine** - Gynecomastia. **Skin** – Urticaria **Visual disturbances** - transient amblyopia, blurring of vision, **chromatopsia** (aberrations of color vision), decreased visual activity, photophobia, **photopsia** (perceived flashes of light), scotoma and **Xanthopsia** (yellow halos).

C. Fatal dose

Digitalin – 15-30 mg **Digoxin** [present in D. lanata] – 10 mg **Digitoxin** – 4mg; **Leaves** 1-2 (2g) Therapeutic blood levels for digoxin are 0.5-1.0 ng/mL. Toxic levels for digoxin are >2.0 ng/mL.

D. Fatal period

24 h.

E. Management

(1) **Gastric decontamination** – by emesis or lavage is rarely needed because digitalis is emetic It is rapidly absorbed from GIT. If required, tannic acid is used.

(2) Whole bowel irrigation.

(3) **Activated charcoal** – Even late administration is useful as both digoxin and digitoxin have enterohepatic and enteroenteric circulation. Multiple Dose Activated Charcoal (MDAC) is useful for the same reason **[ch 32].**

(4) **Steroid-binding resins** - eg cholestyramine and colestipol prevent reabsorption from the GI tract. Also interrupt enteroenteric and enterohepatic circulation.

(5) **Digoxin-Specific Antibody Fragments** or **Fab** [Digibind, DigiFab].

(i) **Availability** – Digibind [38 mg/vial] or DigiFab[40 mg/vial] in lyophilized form.

(ii) **Administration** – (a) Vial is reconstituted with 4 mL of sterile water. (b)

Dose – (I) Acute ingestions - 10-20 vials in both child and adult. (II) Chronic toxicity – Adult – 3-6 vials; children - 1-2 vials. (c) **Used in** - Toxicity with digoxin, digitoxin, and all natural cardioactive steroids, eg nerium, oleander, squill, and toad venom.

(6) **If Fab fragments are not available** - Antiarrhythmics - (a) Lignocaine - 1mg/kg IV bolus. Followed by continuous infusion at 1-4mg/min in adult; 20-50 µg/kg/min in child. (b) Phenytoin [dilantin]- 50 mg/min slow IV. Max 1g in adult; 15-20 mg/kg in child. Maintenance oral dose - 300-400 mg/day in adults; 6-10 mg/kg/day in child. (c) Propranolol and Reserpine - have been found useful.

(7) **Management of bradycardia** -

(i) Mechanism of bradycardia and varying degrees of heart block - digitalis induced effects of -ed vagal tone on SA node rhythmicity and on AV node conduction

(ii) Atropine - 0.5 mg IV in adult; 0.02 mg/kg in child. Repeat every 5 min till normalization of heart rate.

(iii) External or transvenous pacemaker - in severe bradycardia.

(8) **Management of extrasystoles and arrhythmias** -

(i) Magnesium - 20mL of 20% sol^{n} over 20 min by slow infusion. To be used with caution in renal failure

(ii) Potassium salts.

(9) **For hypercalcemia** - Trisodium EDTA.

(10) **Hemodialysis** - Does not remove digitalis, but restores serum K^{+} levels to normal.

(11) **Hemoperfusion** - using antidigoxin antibodies.

(12) **Percutaneous cardiopulmonary bypass** - for therapy resistant cardiac arrest.

(13) **Symptomatic** - Electrolyte disturbances must be treated, eg magnesium and potassium levels which may be abnormally low or high.

F. Postmortem Appearances

Fragments of leaves in GIT – if leaves were consumed **GIT mucosa** – inflamed Pulmonary congestion **Confirmatory** - Detection and measurement of Digoxin and other cardiac glycosides is confirmatory.

G. ML Importance

(1) **Bitter taste** - Although foxglove is widely grown as an ornamental plant, few people ingest it because of its bitter taste

(2) **Accidental ingestion** - may occur from **consumption of contaminated water** in or near which plants have been standing or from **smoke from burning plants**

(3) **Mistaken for comfrey** [Symphytum officinale] - Comfrey leaves resemble those of foxglove when the plant is not in bloom. Comfrey is used as a herbal medicine for treating fractures, tendon injury, GIT ulceration, lung congestion, joint inflammation, and promoting wound healing. Comfrey tea is usually taken for these ailments, and leaves must be gathered from wild. Wild-food gatherers mistake the plant for **comfrey**, and use it in their herbal concoctions. This accounts for most cases of toxicity.

(4) **Iatrogenic poisoning** – Can occur during overambitious treatment with digitalis.

IV. NERIUM OLEANDER

Both ***Nerium oleander*** (white or pink Oleander, Kaner) and ***Thevetia peruviana*** (yellow oleander) belong to the Dogbane family, Apocynaceae. **Description** [**Fig 43.1**] -

(1) N. oleander grows wild in India. It is an evergreen shrub that grows to about **6-7 m** in height.

(2) Leaves are lanceolate [spear-shaped], 10-25 cm long, leathery, dark green on upper surface, lighter beneath; display distinct light yellowish veins.

(3) **Flowers**, usually fragrant, are present in clusters at the tip of twigs. They are white to pink to deep red in color [rarely pale yellow], with 5 spreading petals. Width is 2.5-5 cm.

(4) **Fruit** [seed pod] – Slim, cylindrical, narrow, ribbed pod, about 15 cm long. Upon drying it turns brown and splits, releasing many small seeds with brown silky hairs.

(5) **Sap** is thick, gummy and clear

(6) **Nectar** yields toxic honey [please see ch 39 – Food poisoning for more on “toxic honey”].

Salient features:

(1) **Poisonous parts** - (i) All plants of the plant including (ii) nectar are poisonous. (iii) Seeds and roots contain the highest percentage of cardiac glycosides followed by (iv) fruits and leaves.

(2) The total cardiac glycoside content is higher in plants producing **red flowers** than in plants producing white flowers. Highest concentration occurs during the flowering stage.

(3) **Active principles** – (i) **Adynerin**, (ii) **Digitoxigenin**, (iii) **Folineriin**, (iv) **Nerioside** [Nerin] (v) **Oleandrin** (OD). Also present are (vi) **Oleandrigenin** (ODG), a potentially toxic deglycosylated congener of OD, and other glycosides namely (vii) adynerin, (viii) desacetyloleandrin (ix) gitoxigenin, (x) neriantin, (xi) neritaloside, (xii) odoroside H (xiii) Rosagenin (xiv) strospeside and (xv) urechitoxin.

Fig. 43.1: Nerium oleander

A. Mechanism of Action

All active principles mentioned above are cardiac glycosides resembling digitalis

in action, i.e. inhibition of Na^+ K^+ ATPase.

B. Signs and Symptoms

(1) **Contact** – Dermatitis

(2) **Inhalation** – of emanations from flower, especially when fading cause nausea, headache, dizziness and respiratory difficulty.

(3) **Ingestion** – (A) **Symptoms** – (1) GIT - (i) Profuse frothy salivation (ii) Vomiting and Diarrhea (iii) Difficulty in swallowing and articulation (iv) Abdominal pain (2) Neuromuscular - (i) Muscular twitchings (ii) Tetanic spasms (iii) Lock-jaw (iv) Drowsiness (v) Coma. (B) **Signs** - (1) Pupils - dilated (2) Pulse – initially slow. Later rapid and weak. (3) **RR** - Initially -. Later respiratory paralysis. (4) BP. (5) **Heart** - Atrial and ventricular fibrillation, AV block. In severely poisoned patients, fatal cardioversion-resistant ventricular fibrillation or refractory cardiogenic shock may occur. (6) **ECG abnormalities** – [observed both in N. oleander and T. peruviana] (i) lengthened PR segment, (ii) shortened and depressed S-T interval, (iii) absence of P wave (iv) flattened or inverted T wave. (v) ventricular arrhythmias, (vi) bradyarrhythmias, (vii) sinus and AV block. Death occurs from cardiac failure.

C. Fatal Dose

(1) **Root**: 15-20 g

(2) **Leaves**: 5-15 in number

(3) Children have died after ingesting a handful of flowers.

D. Fatal Period

24-36 h.

E. Management

(1) **Emergency measures** - Resuscitate. Monitor pulse, BP, and oxygen saturation. Place on a cardiac monitor and take a 12-lead ECG. Insert IV cannula and give fluids IV.

(2) **Gastric lavage** or emesis followed by **activated charcoal**.

(3) **Treat** marked **hypotension** (systolic <70 mmHg) and **bradycardia**

[<40/min] - Bolus doses of **atropine** (2–3mg) or an infusion (0.6mg/h). Aim is to keep the heart rate around 70–80 bpm.

(4) **Treat hypokalemia and hypomagnesaemia** - until both concentrations are back in the high normal range. Measure serum electrolytes and magnesium on a continuous basis.

(5) **Antidote** –

(i) Specific antidote is **Digoxin-Specific Antibody Fragments** (Fab) [Please see above under "Digitalis"].

(ii) **Indications** [of Fab]- (a) AV node and/or severe sinus node block (b) ventricular tachycardias, (c) serum K^+ > 5.5 mEq/L [Normal levels 3.5 - 5.0 mEq/L].

(iii) **Dose - 400 mg** over 20 min followed by **400–800 mg** over 4–8h by infusion.

(6) **In the absence of anti-digoxin Fab** -

(i) Administer insulin/dextrose

(ii) Do not give calcium

(iii) Temporary pacing – for severe bradycardia due to AV block

(iv) Low energy DC cardioversion – to treat VF.

F. PM Appearances

Not characteristic

(1) Congestion of all organs

(2) Leaves may be found in stomach.

G. ML Importance

(1) **Suicide** – Root, leaves, fruit or seeds are used as a paste or decoction for committing suicide. In one study in Sri Lanka, oleander and paraquat were responsible for 74% of suicides in people under 25 years old.

(2) **Homicide** – Rare because of bitter taste.

(3) **Abortifacient** – Root applied locally or ingested.

(4) **Folk treatment** –

(i) Swellings – decoction of leaves is applied externally

(ii) Venereal diseases - Root is applied locally or ingested

(iii) Cancers and ulcers – Root is applied as paste

(iv) Miscellaneous – Various parts of the plant used in asthma, cancer, cardiac

illnesses, corns, diabetes mellitus, epilepsy and scabies. However, in none of these conditions is there good evidence for genuine cure.

(5) **Cattle poison** – Juice of root is applied on piece of cloth and inserted into the anus of the animal to kill it [cf **ch 37**"calotropis; **ch 38**"snake venom for similar uses].

(6) **Smoke** – from burning plant is toxic and can cause death.

(7) Heating food directly over burning twigs – Can cause death as poisonous sap is transferred to the food.

(8) Arrow and dart poison.

(9) It can be detected long after death.

(10) During the Persian campaign, **Alexander's army** lost horses that had fed on N. oleander, and some soldiers died who had grilled their meat on skewers made from the wood.

V. THEVETIA PERUVIANA

T.peruviana [syn, Cascabela thevetia, Cerbera thevetia, T.neriifolia][common names; be still tree, lucky nut, pila kaner, yellow bells, yellow oleander] is an evergreen tropical shrub or small tree that bears yellow or orange-yellow, trumpet like flowers.

Salient features:

(1) Family - Dogbane family [Apocynaceae].

(2) **Tree** -

(i) Flowers [**Fig 43.2**]– large, yellow, bell-shaped, 5-7 cm long, 5 cm wide; 5 lobes spirally twisted and spreading.

(ii) Leaves –[**Fig 43.2**]– lanceolate [lance like].

(iii) Fruit [**Fig 43.2**]– Globular, light green, 4-5 cm in diameter, encases a single large nut, which is triangular having a deep groove along the edge. Each nut contains 5 pale yellow seeds. All parts of are poisonous. Highest concentration of cardiac glycosides is in the kernel of seeds, followed by leaves, fruit and sap.

(3) **Active principles** – **Neriifolin**, **Peruvoside**, **Ruvoside**, **Thevetin A**, **Thevetin B**, **Thevetoxin** (all are cardiac glycosides resembling digitalis in action, i.e. inhibition of Na^+ K^+ ATPase.

Fig. 43.2: Thevetia peruviana – Please note yellow bell shaped flowers, lance like [lanceolate] leaves and green globular fruit

VI. NICOTIANA TABACUM (TOBACCO)

Nicotiana tabacum, [syn ***tobacco***] is a perennial herbaceous plant belonging to the ***Solanaceae*** family [Other poisonous plants of the Solanaceae family are Datura, A. belladonna, H. niger, S. tuberosum].

Salient features:

(1) The plant - grows to heights between 1 to 2 meters.

(2) Leaves -

(i) Elliptical or oblanceolate

(ii) Dried leaves [tobacco, tambaku] contain 1-8% of nicotine. They are chewed, snuffed or smoked.

(3) Flowers –

(i) are bell shaped [much like those of oleander]

(ii) clustered at the end of the branches

(iii) Reddish in color at their upper parts

(iv) Have a cylindrical calyx

(4) Fruit – has different forms with globular seeds

(5) **Active principles** - (i) Acrolein (ii) **Anabasine** [an alkaloid similar to the nicotine but less active] (iii) Anatabine (iv) Anatalline (v) Anethole (vi) Anthalin (vii) Cembrene (viii) Choline (ix) **Glucosides** [tabacinine, tabacine],

(x) **Nicotelline** (xi) **Nicotine** (xii) **Nicotianine** (xiii) Nornicotine and (xiv) Pyrene

(6) **Nicotine** –

(i) It is a bitter, colorless to pale yellow, hygroscopic, volatile, oily liquid alkaloid, with an unpleasant pungent odor, and sharp, burning, persistent taste. It gradually becomes brown on exposure to air or light

(ii) Uses – extensively used in agricultural and horticultural work, for fumigation and spraying, as insecticides and worm powders etc.

(7) All parts of the plant are poisonous except ripe seeds. Leaves have the highest concentration of nicotine.

(8) **Frequency of use** –

(i) Tobacco is a socially acceptable drug. Among socially acceptable drugs, it is the 2nd most common drug of abuse after caffeine [please see **ch 41** for most common illicit drugs].

(ii) Smoked in cigarettes, bidis; chewed as betel leaf; inhaled as snuff.

A. Toxicokinetics

(1) **Absorption** -

(i) Nicotine is readily absorbed through all mucus membranes, lungs and skin

(ii) One standard cigarette contains – 15-20 mg of nicotine, of which only 1-2 mg is absorbed by smoking [More than half escapes during idling. A large amount remains in the butt and filter].

(iii) An average cigar contains – 15-40 mg nicotine. About 2-4 mg is absorbed.

(2) **Metabolism** -

(i) Nicotine has a high degree of first-pass metabolism, with 70–80% being metabolized in liver to cotinine.

(ii) Some is metabolized in kidneys and lungs.

(3) **Distribution** -

(i) Rapidly and widely distributed throughout most of the body.

(ii) V_d 1–3 L/kg.

(iii) There is also enterohepatic and enteroenteric circulation - Following IV administration, it appears in GIT.

(iv) Freely distributed in breast milk

(v) Plasma protein binding – low [5 to 20%].

(vi) Crosses - blood brain barrier, placenta.

(4) **Excretion** -

(i) Nicotine is predominately excreted by kidneys

(ii) Negligible elimination in sweat or feces.

(iii) Half-life of nicotine - 2 h [range 1–4 h]. Half-life of Anabasine - 15.9 h.

(iv) Urinary excretion of both nicotine and anabasine are pH dependent, with excretion -ed in an acid urine and by high urine output.

B. Mechanism of Action

(1) Nicotine acts agonistically at nicotinic-type acetylcholine (cholinergic) receptors (nAChRs). It is a classic cholinergic agonist.

(2) These receptors exist widely throughout the body, particularly in

(i) Adrenal medulla

(ii) Autonomic ganglia

(iii) Chemoreceptors of aortic and carotid bodies

(iv) CNS [brain stem, limbic system, midbrain, reticular activating system (RAS)]

(v) NM junctions and (vi) Spinal cord.

(3) Direct stimulation of nicotine receptors in endocrine glands + stimulation of neurohumoral pathways in CNS"-ed release of hormones [ACTH, ADH (vasopressin), catecholamines, cortisol, GH, prolactin, serotonin, **β**-endorphins] and enzymes [amylase, chymotrypsin, trypsin].

(4) **At moderate doses** – Nicotine stimulates the RAS"- in alertness"favorable effects of attention and memory.

(5) **At high doses** – CNS disinhibition"convulsions, tremors.

C. Signs and Symptoms

1. Acute poisoning

(1) **GIT** – (i) Nausea, salivation, vomiting [stimulation of CTZ, - in gastroesophageal reflux due to lowering of sphincter pressure and - acid secretion]

(ii) Abdominal pain

(iii) Burning acid sensation

(iv) diarrhea [both central and parasympathetic excitation].

(2) **Cardiopulmonary** –

(i) Initially - -HR, -RR, Hypertension, followed after about 1 hour by bradycardia, hypotension, shock.

(ii) Later – .HR, .RR, hypotension, cardiac arrhythmias.

(3) **RS** – initially bronchorrhea, Tachypnea. Followed after about 1 hour by hypoventilation and apnea.

(4) **CNS** -

(i) Initially - (a) agitation (b) anxiety (c) ataxia (d) blurred vision (e) confusion (f) headache (g) hyperthermia (h) miosis (i) restlessness (j) sweating (k) tremors (l) vertigo.

(ii) Later - (a) hyporeflexia (b) hypotonia (c) lethargy and weakness (d) mydriasis (e) muscular fasciculations and convulsions and (f) coma.

(5) **Eyes** -

(i) Nystagmus

(ii) Pupils – Constricted at first, but may dilate later

(iii) Photophobia and disturbed vision.

2. Chronic poisoning

(1) **General** - anemia, faintness.

(2) **RS** - Asthma, bronchitis, cough, dyspnea, emphysema, pneumonia, wheezing.

(3) **GIT** - anorexia, vomiting, diarrhea, peptic ulcer.

(4) **CNS** - Alzheimer’s disease, impaired memory, tremors.

(5) **Eyes** - amblyopia, blindness.

(6) **CVS** - angina pectoris, arrhythmias, arterial thrombosis, coronary heart disease, extrasystoles, hypertension, stroke.

(7) **In women** -

(i) Osteoporosis - .estrogen levels due to enhanced hydroxylation of estradiol"-ed risk of osteoporosis.

(ii) Obstetric conditions - abortion, abruptio placenta, placenta previa, pre-eclampsia, preterm labor.

(iii) Neonatal effects - congenital malformations, fetal or neonatal death, growth retardation, SIDS.

(8) **Occupation dermal exposure** – to wet uncured tobacco may produce “green

tobacco sickness", characterized by

(9) .appetite; -BMR"Wt loss [reverse symptoms during withdrawal].

(10) **Long term effects** - pulmonary cancer, non pulmonary cancers - brain, mouth, larynx, esophagus, breast, stomach, liver, pancreas, bladder, cervix.

D. Withdrawal Symptoms

(1) **Symptoms** (i) -appetite and wt gain (ii) -RR (iii) -sweating (iv) -urge to smoke (v) Anxiety and depression (vi) Concentration and memory – impaired (vii) Headache (viii) Hostility (ix) Muscle cramps (x) Sleep disturbances.

(2) **Treatment** – Nicotine Replacement Therapy [NRT]. Use of nicotine products [gum, lozenge, inhaler, nasal spray, transdermal patch].

E. Fatal Dose

(1) Nicotine - 50-100 mg

(2) Crude tobacco – 15-30 g.

F. Fatal Period

5-15 minutes. Rivals cyanide [**ch 44**] as a poison capable of producing quick death.

G. Diagnosis

(1) **plasma nicotine level** > 50 ng/mL indicates serious toxicity. Most valuable test [early withdrawal of blood required because of short half life].

(2) sample of suspected plant is taken to a botanist for identification.

(3) **Corroborative** - (i) glycosuria (ii) polymorphonuclear leucocytosis.

(4) Cotinine levels are -ed in tobacco workers and persons exposed to tobacco smoke.

H. Management

1. Acute poisoning

a. Dermal exposure

(1) Dermal exposure to wet tobacco leaves – wash skin thoroughly with soap

and cold running water to reduce absorption.

(2) The medical staff must wear impervious gloves and gowns during these procedures to avoid secondary exposure.

b. Ingestion

(1) **Protect airways** – as vomitus may be aspirated. Also consider assisted ventilation, intubation, oxygen, +ve pressure ventilation.

(2) **Gastric lavage** – with warm water containing charcoal, $KMnO_4$, tannin.

(3) **Whole bowel irrigation (WBI).**

(4) **Activated charcoal** – MDAC [**ch 32**]. Because nicotine undergoes enteroenteric and enterohepatic circulation.

(5) **Emetics** – contraindicated because of active prior vomiting [**ch 32**]. Further vomiting may lead to fluid and electrolyte imbalances and hypovolemic hypotension.

(6) **-ing Elimination** -

(i) Forced acidic diuresis – Since nicotine is a weak base [pKa = 8.0–8.5], excretion can theoretically be enhanced by acidification of the urine. But not recommended because of seizures and rhabdomyolysis. Furthermore symptoms in nicotine poisoning are short-lived.

(ii) Simple fluid diuresis - safer but unnecessary because of limited urinary elimination.

(7) **Antidotes** -

(i) Atropine sulphate – 1-2 mg IM – for bradycardia

(ii) Hexamethonium - ganglionic blocker. Prevents nicotine-induced seizures. Dose – 25-50 mg s/c

(iii) Mecamylamine (Inversine) - competitive and noncompetitive antagonism to the central effects of nicotine. Has been used in nicotine dependence also [please see below]

(iv) Pempidine – action same as mecamylamine.

(8) **General supportive measures** -

(i) Seizure control [benzodiazepines]

(ii) IV fluids, hemodynamic support, vasodilators [dopamine, noradrenaline] for hypotension [**ch 32**].

2. Chronic poisoning

Aim is to . dependence [. craving] and treat signs and symptoms.

(1) **Clonidine** – Also used in alcohol and opiate withdrawal. Mechanism same [**ch 40**]. Dose 150-200 µg/day for 1 month.

(2) **Nicotine partial agonists** – Bind weakly and competitively to nicotine receptor sites .ing craving.

(i) Cytisine - syn baptitoxine, sophorine.

(ii) Lobeline [CigArrest].

(iii) Varenicline (Chantix).

(3) **Nicotine antagonists** - Mecamylamine - start with 5-10 mg/day orally and - progressively until the subject experiences nicotine blockage and/or toxic effects. Only after 3 weeks subjects undergoing therapy either cease smoking totally or . cigarette consumption. Side-effects of therapy are constipation, urinary retention, abdominal cramps, and weakness. These are responsible for drop-outs in some cases. Useful for recalcitrant nicotine dependence.

(4) **Nicotine replacement therapy [NRT]** - (i) Nicotine gum [Polacrilex] (ii) Nicotine spray (iii) Nicotine transdermal patch.

(5) **Immunotherapy** - Vaccines that would generate antibodies to sequester the drug and prevent its access to the brain. Currently under development for 4 major drugs of abuse – cocaine, methamphetamine, morphine/heroin and nicotine.

(6) **Miscellaneous** – Antidepressants – (a) Doxepin, sertraline - combat depression associated with withdrawal of smoking. (b) Bupropion [Wellbutrin, Zyban] - Atypical antidepressant. Also .es severity of nicotine cravings and withdrawal symptoms.

I. Cause of Death

Respiratory failure

J. Postmortem Appearances

(1) Same as those of asphyxia

(2) **skin** – brownish stains

(3) **Mouth and nostrils** – brownish froth

(4) **Lungs** – pulmonary edema

(5) **GIT** –

(i) Stomach - Smell of tobacco, contents show small fragments of leaves, brownish discoloration of stomach wall

(ii) Hemorrhagic congestion.

K. ML Importance

(1) **Application of wet tobacco compresses to cutaneous eruptions** - can result in poisoning.

(2) **Malingering** – Tobacco leaves are soaked in water for some hours"placed in axillae at bedtime [held in place by a bandage]"poisonous symptoms are seen next day

(3) Nicotine resists putrefaction.

(4) **Manner of poisoning** -

(i) Accidental poisoning –due to (a) ingestion (b) excessive smoking (c) application of leaves or juice to wounds or skin

(ii) Suicidal - rare

(iii) Homicidal poisoning – rare, but occurs from time to time. Please see **Bocarmé case** above

(iv) **infanticide** - (a) nicotine is applied over nipples of a nursing mother and baby allowed to suck milk (b) mixed with milk which is given to infant.

(5) **Smugglers** - sometimes transport tobacco by hiding it under their clothes. They suffer from severe nicotine poisoning, as it is absorbed from skin. In one case a convict brought tobacco in his jail cell by smuggling it in his rectum, and suffered severe nicotine poisoning.

(6) **Insecticide** – Nicotine functions as an antiherbivore with particular specificity to insects; therefore nicotine was widely used as an insecticide in the past [in conc up to 40%, in the 1920s and 1930s]. Currently nicotine analogs such as imidacloprid are widely used. May cause accidental poisoning.

(7) Has been used **to control stray dog population** [for euthanizing stray dogs].

(8) used in **animal tranquillizer darts.**

(9) Lead arsenate is used as a pesticide on tobacco crops"risk of Pb poisoning among smokers [**ch 36**].

(10) **Chop chop tobacco** [syn, loose tobacco] – cheap, home grown, illegal, untaxed tobacco, sold in Australia. The term was coined in the mid-1890s. Originates from the fact that it is produced by chopping up the cured tobacco

leaves. It is popular because it avoids heavy excise and taxation levies and is much cheaper than the legal product. It contains several contaminants eg cabbage leaves, chloride products, grass clippings, hay, twigs and pulp from raw cotton. Mould and fungi are also present which cause toxic responses in the lungs, liver, kidneys and skin eg allergic reactions, asthma, chronic bronchitis, legionnaire's disease and lung cancer.

(11) **Miscellaneous** - Nicotine has differential effects on the left- and right-hand sides of developing neural system. Prenatal exposure to tobacco -es tooth crown asymmetry between antimeric permanent teeth [**ch 3**].

44. Asphyxiants and Toxic Gases

Asphyxiants, ***toxic gases***, ***noxious gases***, or ***irrespirable gases*** refer to gaseous poisons, which kill either by displacing oxygen from the environment (e.g. Nitrogen) or by a variety of more complex mechanisms (e.g. Carbon Monoxide, Chlorine). If oxygen is supplied along with the former, the organism would not be affected. It would be immaterial, if oxygen is supplied with the latter; the organism would still suffer ill health or death.

I. CLASSIFICATION

Asphyxiants may be classified as follows: I. **Simple Asphyxiants –** inert gases, which will not have any action on their own, and would not kill if sufficient oxygen were available along with. They kill by excluding oxygen from the lungs. Ex. **nitrogen**, all inert gases (helium, argon, xenon etc), methane. II. **Respiratory Irritants –** injure air passages or both and produce inflammatory changes. Ex. **Ammonia** (NH_3), tear gas, formaldehyde, chlorine, phosgene, nitrogen dioxide, sulfur dioxide. III. **Chemical Asphyxiants** (***systemic asphyxiants***) **–** produce ill health, disease or death, by specialized mechanisms, such as by combining with Hb. Ex. **Carbon monoxide**, hydrogen sulfide, carbon disulfide, hydrogen cyanide gas, CO_2 [earlier CO_2 was thought to be a simple

asphyxiant]. IV. **Volatile drugs –** essentially chemical asphyxiants, with the exception that normally they are used therapeutically. Ex. **Anaesthetic gases**.

II. CARBON MONOXIDE (CO)

Salient features:

(1) **Physical properties** - Carbon monoxide (syn. Exhaust gas, flue gas, carbonic oxide, carbon oxide) is a colorless, odorless, tasteless and nonirritant gas which is lighter than air and insoluble in water. It burns with a **blue flame**, producing carbon dioxide. It is highly toxic to humans and animals in higher quantities.

(2) Normal **atmospheric CO conc** <0.001%.

(3) CO is formed normally in the body as a byproduct of **heme degradation** (via **heme oxidase**); one molecule of CO is produced per molecule of heme degraded. This produces sufficient CO to result in low levels of COHb even in non-smoking persons (up to **1%**). In **hemolytic anemia** this may rise up to **8%**.

(4) CO, like NO, is a **gaseous neurotransmitter** in the CNS; it can diffuse and signal adjacent cells much like nitric oxide (NO). **Endogenously produced CO** serves as a signaling molecule in multiple cellular functions, such as **inflammation**, **proliferation**, and **apoptosis**.

A. Mechanism of Action

CO is readily absorbed after inhalation. COHb levels can be predicted by the **Coburn-Forster-Kane (CFK) model**. Assuming that the weight of individual is **70 kg**, the model predicts the following:

$$\text{COHb}(\%) = \frac{100}{1 + \frac{643}{\text{ppm CO}}}$$

For example breathing 100 ppm CO would result in

COHb(%) =

$$\frac{100}{1 + \frac{643}{100}}$$

or

$$\text{COHb(\%)} = \frac{100}{7.43}$$

or about **14%**.

This is confirmed by actual volunteer experiments.

(1) **Greater affinity to Hb -** The affinity of CO to Hb is approximately **200-250 greater** than that of oxygen. It also binds to other **Fe-containing hemoproteins**, e.g. myoglobin (affinity 60 times greater than that of O_2), cytochrome P450, dopamine hydroxylase and cytochrome oxidase. **10-15%** of body stores of CO in poisoned patients are extravascular, primarily binding to **myoglobin**. Binding to myoglobin explains myocardial impairment in CO toxicity, especially in IHD patients.

(2) **Leftward shift of the oxyhemoglobin dissociation curve** - This .es the release of O_2 from oxyhemoglobin to tissue.

(3) **Nitric Oxide (NO) induced endothelial damage** - Neuronal cell death and neuronal deficits produced by CO can not simply be explained by above mechanisms. This is because comparable levels of anemia (as that produced by CO) fail to produce similar neuronal lesions. The following mechanism is now thought to be a major pathway of CO toxicity: CO displaces NO from platelets"Displaced NO causes endothelial damage to brain microvasculature"perivascular oxidative stress"activation of excitatory amino acids"neuronal cell loss and neuronal deficits. (NO synthase inhibitors have been shown to prevent CO toxicity via this mechanism).

(4) **Apoptosis** – Another mechanism suggested in CO toxicity. CO exposure has been shown to activate **caspase-1**, a protease involved in cell death. (caspase-1 inhibitors prevent CO toxicity).

B. Elimination

CO is **not metabolized** in the body. Nor is it excreted via skin, bile, perspiration, urine or feces.

(1) Half life of CO in a healthy adult breathing 21% O_2 is **4-5 hours** (240-300 min).

(2) If breathing 100% O_2 at NTP"40 min

(3) If breathing 100% O_2 at 2.5 **AT**mospheres **A**bsolute (**ATA**)"20 min.

C. Sources

CO would always be produced whenever there is **fire in confined spaces** limiting the availability of O_2. some common sources which produce CO in this way are:

(1) Coal gas, smoke from fires and fumes from defective heating appliances (gas and wood stoves, furnace, oil lamps, fire places, kerosene and gas water heaters, angithi). Leaking chimneys

(2) Component of the fumes of coke kilns and lime kilns. Heating unit used only occasionally and not well maintained. Barbecues.

(3) Produced after explosion in mines and detonation of explosives.

(4) Exhaust fumes of internal combustion engines.

(5) **Tobacco smoke**. Average levels of COHb in an average **smoker** is about **4%**. In a heavy smoker it may be up to **20%.** A smoker is estimated to be exposed to **400 to 500 ppm of CO** while actively smoking.

(6) Inhalation of fumes of **methylene chloride** (an ingredient of some commercial paint removers) can cause severe intoxication. Fumes of methylene chloride are metabolized within body to produce CO. People using paint removers in poorly ventilated rooms have been poisoned with CO.

D. Acute Poisoning

1. Signs and symptoms

(1) **Symptoms due to tissue hypoxia** - headache, nausea, vomiting, dizziness, lethargy and a feeling of weakness. **D/d:** (i) acute delirium tremens (ii) alcohol poisoning (iii) cerebral vascular disease (iv) flu (v) food poisoning (vi) hysteria (vii) migraine headache (viii) viral fever.

(2) Symptoms develop in severity as conc of COHb -es [**Table 1**].

(3) CO particularly affects organs that have high O_2 utilization (CVS, CNS)

(4) **Infants** - may be **irritable** and feed poorly.

(5) **Skin** - There may be a **cherry red** discoloration of skin.

(6) **CVS** – Chest pain, palpitations, -Heart rate.

(7) **CNS - Neurological signs** include poor concentration, confusion, disorientation, visual disturbance, syncope and seizures.

(8) **Respiratory –** Shortness of breath.

(9) **Ophthalmoscopic examination - Retinal hemorrhages** may be seen.

(10) Exposure during pregnancy may result in **fetal loss**. [More rapid metabolic rate of fetus, high affinity of fetal Hb for CO].

Table 1: Symptoms of CO poisoning

S.No	Level of poisoning	COHb%	Symptoms
1.	Mild poisoning	0-10%	No appreciable symptoms.
2.		10-20%	(i) Breathlessness [on exertion] (ii) headache [mild]
3.		20-30%	(i) Emotional instability (ii) Exertional dyspnea (iii) Headache [throbbing] (iv) Irritability (v) Judgment [disturbed] (vi) Memory [defective] (vii) Rapid fatigue
4.	Moderate poisoning	30-40%	(i) Cherry red coloration (ii) Chest pain (iii) confusion (iv) dimness of vision (v) dizziness (vi) judgment - impaired (vii) manual dexterity ↓ed (viii) muscular control – loss of (ix) nausea and vomiting (x) perspiration (xi) reaction time - prolonged (xii) sensorimotor task performance impaired (xiii) severe headache (xiv) tinnitus or roaring in the ears (xv) vigilance ↓ed
5.	Severe poisoning	40-50%	(i) Ataxia (ii) collapse [on attempts to exert] (iii) confusion (iv) hallucinations (v) respirations - rapid. **Symptoms may resemble alcoholic intoxication.**
6.		50-60%	(i) Coloration – deepens to pink or red; (ii) Muscle spasms (iii) Pulse – weak; (iv) Respirations - rapid; (v) Syncope or coma with intermittent convulsions; (vi) Tachycardia (vii) Trismus
7.		60-70%	(i) Depth of coma ↑es; (ii) hypotension (iii) myocardial ischemia (iv) incontinence of urine and feces (v) ventricular arrhythmias
8.		70-80%	(i) Profound coma (ii) Pulse – weak and thready (iii) Reflexes – depressed or absent (iv) Respirations – irregular, shallow (v) skin blisters [if person is revived and remains in coma for a prolonged period]
9.		>80%	Rapid death from respiratory arrest

2. Delayed sequelae

Neuropsychiatric syndrome [**delayed CO encephalo pathy**]: (i) Seen in about 30% cases. (ii) Occurs 2–40 days later [due to white matter demyelination]. (iii) Main manifestations are (a) Akinetic mutism (b) cognitive deficits [impaired attention, memory (due to **damage to hippocampus**, which is very vulnerable to anoxia and ischemia) and visuospatial skills] (c) dementia (d) psychosis (e) dystonias [and other motor impairments] (f) mood disorders (g) Parkinsonism (h) personality changes (i) suicidal tendencies.

3. Diagnosis and investigation

(1) **COHb levels** – **Most useful** diagnostic test. COHb conc average 1% in nonsmokers and 4% in smokers. >**2% in a non-smoker** and >**10% in a smoker** confirm exposure to CO.

(2) **Direct CO assays** – with IR spectrophotometry. [CO conc 1 mmol/L = 11% COHb].

(3) **Cardiac monitoring and 12-lead ECG** – essential for detecting ischemia and dysrhythmias.

(4) **Scintigraphy of heart with 99mTc -** has been proposed as a method of choice for evaluation of heart injury in patients after acute CO intoxication.

(5) **Creatine phosphokinase** (CPK) – slightly raised. Results from rhabdomyolysis.

(6) **Troponin** - -(diffuse cardiac myonecrosis).

(7) **Serum S100B levels** - -. S100B is a structural protein in astroglia. Released from brain after hypoxic stress.

(8) **Plasma glutathione** - -in rats exposed to CO. Oxidative stress"glutathione release from RBCs (currently under investigation in humans).

(9) **Serum pH** and **lactic acid** levels – Monitor closely. **Anaerobic metabolism** in the presence of **tissue hypoxia** generates lactic acid (**metabolic acidosis**). - lactic acid is a highly reliable indicator of CO poisoning.

(10) **Neuropsychological testing**

(11) **Neuroimaging:**

(i) **CT**- Symmetric **low-density areas** in **globus pallidus**, **putamen**, **caudate nuclei** (within 12 hours of exposure). Negative CT up to 1 wk"favorable outcome.

(ii) **Xenon enhanced CT** – Shows .**blood flow in cerebral cortex**.

(iii) **MRI** – superior for detecting basal ganglia lesions. Periventricular white matter changes.

(iv) **PET** scan - .**blood flow** in frontal and temporal cortex.

(v) **EEG mapping** (vi) **SPECT** – (Single photon Emission computer tomography). Very promising. Shows .**blood flow in cerebral cortex**.

4. Management

(1) **Removal** - from source.

(2) **Airway** - Immediately **secure airway** and ensure **adequate ventilation**.

(3) **Oxygen therapy:**

(i) **Normobaric oxygen [NBO]** – Oxygen at NTP

(ii) **Hyperbaric oxygen [HBO] –** Oxygen at higher pressures, up to 3 ATA. Administer HBO for 90-120 min. Benefits of HBO"(a) .es half life of CO (b) -es amount of dissolved O_2 by 10 times (c) prevents lipid peroxidation in cerebral neurons, thereby minimizing incidence of neurologic damage (d) prevents leukocyte adherence to brain microvascular endothelium (e) accelerates regeneration of inactivated cytochrome oxidase. This is the treatment of choice in patients who present with syncope, coma, or seizure, focal neurological deficit, if COHb > 25% (or >15% in pregnancy).

(4) **Sodium bicarbonate** – If pH < 7.15. Exercise caution while administering $NaHCO_3$, because CO_2, a by-product of its metabolism, could lead to **respiratory acidosis**. Proper ventilation must be maintained.

(5) **Close follow-up** - Required for delayed sequelae.

5. Fatal dose

COHb conc.>60%

6. PM appearances

(1) **Cherry red coloration** – of skin, conjunctivae, mucus membranes, nail beds, all internal organs.

Salient features:

(i) Seen when conc. of CO is > 40%. (ii) In dark colored individuals, seen more easily in the inner aspects of lips, tongue, nail beds, palms and soles. (iii) D/d – CN poisoning, exposure of dead body to cold (iv) With onset of putrefaction – color changes to dark green and then brown.

(2) **Blood** – fluid

(3) Serous effusions in all body cavities

(4) **Skin blisters** [cutaneous bullae] – in wrists, interdigital surfaces of fingers, buttocks, calves and knees. Friction areas are affected more. Complete list of poisons causing blisters – [**ch 40**].

(5) **Trachea** – sometimes soot may be there

(6) **Lungs:**

(i) Congestion

(ii) Pulmonary edema

(iii) Pleural hemorrhages
(iv) Bronchopneumonic consolidation
(7) **Heart:**
(i) Pericardial hemorrhages
(ii) Frank MI [especially in the presence of pre-existing coronary disease]
(iii) Focal areas of necrosis [in delayed deaths].
(8) **Brain:**
(i) Consistency – firmer than normal. Brain retains its shape better after removal from skull
(ii) Meninges – show hemorrhages
(iii) Basal ganglia – necrosis and cavitation, especially in globus pallidus and putamen [in delayed deaths] - considered most characteristic lesions. Other areas affected are cerebellum, cerebral cortex, hippocampus and substantia nigra. This occurs because these areas have limited vascularity and a watershed blood supply.
(iv) White matter – (a) punctiform and ring shaped hemorrhages with widespread edema (b) Ganglion cells – selective cellular necrobiosis.

Fig. 44.1: Cherry red PM staining in CO poisoning.

E. Chronic Poisoning

Chronic CO poisoning involves exposure to lower levels of the CO for longer periods (>24 hours). Typically it results in lower blood CO (COHb) concentrations. Exposure may continue for many days, months, even years, eg in polluted industrial environments. (**Diff**: **acute** - one exposure lasting less than 24 hrs; **chronic** – exposure for more than 24 hrs).

1. Symptoms and signs

(1) Persistent headaches, lightheadedness, depression, confusion, memory loss.
(2) **CVS patients** – symptoms may worsen (chronic hypoxia to heart).
(3) **Permanent neurological damage** (chronic hypoxia to brain).
(4) **Visual disturbances**
(5) **Elevated RBC count**. Symptoms resolve, when removed from source.

2. Diagnosis and investigation

PET scan - metabolism .in the **orbitofrontal** and **dorsolateral prefrontal cortex** and **temporal lobes**.

F. Different Air Concentration of CO and Its Effects

1% CO level (1000 ppm) causes death in 30 min without exertion and in 10 min with exertion. 5% (5000 ppm) kills in 5 min. CO conc >50% (50,000) would kill within seconds. Various %COHb and associated symptoms are given in (**Table 1**)

COHb%	Symptoms
0-10%	No appreciable symptoms
10-20%	Mild headache. Breathless on moderate exertion
20-30%	Nausea, vomiting, throbbing headache, drowsiness, fainting,

G. ML Importance

(1) **Accidental CO poisoning** - is usually found in poor people during winters,

who sleep in small rooms with coal burning inside for warmth. Holiday campers have been found dead, who slept in tents with propane gas stoves or charcoal grills burning inside.

(2) **Bruises** - formed during [or after] CO poisoning are cherry red in color [please also see ch 12 – Mechanical inj].

(3) **COHb levels are -ed in burns**. Presence of COHb is considered the surest sign of antemortem burns [**ch 14**].

(4) **Employer's liability to pay compensation in cases of occupational disease due to CO exposure** – Same as MLI of Arsenic [**ch 36**].

(5) **Hookah use** - Hookah [also known in different cultures as goza, hubble bubble, Narghile, qalyan, shisha, waterpipe] can lead to CO poisoning. A 25-year-old man was brought to the emergency department (ED) after experiencing two syncopal episodes in 1 week. An arterial blood gas analysis was performed which revealed a high COHb level of 31.1%. Both syncopal episodes had developed after hookah use.

(6) **Postmortem production of CO** – CO is produced due to decomposition of Hb and myoglobin. This can cause a false interpretation of death due to CO poisoning. In a case of true CO poisoning spuriously high concentrations [up to 20%] may be recorded.

H. Tests

1. Hoppe-Seyler's test

Blood to be examined (1 vol) + **NaOH** (2 vol)"if COHb present, **solution becomes red**; otherwise dingy brown mass with a green shade. Spreading thin on a white surface such as a porcelain tile brings out the colors better. Developed first by the German Ernst **Felix Immanuel Hoppe-Seyler** (1825–1895).

2. Katayama test

Blood to be examined (10cc) + water (50cc) + orange-red **ammonium sulphide** sol^{n}(4 drops) + 30% **acetic acid** (4 drops). Mixture is filtered. If **COHb** present, **filtrate remains red**. If normal blood present, filtrate becomes green or grey.

3. Kunkel's test (tannic acid test)

Blood to be examined (1cc) + water (10cc) + 3% **tannic acid** (4

drops)"**Crimson red coagulum** indicates presence of COHb.

4. Wetzel's test

Blood to be examined (1 vol) + **water** (4 vol) + 1 % **tannin** sol^{n} (3 vol). If COHb is present, the blood becomes **carmine red**; normal blood slowly assumes a grayish hue.

5. Spectroscopic test

Characteristic absorption bands between **Fraunhofer's lines D and E**.

6. Potassium ferrocyanide test

Blood to be examined (15 cc) + 20% **Potassium ferrocyanide** (15 cc) + **dilute acetic acid** (2 cc)"Shake gently. If COHb is present, **red coagulum** will form; normal blood"dark brown coagulum.

III. CHLORINE

A. General

(1) **Properties** - (i)**Chlorine** is a commonly used highly toxic industrial gas having a greenish-yellow color and an unpleasant irritating odor. It was discovered in 1772, by Carl Wilhelm Scheele. (ii)Oxidizing agent; causes extensive destruction of organic tissue

(2) **Uses** - Berthollet recognized that it could be used to remove color from cloth. Tennant in 1799 made **bleaching powder** (calcium hypochlorite) from it, which was safer to transport, and remains in widespread industrial use. Chlorine is currently used for (i) bleaching of wood pulp newsprint and for (ii) chlorination of water, disinfection and bleaching. (iii) It has been used as a war gas [please see below].

B. Signs and Symptoms

Exposure is likely in laboratories, bleaching powder factories and other chemical works.

1. Acute

a. Symptoms

Has irritating effect on all mucosal surfaces, because of formation of hydrochloric and hypochlorous acids. Cl_2+H_2O"HCl+ HOCl

(1) **Eyes** – intense irritation, redness, lacrimation
(2) **GIT** - Nausea, vomiting, mild gastritis
(3) **Respiratory** – Spasm of glottis, extreme dyspnea, tachypnea, oral mucositis, violent cough, haemoptysis
(4) **General** – dehydration, stupor, and syncope.

b. Signs

-ed respiratory tract sounds and wheezes.

c. Lab

(1) **Complete blood count** - erythrocytosis and lymphopenia.
(2) **Biochemistry** - Serum hepatic enzymes and creatine kinase-
(3) **Arterial blood gas conc** - consistent with hypoxemia and hyperventilation.
(4) **Chest X-ray:**
(i) Widespread pulmonary alveolar infiltrates predominantly affecting the ventral portions of both lungs, consistent with noncardiogenic pulmonary edema.
(ii) Taken in expiration shows an -in lung volume"Indicates pulmonary edema will develop.

2. Chronic

In chemical works chronic poisoning may occur causing anaemia, cachexia, emaciation, progressive wasting, gastritis, dental caries (because of acid production), bronchitis, and emphysema.

C. Fatal Dose

Exposure to 1 part in 1000 causes death in 5 min.

D. Fatal Period

Few hours to 2 days.

E. Management

(1) Remove from toxic atmosphere, fresh air, steam inhalations
(2) supplemental O_2
(3) Nebulized sodium bicarbonate.
(4) IV electrolytes
(5) **General** - treatment of shock, circulatory failure, pulmonary edema [**Atropine** and rapidly acting **diuretic** (frusemide) can counter pulmonary edema].

F. Cause of Death

Cardiac failure following pulmonary congestion and inflammatory edema of lungs.

G. PM Appearances

1. Acute poisoning

(1) Signs of asphyxia
(2) Respiratory tract – Inflamed
(3) Lungs – congested, edematous, alveolar walls – ruptured, hemorrhages and thrombosis in lung beds
(4) GIT – esophagitis, gastritis
(5) Blood - -ed viscosity.
(6) **Chemical tests** - HCl and HOCl may be recovered from tracheal walls [diagnostic].

2. Chronic poisoning

As mentioned earlier under ch signs and symptoms.

H. ML Importance

(1) **Leak from storage tanks** – On **July 14, 2010**, chlorine leaked out from one of the 140 canisters of chlorine lying unattended at the **Mumbai Port Trust**. More than 120 people were admitted with respiratory symptoms, although none died. May lead to negligence suits
(2) **Leak during transportation** - Chlorine can leak during transportation and cause fatalities. Transportation is predominantly by rail car. Rail car accidents

are rare, but potentially catastrophic, as the rupture of a normal 90-ton rail car could release a potentially lethal, **20-mile-wide cloud** of chlorine

(3) **Exposure in factories** and **laboratories**

(4) **War gas** – Used as a war gas. In 1st World War, several thousand soldiers died due to chlorine.

IV. HYDROCYANIC ACID (HCN)

Hydrogen cyanide (HCN) was isolated in **1782** by **Scheele** (1742–1786), who became its first victim in 1786 when he accidentally broke a vial of the chemical in the laboratory and died from vapor poisoning. The name cyanide (Gk Kyaneos, dark blue) is derived from the colorful Prussian blue from which HCN [syn, prussic acid] was first synthesized.

A. Properties

Boiling point is 26°C.

B. Uses

Dyeing, electroplating, fumigants, manufacturing processes [several], metal extraction, metal hardening, metal polishes, pesticides, photography, printing.

C. Mechanism of Action

(1) **Inhibition of cytochrome oxidase** - Cyanide has a **high affinity for Fe^{3+} ions**. In normal cells, cytochrome oxidase has a high concentration of Fe^{3+} ions. CN attaches to cytochrome-C oxidase and stops cellular respiration [causes **histotoxic anoxia - ch 8**].

(2) Other enzymes, which have high concentration of Fe^{3+} ions are **carbonic anhydrase**, **decarboxylases**, **succinic dehydrogenase** and **superoxide dismutase**; these are inhibited as well. Their inhibition has no recognizable relation to the toxic action.

D. Toxicokinetics

(1) **Absorption:**

(i) Rapidly absorbed from GIT, resp system and skin
(ii) Delayed - (a) when taken on full stomach (b) with alcohol

(2) **Conversion** - Cyanide salts are acted upon by stomach HCl to generate HCN. Achlorhydric individuals may be difficult to be poisoned by cyanided salts [please see Rasputin's case below]. However water in stomach may still produce HCN.

(3) **Metabolism** - Cyanide precursors are present in many natural foods [cyanogenetic plant products]. Thus human body has evolved a natural route of CN detoxification. It converts cyanide to thiocyanate with enzyme **rhodanese** (thiosulfate cyanide sulfurtransferase)**,** a mitochondrial enzyme present in great amounts in liver and kidneys, acting as a catalyst. **Glutathione** acts as a sulfur donor [**Fig 44.1**]. Humans can remove **1 mg of cyanide/kg/h** by converting it to thiocyanate.

(4) **Excretion:**
(i) Mainly in urine as thiocyanate
(ii) Small amounts unchanged in breath.

E. Acute Poisoning

1. Signs and symptoms

Most rapid of all poisons. Causes histotoxic anoxia [Gordon's classification"**ch 8**]. Cyanide toxicity should be considered in patients with sudden CVS collapse, especially in the appropriate context of occupational exposure [e.g. laboratory or industrial work] or in a fire victim with hemodynamic instability, -ed lactic acid, or coma.

a. Inhalation

When inhaled as a gas, action is instantaneous. (1) Sense of constriction about the throat and chest (2) Dizziness, vertigo (3) Insensibility (4) Death from respiratory failure.

b. Ingestion

Symptoms may not appear immediately.

i. Small [less than fatal] doses

(1) Nausea (2) Giddiness (3) Headache (4) Confusion (5) Loss of muscular power.

ii. Intermediate [fatal] dose

(1) Symptoms occur immediately.

(2) In some cases appear after 1 min or so. During this time, may perform certain voluntary acts eg corking or throwing away the bottle, or walking a little distance. This may give impression of death being homicidal, eg when a person takes CN and throws a bottle out of the window.

(a) CNS

Initially: (1) Anxiety (2) Confusion (3) Drowsiness (4) Excitement (5) Faintness (6) Headache (7) Perspiration (8) Vertigo. Towards the end (9) Stupor (10) Coma and (11) death.

(b) CVS

(1) **Initially** – hypertension, reflex bradycardia, sinus arrhythmia

(2) **Later** – Hypotension, reflex tachycardia, cardiovascular collapse.

(c) Resp system

(1) **Breath:**

(i) Odor of bitter almonds - (a) Detectable at an air concentration of **1 ppm** by some persons, but up to 50% of population is unable to recognize the odor. (b) It is believed to be due to a genetic defect similar to color blindness. (c) Believed to be common among medical men. (d) Certain firms, where cyanide is used in a manufacturing process will not engage staff unable to smell cyanide. (e) Alternative method for those who cannot smell cyanide – **HCN vapor taints cigarette smoke**, and is thus detected by smoking. Also the flavor of tobacco is rendered a distinctly unpleasant smell. Other poisons making tobacco smell unpleasant are mercury and phosgene. Those able to smell CN should not smoke, because CN odor is very delicate and may be masked by cigarette smoke. (f) Nitrobenzene has similar smell and may cause confusion

(ii) Ammoniacal smell sometimes - KCN"formamide "Formic acid+NH_3.

(2) **R/R:**

(i) Initially - Tachypnea and dyspnea. Due to stimulation of chemoreceptors and respiratory center by CN.

(ii) Later – severe respiratory depression"R/R." Cyanosis.

(d) Neuromuscular

(1) Convulsions [epileptiform or tonic; localized or generalized (more commonly)] (2) Cramps (3) Opisthotonus and trismus (4) Prostration (5) Twitchings (6) Paralysis towards the end.

(e) GIT

(1) Nausea, vomiting [rarely] (2) Salivation (3) Taste - Bitter, acid burning (4) Throat - Constriction, numbness (5) KCN and NaCN cause corrosive burns on mouth, throat and stomach. May cause epigastric pain due to this.

(f) Renal

Acidosis.

(g) Temperature

Hyperthermia.

(h) Skin

(1) Bullae (2) Perspiration.

(i) Eyes

(1) Glassy, prominent (2) Pupils – dilated, unreactive.

(j) In cases of survival

Delayed neurologic sequelae develop especially in the form of Parkinsonian symptoms.

iii. Massive [larger than fatal] doses

(1) Sudden loss of consciousness.
(2) No voluntary act can be performed after ingestion.
(3) Prompt death from respiratory arrest.

2. Fatal dose

a. Ingestion

(1) **HCN** - 50 to 60 mg (2) **NaCN, KCN** – 200 to 300 mg.

b. Inhalation

Air concentration of HCN: (i) 1:50,000 is fatal in few hours (ii) 1:10,000 within few minutes (iii) 1:2,000 almost immediately.

3. Fatal period

(1) **HCN - 2-10 minutes**. Rivals nicotine [**ch 43**] as a poison capable of producing quick death.

(2) **KCN** or **NaCN - 30 min**, because of delay of chemical conversion of salt to HCN by gastric juices.

4. Tests

Lee-Jones Test

5 ml gastric aspirate + Few crystals of ferrous sulphate + 5 drops of 20% NaOH"Boil and cool"Add 10 drops of 10% HCl"Greenish blue colour [or precipitate] which intensifies on standing indicates cyanide, which is due to the formation of **Prussian blue**. Purple color is seen in the presence of salicylate.

5. Management

(1) Treatment should be started immediately because fatal period is very short.

(2) **Principles:**

(i) To restore cellular respiration [by giving 100% O_2 etc]

(ii) To remove unabsorbed CN [by decontamination]

(iii) To reverse cyanide-cytochrome combination [by antidotes].

a. Immediate

(1) Mouth-to-mouth resuscitation should not be attempted.

(2) Clear airways

(3) 100% O_2 by tight fitting face mask or ventilate via endotracheal tube if necessary. There is inconclusive evidence of further benefit from the use of **hyperbaric oxygen**.

(4) Establish iv access.

(5) Check arterial blood gases. Lactic acidosis indicates severe poisoning [**Mechanism**: Oxidative phosphorylation blocked"-ed glycolysis ".ed utilization of lactic acid"lactic acid accumulation].

(6) If poisoning has occurred by inhalation, remove patient at once to fresh air

and start artificial respiration as above.

b. Antidotes

i. Classes

CN Antidotes can be divided in **3 broad classes** - Methemoglobin [MethHb] generators, sulfur donors and direct binding agents. Of these, MethHb generators are given in combination with sulfur donors. This combination is the drug of choice in CN poisoning. Direct binding agents may be given alone.

(a) Methemoglobin generators

(1) **Principle** - to generate an **alternate supply of Fe^{3+} ions**, so CN ions release cytochrome oxidase and attach to fresh supply of Fe^{3+} ions. **Methemoglobin** is an Hb molecule in which Fe is in Fe^{3+} state. Process can be explained biochemically in 3 stages

(i) Stage of cytochrome oxidase inactivation : **CN + Cytochrome oxidase"CN-cytochrome oxidase complex**. This inactivates cytochrome oxidase, stopping cellular respiration

(ii) Stage of MethHb generation: **MethHb generators [eg nitrites] + Hb"MethHb**.

(iii) Stage of cytochrome oxidase recovery: **CN-cytochrome oxidase complex + MethHb"CN-MethHb [cyanmethemoglobin] + Free Cytochrome oxidase**. This happens because MethHb competes with cytochrome oxidase for CN. Since it has greater affinity for CN, it leaves cytochrome oxidase and attaches itself to MethHb.

(2) Methemoglobin [MethHb] generators are **oxidizing agents** that change the ferrous (Fe^{2+}) ion of Hb to the ferric (Fe^{3+}) ion. The resultant **methemoglobin** binds stronger with cyanide than cytochrome oxidase"Forms cyanmethemoglobin.

(3) Levels of MethHb should not exceed **25%**, otherwise death can occur due to MethHb.

(4) The drugs used are

(i) **Amyl nitrite** [$C_5H_{11}NO_2$]

(ii) **Sodium nitrite** [$NaNO_2$]

(iii) **Methylene blue** - Methylene blue (methylthionine chloride) was once

recommended, but is not found to be useful.

(iv) **Phenones** - (a) **4-*Di*Methyl *Amino*Phenol** (4-DMAP) - (I) This German-developed compound is used in the German military and by the civilian population. (II) Dose - 3mg/kg i.v. produces a level of 15% methHb within 1 minute. (III) Disadvantages - (1) IM administration causes necrosis in the area of injection, -in pain, fever, and muscle enzymes. (2) Extremely high levels of methHb may occasionally result. (3) may be a mutagen. (b) **p-aminoheptanoylphenone [PAHP]** (c) **p-aminooctanoylphenone [PAOP]** (d) **p-aminopropiophenone [PAPP]**. PAHP is the safest phenone of the series.

(b) Sulfur donors

Cyanmethemoglobin formed in the above step cannot be detoxified by the body by itself. A sulfur donor is needed for its detoxification. It acts by taking away CN ion from cyanmethemoglobin and forming thiocyanate. This is unlike sulfmethemoglobin, which is formed during treatment of H_2S. Since body can detoxify it on its own, no sulphur donor is needed in H_2S. During treatment of CN poisoning **sodium thiosulphate** is administered which acts as a sulfur donor, with rhodanese catalyzing the reaction [**Fig 44.2**].

Addition of sulfur to the cyanide molecule (formation of thiocyanate) is irreversible. Thus cyanide can not diffuse back in the system. Thiocyanate (though it contains cyanide) is harmless and is excreted in the urine.

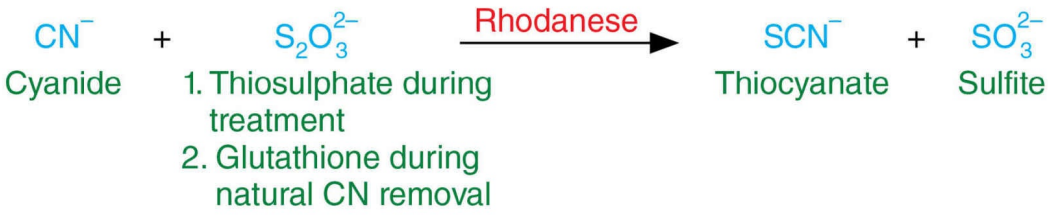


Fig. 44.2: Removal of CN with sulphur donors. Glutathione is a natural sulphur donor used by the body to remove CN produced during normal metabolism. It is however not enough to remove toxic CN ingestions. Exogenous sulphur donor in the form of thiosulphate is needed.

(c) Direct binding agents

Bind directly to CN rendering it non-toxic. **Ex**

(1) **Dicobalt edetate** [Kelocyanor]. Cobalt binds to CN ion directly. Dose - 300mg IV over 1 minute. If there is no response in 1 minute, repeat up to a max. of 900mg.

(2) **Hydroxocobalamin** (syn, **Vit B_{12a}**) [Cyanokit].

(i) **Mechanism** - (a) Combines with CN to form cyanocobalamin [Vit B_{12}] which is renally excreted (b) One molecule of hydroxocobalamin binds one molecule of CN. According to weight, 4 g of hydroxocobalamin combines with 80 mg of CN.

(ii) **Advantages** - (a) relatively safe (b) does not compromise the blood's oxygen-carrying capacity (c) unlike the nitrites or sodium thiosulfate, does not produce hypotension.

(iii) **Dose - 5 g** slow i.v. as infusion in 200 ml saline sol^{n} over 30-min. Infusion repeated if necessary. The pediatric dose is 70 mg/kg, up to a maximum of the adult dose, administered at the same infusion rate. Should not be administered through the same line as other agents, as no data is available on the compatibility of hydroxocobalamin with other substances.

(3) **Cyanohydrin-Forming Drugs:**

(i) Aldehydes and carbonyl-related compounds form **cyanohydrins** from cyanide.

(ii) This action may be a mechanism for improving the protection provided by nitrites.

(iii) Examples: (a) Dihydroxyacetone [DHA] (b) Glyoxal (c) Pyruvate (d) Reducing sugars (e) **α**-ketoglutaric acid [**α**-KG]

(iv) Glyoxal may be the most effective compound of the group.

(v) **Limitations** - (a) relatively short half-lives of reversible reactions (b) high dosage required. Dose of **α**-KG is 2g/kg.

ii. Method of administration

2 Methemoglobin generators and 1 sulfur donor are used in sequence in **three steps. Eli Lilly & Company**, Indianapolis once manufactured the so-called **Lilly antidote kit** or **Lilly Cyanide kit**, which contained all 3 major antidotes of cyanide. This company no longer manufactures it. It is now available from **Taylor pharmaceuticals**, a Division of Akorn, Inc. Each package contains 12 amyl nitrite pearls, two 10-mL vials of 3% sodium nitrite, and two 50mL vials of 25% sodium thiosulfate. This kit is the mainstay of cyanide antidotal therapy.

(1) 0.2 ml **Amyl nitrite** ampoule is broken, a handkerchief soaked in it and patient asked to inhale over it for 15-30 s.

(2) **Sodium nitrite** - 10 ml of 3% given i.v. very slowly over a period of 5 minutes [otherwise there is a risk of developing hypotension, sodium nitrite being a strong vasodilator]. Do not remove needle. All nitrites including amyl nitrite are vasodilators. Vasodilatation is disadvantageous in conditions such as hypotension, hypoxia, hypercapnia and acidosis which are commonly seen in cyanide poisoning; however, their function to form methemoglobin is given priority.

(3) **Sodium thiosulphate** - slow IV infusion of 50 ml of 25% sodium thiosulphate in 1000 ml of 5% glucose solution [infusion rate 5 ml/min].

(4) **Repeat** – both sodium nitrite and sodium thiosulfate at half the initial dose at the end of 1 h, if symptoms persist or reappear.

c. Decontamination

(1) GIT:

(i) Decontamination is typically performed after antidote administration.

(ii) Gastric lavage - Solutions used (a) activated charcoal (b) $KMnO_4$ 1:5000. Leave 200 mL in stomach (c) mixture of citric acid [3%], ferrous sulfate [15%] and sodium carbonate [6%] (d) mixture of sodium bicarbonate, ferrous and ferric chloride; or a mixture of potassium carbonate with ferrous and ferric sulfate. Both act as chemical antidotes. Iron combines with CN to form inactive **Prussian blue** [syn, Ferric Ferrocyanide; Ferric hexacyanoferrate; $[Fe_4[Fe(CN)_6]_3$ or $Fe_7(CN)_{18}]$. Please co-relate with **Thallium poisoning** [**ch 36**], where potassium salt of Prussian blue is given as an antidote. (e) sodium thiosulfate [5-10%]

(2) Skin - Thorough washing of the affected area with soap and water.

d. Exchange transfusion

If methHb levels>50%.

e. Supportive therapy

(1) Observe for 24-48 h as CN toxicity may recur.

(2) For lactic acidosis resulting from anaerobic metabolism - $NaHCO_3$ IV.

(3) Control of seizures - Anticonvulsants eg diazepam.

(4) Cardiopulmonary resuscitation.

6. Cause of death

Respiratory failure.

7. PM appearances

(1) **Precautions** - Pathologist and all paramedical staff should wear masks. Exposure may also be reduced by carrying out autopsy in the open or putting on the exhaust.

(2) **Odor** - Characteristic odor of “bitter almonds” from the body. Cranial cavity is opened first as odor is well marked in brain

(3) **PM staining** – Pinkish, because of oxyhemoglobin, as oxygen could not be utilized by cells. Oxygen remains in the cells as oxyhemoglobin. Color of cheeks may be pinkish.

(4) **Eyes** - Bright, glistening, prominent. Pupils dilated.

(5) **Jaws** - firmly closed

(6) **Blood stained froth** – at mouth, in trachea and bronchi

(7) **Oral and perioral erosions**

(8) **Stomach wall:**

(i) Congested.

(ii) May be eroded [hemorrhagic] or blackened due to formation of **alkaline hematin**.

(9) **Lungs** – edematous

(10) **Serous cavities** - ecchymosed

(11) **Viscera** – congested. May be discolored blue or dark green if methylene blue had been given as a treatment.

(12) **Blood vessels** - All vessels of the body including veins contain oxygenated blood

(13) **CNS** - Degenerative changes

(14) **Chemical examination** – In fatal cases, blood contains about **12mg/L** of cyanide [range 1-53 mg/L].

8. Laboratory identification

HCN is an extremely volatile chemical.

(1) **Blood** – should be covered with a layer of liquid paraffin to avoid

evaporation

(2) **Alkali** - should be added to blood and viscera

(3) **Viscera** - should be stored in well stoppered bottles.

(4) **Analysis** - should be done immediately. Up to 70% may be lost after some weeks of storage from reaction with tissue components and conversion to thiocyanate.

F. Chronic Poisoning

(1) **Etiology** - Produced by continued inhalation of low conc of CN over long periods. Usually occurs in smokers after a long period.

(2) **Symptoms:**

(i) Leber hereditary neuropathy.

(ii) Tobacco amblyopia [progressive loss of visual function]

(iii) Tropical ataxic neuropathy. Not all smokers are affected. Affected smokers have lower plasma cyanocobalamin and thiocyanate concentrations than unaffected smoking counterparts, suggesting a reduced ability to detoxify cyanide. Cessation of smoking and administration of hydroxocobalamin often reverses symptoms.

(iv) Thyroid disorders - Thiocyanate is a competitive inhibitor of iodide entry into the thyroid, thereby causing the formation of goiters and the development of hypothyroidism.

G. ML Importance

(1) **Achlorhydria** – Action of KCN and NaCN depend on the presence of HCl in the stomach (converts them to HCN). Russian politician **Grigori Rasputin** (1869 – 1916) was given pastries laced with KCN, but he survived, because he had achlorhydria [due to chronic alcoholism]

(2) **Blood stain solvent** - KCN is an important solvent for blood stains [**ch 29**].

(3) **Building fires:**

(i) Cyanides are produced during the combustion of many nitrogen-containing compounds [melamine, nylon, synthetic polymers, polyurethane etc]. These are parts of clothes and common domestic furnishings. Their combustion produces HCN.

(ii) Thus fire victims in a burning building [having polyurethane furniture] may

show HCN in their blood; indeed, the greatest danger of CN toxicity is from smoke in **household** or **building fires**.

(iii) Levels – usually up to 0.3mg%.

(4) **Cherry laurel water** - or aqua laurocerasi [distilled from leaves of cherry laurel] contains HCN. Has been used for asthma, cough, indigestion and dyspepsia. Can cause poisoning. The **Arabic emperor Nero** used cherry laurel water to poison the wells of his enemies

(5) **Embalming** – If there is suspicion of CN poisoning, body should not be embalmed [**ch 9**]. Formaldehyde rapidly destroys cyanide. Even containers used to send specimens to lab must not have been used to hold formalin-fixed material.

(6) **Judicial Execution** [**Judicial gassing**]: (i) In some US states, judicial execution is done with cyanide. (ii) Other gases used - Rarely CO and CO_2 are also used. For all official methods of judicial execution, please see **ch 40**.

(7) **Latent Fingerprint development** by cyanoacrylate fuming may generate HCN, especially if cyanoacrylate is heated >200°C.

(8) **Manner of poisoning:**

(i) Accidental poisoning – From (a) **inhalation** of HCN vapors used as a fumigating agent (b) **ingestion** of HCN mistaking it for some drug (c) **injection** of KCN into rectum (d) **Topical application** to a raw wound

(ii) Homicide – rarely.

(iii) Suicide – especially by terrorists [especially LTTE], who hide pellets in hollow teeth.

(9) **Putrefaction:**

(i) Production during putrefaction – Fatal cyanide poisoning has occurred because of **handling spoiled fish**. It is formed by the action of Chromobacterium violaceum and Pseudomonas aeruginosa on fish proteins [please see production of CN by living organisms above].

(ii) Resists putrefaction – has been detected in exhumed bodies upto 18 months after death.

(iii) Retards putrefaction, and acts as a preservative to some extent

(10) **Chemical terrorism and warfare** – CN is an agent that can be used in terroristic activities.

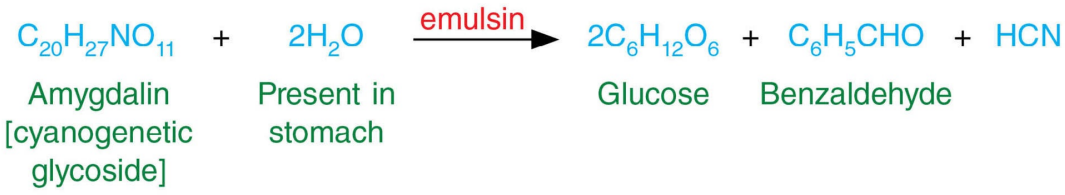


Fig. 44.3: Production of CN by cyanogenetic glycosides within the stomach of humans and animals

V. HYDROGEN SULFIDE (H_2S)

Hydrogen sulfide is a colorless, flammable, gas with the characteristic foul odor of rotten eggs.

Salient features:

(1) **Production:** (i) Natural - It is formed during decomposition of organic substances containing sulfur. Thus foun d in cesspools [an underground reservoir for liquid waste], privy vaults, sewers, swamps, and volcanic gases (ii) Industrial - Produced as a byproduct during distillation of petroleum oil, in manufacture of artificial silk, gas works, glue factories, tannery vats and in other industries where sulphur compounds are used.

(2) **Physical properties** - Heavier than air; tends to collect at ground level.

(3) **Sewer gas** is a mixture of gases [H_2S, NH_3, CH_4, CO_2, SO_2 and nitrogen oxides] produced and collected in sewage systems by the decomposition of organic wastes.

(4) **Sour gas** is natural gas (mined from earth as fossil fuel) containing significant amounts of H_2S. The human body produces small amounts of H_2S and uses it as a **signaling molecule**.

A. Mechanism of Action

(1) **Inhibition of cytochrome oxidase:** (i) Action similar to that of CN and PH_3 [**ch 35**]. H_2S adheres to cytochrome-c oxidase and inhibits its action. Cellular respiration comes to a halt. For this reason, its toxicity and rapidity of action are comparable with that of HCN and even treatment is essentially similar. (ii)

Lack of oxygen to brain causes sudden loss of consciousness [H_2S often known as "**knockdown gas**"].

(2) **Interaction with vital macromolecules** - H_2S interacts with a number of other enzymes and other macromolecules, including **hemoglobin** [leading to formation of **sulfhemoglobin** – responsible for greenish discoloration of putrefaction (**ch 9**)], **methemoglobin** [formation of **sulphmethemoglobin**] and **myoglobin**. Most macromolecules are held together by disulphide bonds, which are easily disrupted by H_2S.

(3) **Olfactory paralysis** - occurs very quickly at higher levels. Although not a mechanism of death, it contributes to death, by removing warning signals.

B. Signs and Symptoms

1. Acute exposure

a. Exposure to low conc [<250 ppm]

(1) Cyanosis

(2) **Irritation of all mucus membranes**

(i) Eye - (a) Cornea opacity (b) Corneal ulceration (c) Keratoconjunctivitis [**Gas eye**]. (d) blepharospasm, lacrimation and photophobia (e) blurred vision (f) colored halos are seen (g) Corneal bullae (h) Ocular pain

(ii) Nose – (a) Olfactory fatigue – occurs 2-15 min after exposure at 100 ppm. Recovery of smell may take several months (b) Rhinitis

(iii) Throat – sore throat

(iv) Bronchus – Bronchitis

(v) Lungs – Acute lung injury, pulmonary edema

(3) **Dullness and sleepiness** – death may result during sleep without the victim regaining consciousness [same as in CO]

(4) **Discoloration of coins** - in the victim's pocket due to oxidation may be a clue to H_2S exposure. [cf lightning where they are molten, fused together or even magnetized – **ch 16**].

b. Exposure to moderate conc [250-500 ppm]

(1) **General** -Nausea, vomiting, diarrhea, feeling of oppression

(2) **CNS** - Amnesia, Disorientation, Dizziness, Headache, Vertigo, Coma

(3) **Resp system** – cough, labored breathing, apnea
(4) **CVS** - Hypotension, Palpitations, Tachycardia
(5) **Skin** – burning, erythema, itching, severe pain, especially in moist area [gas dissolves]
(6) **Muscular** - Muscle cramps, prostration, weakness
(7) **Metabolic acidosis** – secondary to anaerobic metabolism, which in turn results in CNS, respiratory and myocardial depression.

c. Exposure to high conc [750-1000 ppm]

(1) **General** - Weakness, malaise, sweating, abrupt physical collapse [knockdown]
(2) **CVS** – cardiac arrhythmias, conduction defects
(3) **CNS** – amnesia, confusion, delirium, hallucinations, convulsions, Nystagmus, Somnolence 4.Respiratory paralysis, asphyxial seizures, delirium, death.

d. Recovery

Associated with
(1) Impairment of vision, hearing, smell
(2) **Neurological sequelae:**
(i) Memory failure [**amnestic syndrome**]
(ii) Ataxia, rigidity, tremor [due to damage to basal ganglia].

2. Chronic exposure

May occur in industrial workers.
(1) **General** – Headache, Weakness, Nausea, Weight loss
(2) Gas eye is common. Associated with reversible chromatic distortion and visual changes.
(3) **Basal ganglia abnormalities** [ataxia, dystonia, choreoathetosis]
(4) **Spontaneous abortions** in female.

C. Diagnosis

(1) Rotten egg odor from patient
(2) Blackening of copper and silver coins in patient's pockets; darkening of jewellery

(3) -blood sulfide [normal <0.05 mg/L].

D. Scene of Poisoning

At the scene, filter paper moistened with lead acetate will turn black.

E. Fatal Dose

(1) **20ppm** - Maximum allowable conc
(2) **400-700ppm** - dangerous after an exposure of half to 1 h.
(3) **>1000 ppm** - instantly fatal.

F. Fatal Period

Few minutes to few hours.

G. Management

(1) Immediate removal to **fresh air**
(2) Artificial respiration
(3) **Oxygen therapy:**
(i) Hyperbaric oxygen [HBO, **100% O_2** at 2.5 atm]
(ii) High-flow oxygen [**ch 32**]
(4) **Antidote:** Nitrites [dose same as that in CN]"induce methemoglobinemia"H_2S has greater affinity for methHb"releases its hold on cytochrome oxidase and attaches to methHb"formation of sulfmethemoglobin"spontaneously detoxified by body [cf CN in which cyanmethemoglobin is formed, which cannot be detoxified by body on its own. A sulphur donor is needed].

H. Spot Test

A filter paper moistened with **lead acetate** exposed at the scene will blacken if gas is present.

I. PM Appearances

(1) **Odor of rotten eggs** – from the body. Most characteristic sign
(2) **Signs of asphyxia** – especially cyanosis

(3) **Demonstration of sulfmethemoglobin** - in blood
(4) **Brain:**
(i) Edematous
(ii) Greenish discoloration of grey matter [due to sulfhemoglobin]
(5) **Lungs:**
(i) Acute bilateral pulmonary edema
(ii) Chronic passive congestion.
(iii) Cut sections show extremely wet frothy congested surfaces. Color - diffuse red, red-brown or purple
(6) **Tracheobronchial tree:** Lined with a tenacious pale grayish-yellow to grayish-red material. Contains a great amount of mucus which extends deep into the bronchiolar system
(7) **Liver and spleen** - chronic passive congestion
(8) **Color** – of blood and viscera is greenish [due to sulfhemoglobin].

J. ML Importance

(1) **Accidental poisoning** - Poisoning by H2S is almost always accidental [most commonly in sewer workers].
(2) **Suicidal** – rare, but has been described.
Detergent Suicide - In Japan detergent suicide is especially common since 2007. (a) Called so because carried out by mixing 2 common household chemicals – usually detergents - to produce H_2S, one an acid [generally toilet bowl cleaner] and the other, a sulfur source [insecticides or bath salts]. (b) Common availability of this information over the internet has contributed to a dramatic increase in the number of such cases [internet assisted suicide, cybersuicide]. While in 2007, there were only 27 cases of H_2S suicides in Japan; they increased to 517 cases between Jan and July 2008. The number has been continually rising. (c) Typically the suicide would sit in an enclosed environment [eg cars, closets] and post notes warning the first responders [paramedics, doctors etc] of the toxic gas within. Yet there have been reports of injuries to first responders.
(3) **Gas masks** – Workers going down in sewer lines to clean them must wear gas masks. Quick death may occur otherwise
(4) **Stink bombs** – Small phials containing an aqueous soln of the gas are

sometimes used by schoolboys for pranks.

(5) Decomposition occurs faster

(6) **Employer's liability to pay compensation in cases of occupational disease due to H_2S exposure** – Same as MLI of Arsenic [**ch 36**].

VI. WAR GASES AND RIOT CONTROL AGENTS

War gases are gases used to incapacitate or kill enemy (against soldiers and citizens of an enemy country) in times of war. ***Riot control agents*** are gases used by law enforcement agencies during riots (generally against own citizens). Killing is not the aim; it is merely to disperse mob. Thus less toxic agents are preferable. Although the term "war gases" is used, it includes both solids and liquids, which rapidly convert into gas on being liberated. Inhalation causes problems, but direct contact with the liquid or solid forms also causes symptoms. The two are best studied together.

A. Classification

War gases and riot control agents may be classified in two ways.

1. According to persistence [environmental classification]

a. Non-persistent

They are gases, or solids which when liberated are rapidly converted into gas or smoke, which is quickly dissipated, leaving the area free from pollution. Good as riot control agents, but not very good as war gases.

b. Persistent

Persistent gases are solids or liquids which on being exposed to the air change to gas only slowly [eg mustard gas, lewisite]. They thus persist in the environment for long. They are not good as riot control agents, as they can affect citizens for long periods after they have been liberated.

2. According to their physiological action [medical classification]

Given in **Table 2**.

Table 2 [with **Memory Aid 1**]**:** Classification of war gases and riot control agents

VALSHoN

Vesicants or blistering agents

Asphyxiants or lung irritants

Lachrymators or tear gases

Sternutators or nasal irritants

Hemolytics – act on RBCs

Gases with miscellaneous [**o**ther] effects

Nerve gases

a. Vesicants or blistering agents

Vesicants (***blistering agents***) are cytotoxic alkylating compounds, which are rarely lethal but inflict painful burns and blisters, requiring medical attention even at low doses.

Salient features:

(1) **Main blistering agents** are (i) Sulfur mustard [king of the battle gases. Also known as mustard gas] (ii) Nitrogen mustard (iii) Lewisite and (iv) Phosgene oxime (halogenated oxime).

(2) Of these mustard gas and lewisite are most dangerous. They are **liquids**, which continue to give off vapor for days or even weeks.

(3) **Sulfur mustard** – Also known as yellow cross.

(4) Because vesicants can inflict many casualties and create confusion and panic, they were used in battle throughout the twentieth century.

i. Symptoms

Sulfur mustard is a **powerful vesicant**, **mutagenic** and **carcinogenic**. Exposed victims rarely suffer immediate symptoms; they may thus unknowingly receive high dosages. The predominant organs affected are the **skin, eyes, and lungs**.

(1) **Skin** - However, within 24 hours of exposure to mustard agent, victims experience **intense itching** and **skin irritation** which gradually turns into **large blisters filled with yellow fluid**. These are highly debilitating chemical burns.

(2) **Eyes**- Conjunctivitis, swelling of eyelids swell, temporary blindness.

(3) **Lungs** - Bleeding and blistering within the respiratory system; mucous membrane damage; pulmonary edema.

ii. Prevention

Respirators protect the face and respiratory tract against vesicants, but they would still damage any other part of the body they come in contact with.

iii. Treatment

(1) **Decontamination:**

(i) Remove clothing

(ii) Remove any visible agent on the skin [wiping off the agent with dry powders (such as flour, powdered soap, or dirt), showering, washing with soap and water, or using resin decontaminants].

(iii) Bleaching powder - rapidly neutralizes mustard gas and Lewisite, and is used in the form of a jelly, or as a 0.5% hypochlorite solution. (iv) Move to an area free of vapor hazards (v) irrigate eyes with water.

(2) **Antidotes:**

(i) **N-acetyl-cysteine** .es the inflammatory response in mustard exposure

(ii) **Povidone-iodine ointment** – applied within 20 minutes of exposure to mustard liquid protects the skin from vesication

(iii) **BAL** [binds the arsenic group of lewisite].

b. Asphyxiants or Lung irritants

Asphyxiants [syn, respiratory irritants, suffocants], cause irritation of lungs.

Salient features:

(1) **Examples:** (i) Chlorine [please see above for details] (ii) Chloropicrin (iii) Diphosgene (iv) Phosgene.

(2) **Toxicity** - Phosgene [$COCl_2$] is most toxic; chlorine is least toxic. Phosgene is 10 times and Chloropicrin, 4 times more toxic than Chlorine.

(3) The ease with which Chlorine and Chloropicrin are detected by their irritant nature makes them as less suitable war gases.

(4) **Mode of delivery** - gas shells, tanks. Chloropicrin and diphosgene are liquids. Can be used in gas shells.

(5) **Site of action** - lung alveoli

(6) **Signs and symptoms:** (i) General - headache, restlessness, vomiting (ii) RS - coughing, cyanosis, dyspnea, stertorous breathing, tightness of chest (iii) Mucus membranes - watering of eyes (iv) Collapse (v) Death occurs in 24-48

h due to acute pulmonary edema.

(7) **Management:** (i) Eye wash with boric acid (ii) Oxygen (iii) Antitussives (iv) Antibiotics (v) Adrenaline.

c. Lachrymators or tear gases

Lachrymator (L lacrima, "tear"), ***lachrymatory agent*** or ***tear gas*** is a non-lethal chemical compound that stimulates the corneal nerves in the eyes to cause tearing, pain, and even blindness.

Salient features:

(1) **Symptoms:** (i) Irritate all mucous membranes (oral, nasal, conjunctival and tracheobronchial). (ii) Burning sensation (iii) immediate forceful and uncontrollable shutting of the eyes (iv) tears streaming from the eyes (v) coughing (vi) running nose full of mucus (vii) burning in the nose and throat areas

(2) **Examples** – [A] Primarily used as war gases - Principal lachrymators or tear-gases used in **World Wars I and II** were (i) Acrolein (ii) Benzyl bromide (iii) Bromoacetone (iv) Bromobenzyl cyanide (B.B.C.) [liquid and persistent] (v) Chloraceto phenole (C.P.A.) [solid and non-persistent] (vi) Ethyl iodoacetate (K.S.K.) [liquid and persistent. Called so because used by Germany's '**K**ommando **S**pezial**k**räfte'] (vii) Monobromomethyl ethyl ketone (viii) Xylyl bromide [B]Primarily used as riot control agents - (i) **CN** [syn Phenacyl chloride] – (a) Supplied to paramilitary and police forces in a small pressurized aerosol can known as tear gas or "**Mace**" [brand-name]. (b) **Symptoms** – In addition to general lachrymator symptoms mentioned earlier (i) Rarely may cause syncope, disorientation, allergic dermatitis. (ii) At high concentrations causes corneal epithelial damage and chemosis. Rarely death due to pulmonary injury. (c) Because of its greater toxicity, it has largely been supplanted by CS and pepper spray. (ii) **CR** [syn Dibenzoxazepine]. Developed by the British Ministry of Defense as a riot control agent in the late 1950s and early 1960s. (iii) **CS** [syn Orthochlorobenzylidene malononitrile, 2-chlorobenzalmalononitrile, tear gas, $C_{10}H_5ClN_2$] – (a) First synthesized in **1928** by two Americans, **C**orson and **S**toughton. Their first letters gave the name to this gas, but a new name was later derived from this acronym [**C**aptor **S**pray]. (b) Of about 15 different types of tear gases in use, **CS is the most popular** due to its strong effect and lack of toxicity (c) **Symptoms** - general

lachrymator symptoms mentioned earlier. Rarely miscarriages, chromosome changes. Cyanide is formed upon its metabolism. (d) **Treatment** - Washing with 5% sodium bisulfite. (iv) **OC** (from "**O**leoresin **C**apsicum") [syn Pepper spray, capsicum spray] - (a) Active ingredient – Capsaicin (b) Generally non-lethal, may be deadly in rare cases (v) nonivamide (vi) Bromoacetone (vii) phenacyl bromide (viii) xylyl bromide and (ix) syn-propanethial-S-oxide (found in onions).

d. Sternutators or nasal irritants

Sternutators [from Latin sternu***o***, sneeze] or nasopharyngeal irritants are nonlethal agents capable of producing extreme irritation of the nose and throat.

Salient features:

(1) The **symptoms** are comparatively short in duration (i) Intense pain and irritation of not only nose, sinuses and throat, but also of eyes (ii) sneezing (iii) nausea and vomiting [thus sometimes known as "vomiting gases" also] (iv) severe headache (v) salivation (vi) tightness in the chest and prostration

(2) Sternutators are not effective when a mask is worn. However their inhalation before the application of mask makes mask wearing very uncomfortable. Person feels compelled to remove it and thus subject himself to the effects of more disabling gases which usually accompany or immediately follow the use of the sternutators.

(3) **Examples:** (i) Pepper [black and red] (ii) Organoarsenicals eg (a) Diphenylchloroarsine (DA) (b) Diphenylamine chlorarsine [also known as Adamsite or DM] (c) Diphenylcyanoarsine (DC) (d) phenyldichloroarsine

e. Nerve gases (Paralysants)

Nerve gases [syn, nerve agents] are a class of OP compounds.

Salient features:

(1) These are classified as weapons of mass destruction [WMD] by the UN April 1991. Their production and stockpiling is outlawed by the Chemical Weapons Convention of 1993; it officially took effect on April 29, 1997.

(2) **Classes of nerve agents:** (i) G-series - (a) Named so because developed by German scientists. This naming system was created by the US when it uncovered the German activities (b) First and oldest family of nerve agents. (c) **2nd generation chemical weapons** [1st generation - vesicants, asphyxiants

etc] (d) **Non-persistent** in contrast to V series which are **persistent**. (e) All members in this class were discovered and synthesized during or soon after World War II, led by Dr. Gerhard Schrader. (f) Examples - w **GA** [**tabun**] - first to be synthesized in 1936. Origin of tabun and other names except sarin are not clear. Probably represent some kind of code names. w **GB** [**sarin**] in 1939. Discovered by **Schrader** and his team and named after their initials: Schrader, Ambrose, Rüdiger and van der Linde. The term **GC** was not taken because it was already in medical use. w **GD** [**soman**] in 1944 w **GE** - Ethyl sarin w **GF** [**cyclosarin**] in 1949. ABDF were added after G in order of discovery. There are no GC and GE.

Memory Aid 2: Major nerve gases (paralysants)

Take **Sa**ndwich to **S**chool on an **E**nglish **Cye**le - **Ta**bun, **Sa**rin, **S**oman, **E**thyl sarin, **Cye**losarin

(ii) V-series - (a) V stands for Venomous or Viscous. (b) **3nd generation chemical weapons** - Prevalent during cold war years [1947–1991] (c) Examples - VE, VG [Amiton], VM, VR, VX [best known] (d) All V-agents are **persistent**; they do not degrade or wash away easily and can therefore remain on clothes and other surfaces for long periods. This allows V-agents to be used to blanket terrain to guide or curtail the movement of enemy ground forces. (e) They are oily in consistency. Contact hazard is mainly dermal. (iii) Novichok agents - The Novichok [Russian for "newcomer"] agents are a series of OP compounds that were developed in the Soviet Union from the mid 1960s to the 1990s. (a) Belong to "**4th generation chemical weapons**". (b) More toxic than all other nerve agents. (c) Objectives - (I) To be undetectable using standard NATO chemical detection equipment (II) To defeat NATO chemical protective gear (III) To be safer to handle (d) Examples - Novichok-5, novichok-fracture, novichok-7 (iv) Insecticides - phenothiazines, OP [dichlorvos, malathion and parathion]

(3) **Portals of entry:** (i) Respiratory system [because they are easily vaporized or aerosolized] (ii) skin

(4) **Signs, symptoms, management** - same as that of OP [**ch 35**]

(5) **Protection** - Full body suit must be worn in addition to a respirator for protection.

f. Gases with miscellaneous effects

Affect other systems:

(1) **Agents acting on blood:** (i) Arsine [**ch 36**] (ii) Cyanogen (iii) cyanogen bromide (iv) cyanogen chloride (v) HCN (vi) phosgene (vii) vinyl arsine.
(2) **Psycho Agents:** (i) BZ - 3-quinuclidinyl benzilate (ii) K - LSD (iii) SN - Sernyl (PCP)

45. Pharmaceutical Toxicology

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS AND ANTIPYRETIC ANALGESICS

Nonsteroidal anti-inflammatory drugs [NSAIDs] are drugs with analgesic, antipyretic and anti-inflammatory effects. They are non-narcotic. The term "nonsteroidal" distinguishes them from steroids, which have a similar eicosanoid-depressing, anti-inflammatory action. Main **steroidal anti-inflammatory drugs [SAIDs]** are

(1) Dexamethasone [Decadron]
(2) Methylprednisolone [Medrol]
(3) Prednisone [Deltasone]
(4) Prednisone [Sterapred, Sterapred DS].

A. Classification

(1) **Nonselective COX inhibitors** [traditional NSAIDS]
(i) **Salicylates** - (a) Aspirin (acetylsalicylic acid) (b) Diflunisal (c) Salsalate
(ii) **Propionic acid derivatives** - (a) Fenoprofen (b) Flurbiprofen (c) Ibuprofen (d) Ketoprofen (e) Loxoprofen (f) Naproxen (g) Oxaprozin
(iii) **Anthranilic acid derivatives** - Mefenamic acid [Ponstel, Ponstan]
(iv) **Aryl-acetic acid derivatives** - (a) Aceclofenac (b) Diclofenac (Safety alert by FDA)
(v) **Oxicam (Enolic acid) derivatives** - (a) Droxicam (b) Isoxicam (c)Lornoxicam (d) Meloxicam (e) Piroxicam (f)Tenoxicam
(vi) **Pyrrolo-pyrrole derivatives** - (a) Ketorolac
(vii) **Indole derivatives** - (a) I ndomethacin

(viii) **Pyrazolone derivatives** - (a) Phenylbutazone (b) Oxyphenbutazone

(2) **Preferential COX-2 inhibitors:**

(i) Meloxicam

(ii) Nabumetone

(iii) Nimesulide [systemic preparations are banned by several countries for the potential risk of hepatotoxicity]

(3) **Selective COX-2 inhibitors (Coxibs):**

(i) Celecoxib [FDA alert]

(ii) Etoricoxib

(iii) Parecoxib

(iv) Rofecoxib [withdrawn from market]

(v) Valdecoxib [withdrawn from market]

(4) **Analgesic-antipyretics with poor antiinflammatory action** -

(i) Para aminophenol derivates - Paracetamol [acetaminophen]

(ii) Pyrazolone derivatives - Metamizol [Dipyrone], Propiphenazone

(iii) Benzoxazocine derivative - Nefopam

(5) **Others** - Licofelone [dual COX/LOX (Lipoxygenase) inhibitor].

B. Mechanism of Action

(1) NSAIDs are nonselective inhibitors of the enzyme cyclooxygenase (COX). Inhibit both the cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2) isoenzymes. Most NSAIDs inhibit COX reversibly, but **salicylates** inhibit it irreversibly. **Acetaminophen** inhibits COX centrally.

(2) COX catalyzes the formation of prostaglandins and thromboxane from arachidonic acid.

(3) Since COX-1 serves physiological “house-keeping” functions, selective inhibition of COX-2 is preferable

(4) Prostaglandins act as messenger molecules in inflammation.

(5) Inhibition of prostaglandins reduces inflammation.

C. Nonselective COX Inhibitors [traditional NSAIDS]

1. Salicylic acid and derivatives

Preparations of salicylic acid include

(1) **Acetylsalicylic acid** - Please see below
(2) **Methyl salicylate** - colorless liquid, aromatic odor, sweetish taste
(3) **Sodium salicylate** - odorless, white scaly crystals. Unpleasant saline taste.

2. Acetylsalicylic acid [Aspirin]

a. General

(1) It is a white, odorless, crystalline powder, having a slight acidic taste.
(2) Popularly used as an analgesic and antipyretic.

b. Absorption, metabolism, excretion

(1) **Absorption** - is rapid from stomach, because of its low pKa (3.5; 50% is unionized at pH=3.5; only unionized form can be absorbed). Theoretically at higher pH in the intestine, absorption should be less (because of -ionization), but this is offset by the large surface area of the small bowel. Thus absorption is rapid from intestine also.
(2) **Maximum plasma conc achieved in** – 30-60 min.
(3) **Metabolism** - Salicylates are conjugated with glycine and glucuronides in the liver
(4) **Elimination** - by kidneys. More than 30% of ingested salicylate is eliminated as free salicylic acid in alkaline urine and as little as 2% in acidic urine (hence the role of alkalinization of urine in its treatment).

c. Blood therapeutic levels

15-30 mg%. Toxicity appears at >30 mg%. Levels >100 mg% are fatal.

d. Acute poisoning

i. Signs and symptoms

Imp: Many of the signs and symptoms may be mistakenly attributed to the illness for which the salicylates were administered (eg hyperthermia), with disastrous consequences.

(a) GIT

These occur immediately.

(1) Mild burning pain in throat and stomach,

(2) Nausea, vomiting, thirst, diarrhea [occasionally]" Then there may be a latent period of several hours. During latent period, only mild -in temp, sweating and lassitude is seen"After this other symptoms follow [mentioned below].

(b) CNS

(1) Agitation (2) Cerebral edema (3) Coma [rare] (4) Convulsions (5) Delirium (6) Hallucinations (7) Hyperactivity (8) Lethargy (9) Pupils - dilated (10) Stupor (11) Syndrome of inappropriate antidiuretic hormone (12) **Tinnitus** [most pathognomonic] following by diminished auditory acuity or even complete hearing loss (13) Vertigo.

(c) CVS

(1) **Pulse** - rapid and irregular

(2) Hypovolemia, severe dehydration.

(d) Pulmonary

(1) Hyperpnea and tachypnea (2) Respiratory alkalosis (3) Acute lung injury.

(e) Renal

(1) Tubular damage (2) Proteinuria (3) NaCl and water retention (4) Hypouricemia.

(f) Skin

flush and moist.

(g) Metabolic

(1) Diaphoresis (2) **Hyperthermia** [resulting from the uncoupling of oxidative phosphorylation. An indication of severe toxicity and typically a preterminal condition]. Temp may be up to **42°C** (3) Hypoglycemia or hyperglycemia (4) Hypoglycorrhachia [.conc^{n} of glucose in CSF] (5) Ketonemia (6) Ketonuria.

(h) Hemorrhagic diathesis

Because salicylates interfere with coagulation mechanisms.

(i) Acid-base and electrolyte disturbances

(1) Metabolic acidosis [due to -ed excretion of $HCO3^{-}$, Na^{+}, K^{+}] and

(2) Respiratory alkalosis predominate early

(3) Metabolic alkalosis [sets in if there is -vomiting]

(4) Respiratory acidosis [late, grave prognosis]

(5) Hyponatremia or hypernatremia
(6) Hypokalemia
(7) -Anion gap.

(j) Diagnosis

virtually confirmed if there is – Tinnitus + -Anion gap metabolic acidosis + Respiratory alkalosis + Ketonemia.

(k) Reye's syndrome

First described by an Australian pediatrician **Douglas Reye** in **1963**. Occurs if aspirin is given to children **<15 y** with viral fevers usually influenza or varicella. The classic features are (1) rash (2) vomiting and (3) liver damage. Other associated findings (4) encephalopathy (5) fatty infiltration of the viscera (6) hypoglycemia. Potentially fatal. Cause is damage to mitochondria in liver cells.

(l) Late complications

Esophageal strictures.

(m) Idiosyncratic reactions

Occur in 0.2% patients. Even therapeutic doses produce alarming symptoms (1) Angioneurotic edema (2) Bronchial spasm (3) Cyanosis (4) Erythema of face with edema of eyelids (5) GIT hemorrhages [due to capillary damage] (6) Hypotension (7) Maculopapular exanthemata (8) Mucus membranes - edema with hypersecretion [eg excessive salivation, laryngeal edema]. Erosion and ulceration in severe cases (9) Rhinitis [vasomotor] (10) Urticaria (11) Vomiting.

ii. Laboratory investigations

(1) **Blood:** (i) Hypokalemia (ii) hypoprothrombinemia (iii) Prolonged clotting time, prothrombin time [PT].
(2) **Urine:** (i) strongly acidic. (ii) Contains (a) acetone (b) albumin (c) bile [traces] due to hepatitis.

iii. Management

(1) Emetics
(2) **Gastric lavage:**
(i) When a large number of tablets have been ingested, they may form a large dirty gray lump [bezoar], which may not pass for a long time [lumps not

easy to pass + pylorospasm].

(ii) Lavage with $NaHCO_3$ is helpful in dissolving such bezoars.

(3) **Activated Charcoal (AC):**

(i) 1 gram of AC adsorbs 550 mg of salicylic acid.

(ii) A 10:1 ratio of AC:salicylate ingested results in maximal efficiency.

(iii) Multiple-dose activated charcoal (MDAC) is better than single-dose AC.

(4) Cathartics

(5) **Urine alkalinization** - [**ch 32**].

(6) Peritoneal dialysis

(7) **Hemodialysis** – Very effective.

(8) **Exchange transfusion** - in severe cases

(9) **For idiosyncratic reactions** - ACTH, antihistamines.

(10) **Supportive measures:**

(i) Cerebral edema and -ICP [correction of] - **ch 32.**

(ii) Convulsions - **ch 32**.

(iii) Fluid and electrolyte imbalance [correction of]

(iv) Hemorrhages [control of] - (a) blood or blood products - fresh frozen plasma (b) Vitamin C (c) Vitamin K - 2.5-5mg IV daily to correct hypoprothrombinemia

(v) Hyperpyrexia [treatment of] - **ch 32**.

(vi) Hypocalcemia [treatment of] - **ch 32.**

(vii) Hypoglycemia [correction of] - **ch 32.**

(viii) Metabolic acidosis [correction of] - with $NaHCO_3$.

iv. Fatal dose

(1) Sodium salicylate and aspirin = 15-20 g

(2) Salicylic acid = 70-80 g

(3) Methyl salicylate=10-20 ml. For fatal blood levels, please see “therapeutic levels” above.

v. Fatal period

Few minutes to several hours

vi. Tests

(1) Ferric chloride test - 1mL of urine + few drops of 10% ferric chloride"purple color indicates salicylates
(2) Trinder spot test - 1mL of urine + 1mL of Trinder reagent"purple color indicates salicylates.

vii. Cause of death

Mortality rate is 7-8%.

(1) **Early stages:** (i) Acidosis (ii) peripheral failure due to shock (iii) uremia
(2) **Late stages:** (i) Cardiac arrhythmias and sudden cardiac arrest can occur anytime up to a day or so (ii) Respiratory failure.

viii. PM appearances

(1) **Pupils** – dilated
(2) **Skin** – Petechia, rashes
(3) **CNS** - cerebral edema
(4) **Heart** - Subepicardial hemorrhages
(5) **Lungs:** (i) Pulmonary edema and some collapse (ii) subpleural hemorrhages
(6) **GIT:** (i) congested, (ii) erosions, (iii) hemorrhagic gastritis, (iv) black altered blood in stomach, (v) concretions of tablets [sometimes]
(7) **Liver** – Hepatitis
(8) **Renal** – Widespread shedding of renal tubular cells into the calyces of kidneys and into the urine.
(9) **If patient survives for a few days** – the myocardium, liver and kidneys are soft, dirty in appearance and greasy to touch.

e. Chronic poisoning [Salicylism]

Chronic salicylate poisoning most typically occurs in the elderly as a result of unintentional overdosing on salicylates used to treat chronic conditions such as rheumatoid arthritis and osteoarthritis.

Signs and symptoms

(1) Hearing loss and tinnitus, nausea, vomiting, dyspnea and hyperventilation, tachycardia, hyperthermia, and neurologic manifestations such as confusion, delirium, agitation, hyperactivity, slurred speech, hallucinations, seizures, and coma.

(2) **Delayed recognition** - The slow onset and lesser severity of signs in the elderly often causes delayed recognition of the true etiology of the patient's presentation.

(3) **D/D** - Typically, ill patients who suffer from chronic salicylate poisoning may be misdiagnosed as having delirium, dementia, encephalopathy of undetermined origin, diseases such as sepsis (fever of unknown origin), alcoholic ketoacidosis, respiratory failure, or cardiopulmonary disease, especially congestive heart failure, acute pulmonary edema, or unstable angina.

f. ML Importance

(1) Almost always suicidal. Because of bitter taste, ingestion of large quantities cannot occur accidentally

(2) **Frequent monitoring** – Elderly people who are chronically taking aspirin for painful conditions must be frequently monitored. Failure of recognition of symptoms may result in allegations of negligence against doctors.

D. Analgesic-Antipyretics with Poor Antiinflammatory Action

1. Paracetamol (Acetaminophen)

a. General

(1) Paracetamol is an aniline analgesic.

(2) It is the active metabolite of phenacetin, once itself popular as an analgesic and antipyretic. But unlike phenacetin, paracetamol is not a carcinogenic at therapeutic doses.

(3) The words **paracetamol** (used in India) and **acetaminophen** (used in the US, Canada and some other countries) both come from chemical names for the compound: **par**a-**acet**yl**am**inophen**ol** and para-**acet**yl**aminophen**ol. The chemical name can also be written as N-**a**cetyl-**p**ara-**a**mino**p**henol; it is then known as APAP.

b. Metabolism

Absorbed rapidly from GIT, and metabolized quickly in the liver, via four pathways (1) glucuronidation (60%) (2) sulfation (35%) (3) conjugation with

cysteine (3%) (4) Cytochrome P-450-mediated N-hydroxylation to form **N-acetyl-p-benzoquinoneimine (NAPQI)** (1%).

c. Mechanism of toxicity

NAPQI is a strong oxidizing agent. It binds covalently to hepatocytes, causing liver necrosis and occasionally to renal cells causing renal damage. It is normally detoxified by conjugation with reduced glutathione and excreted in the urine as mercapturic acid and cysteine conjugates.

d. Signs and symptoms

i. Acute poisoning

The usual clinical course can be divided into three phases.

(1) **Phase I** – (½-24 hr). Nausea, vomiting, diaphoresis, pallor, mild drowsiness.

(2) **Phase II** – (24-48 hr) symptom free. LFT abnormal

(3) **Phase III** – (3-5 days) Symptoms of severe hepatic necrosis [eg asterixis (coarse flapping tremor of hands)] with jaundice, Right upper quadrant tenderness, coagulation defects, hemorrhages, fetor hepaticus, hypoglycemia, hepatic encephalopathy, and in rare cases, renal failure and cardiomyopathy.

ii. Chronic poisoning

(1) **Circumstances** - Occurs when an individual consumes large doses of paracetamol over years for control of pain.

(2) **More common in** - alcoholics, AIDS patients [in whom there is depletion of glutathione], patients receiving cytochrome P450 inducers [barbiturates, carbamazepine, isoniazid, phenytoin, rifampicin], and in children where dosages have been wrongly calculated.

(3) **Clinical features:** (i) .temp (ii) Anorexia (iii) Hepatomegaly (iv) Lethargy (v) oliguria (vi) Vomiting

e. Fatal dose

(1) 20-25 g(40-50 tablets). 20 tablets in normal persons may produce severe hepatotoxicity

(2) more dangerous in alcoholics, so they succumb at lower doses

(3) Children <10 y are more resistant.

f. Fatal period

2-4 days.

g. Management

(1) Gastric lavage

(2) Activated charcoal (should not be given if NAC or methionine are intended to be administered, as it would bind them)

(3) **Antidotes** - (a) **N-acetyl cysteine** or NAC (Mucomist, Mucosil). (i) Oral loading dose 140 mg/kg followed by 70 mg/kg every 4 hours for 17 doses [total 18 doses] (ii) The golden time for administration is the first 8 h after overdose. (I) Also effective (II) NAC has a foul smell and taste. Thus if given orally it must be diluted with soft drinks to make a 5% sol^{n}. The sol^{n} must be consumed within one hour of preparation. (III) Mech of Action - NAC prevents paracetamol toxicity by following mechanisms (i)Prevents binding of NAPQI to hepatocytes (ii) Directly binds to NAPQI (iii) Enhances synthesis of additional glutathione (iv) Acts intracellularly as a glutathione substitute (v) Reduces NAPQI back to paracetamol. (IV) NAC within paracetamol - It has been suggested that for high risk populations such as psychiatric patients [particularly those with depression and personality disorders who may attempt suicide by AP], paracetamol tablets should incorporate a sustain release preparation of NAC. (b) **Methionine** - (i) Oral antidote. (ii) Glutathione precursor. (iii) Dose – 2.5 g at 4 hourly intervals up to 4 doses. (c) **Cysteamine** - Once popular, but not now, because efficacy is less if administered late (>10 h after ingestion).

(4) **Liver transplantation** – When fulminant liver failure develops.

(5) **Treatments of *no use* in paracetamol** - (a) Forced diuresis - as only 5% is excreted in urine. (b) Hemodialysis - reduces the half-life of paracetamol, but there is no evidence that it alters the clinical course. (c) hemoperfusion with activated charcoal (d) peritoneal dialysis. These lines of treatments should be avoided.

h. PM appearances

(1) Acute centrilobular hepatic necrosis

(2) Acute tubular necrosis

(3) Necrosis of myocardium
(4) Cerebral edema.

i. ML importance
(1) Mostly suicidal
(2) Rarely accidental
(3) Homicidal poisoning virtually unknown because of large dose required
(4) **Organ donation after paracetamol poisoning** – Can be done.

46. Drug Dependence and Abuse

I. INTRODUCTION

A. Definitions

1. Drug
Any substance, other than that required for the maintenance of normal health, which when administered to a living organism modifies one or more of its functions.

2. Drug habituation
Psychological and emotional dependency resulting from repeated consumption of a drug (caffeine in tea and coffee, nicotine in cigarettes).

3. Drug addiction
Physical, psychological and emotional dependency resulting from repeated consumption of a drug (opium, cocaine, cannabis). Compulsive use - There is compulsive use of drugs (**craving**) resulting in physical, psychological and social harm. Use is continued despite awareness of that harm. **Relapse** - Relapse is the return to drug taking following extended periods of abstinence. Relapse is

characteristic of addiction and is a major problem for its treatment. **Table 1** lists major differences between drug habituation and drug addiction. Drugs known to cause addiction - include illegal drugs as well as prescription or over-the-counter [OTC] drugs [**Prescription drug abuse**, Kiddie dope].

Table 1: Difference between drug habituation and drug addiction

S.No	Trait	Drug addiction	Drug habituation
1.	Compulsion to take drug	Present. May resort to crime to acquire drug	A psychological desire but no compulsion
2.	Dose	Tendency to increase dose with time.	No such tendency. An individual who was comfortable with a cup of tea a year ago, would still be comfortable with the same amount
3.	Dependence	Mainly physical	Psychological
4.	Withdrawal symptoms	Characteristic symptoms	None
5.	Preoccupation with drug use	Important alternative pleasures or interests given up or reduced because of drug use	Preoccupation with drug use is not there
6.	Time spent	Great deal of time spent in activities necessary to obtain, take, or recover from the effects of the substance	Not so
7.	Persistent use despite clear evidence of harmful consequences	Yes	Not so
8.	Harm	Both to individual and society	Harm minimal. If any, only to the individual

4. Drug dependence

Drug dependence includes both drug habituation and drug addiction. The terms drug habituation and drug addiction were first defined in **1957** by World Health Organization (WHO) Expert Committee on Addiction-Producing Drugs. In **1964**, a new WHO committee suggested the term drug dependence.

Types of Drug dependence

A proposal has been made to classify **drug dependence** into **7 types**, but it has not been widely accepted. The suggested types are (1) Morphine Type (2) Barbiturate-Alcohol Type (3) Cocaine Type (4) Cannabis (Marihuana) Type (5) Amphetamine Type (6) Khat Type (7) Hallucinogen (LSD) Type.

i. Morphine type

Characteristics:

(1) Psychic dependence - Strong; an overpowering drive or compulsion to continue taking the drug and to obtain it by any means, for pleasure or to avoid discomfort.

(2) Physical dependence - Develops early. -es in intensity, paralleling - in dosage. This requires a continuation of administration of the same drug, or a congener, to prevent the appearance of the symptoms and signs of withdrawal. Withdrawal of the drug or administration of a specific antagonist precipitates a definite, characteristic, self-limited abstinence syndrome.

ii. Barbiturate-alcohol type

(1) This type has been named thus, because the signs and symptoms of alcohol and barbiturate intoxication are similar [**ch 40**].

(2) Within this type, loose subtypes of the two drugs have been described [**barbiturate type** and **alcohol type**] because there are psychological and sociological differences between the two.

(3) **Characteristics** of **barbiturate** type - Similar to morphine type dependence.
Differences -

(i) In contrast with morphine type, there is a dose limit up to which a person can become tolerant in barbiturate type. This limit is characteristic of the individual and varies widely.

(ii) Following withdrawal of barbiturates, tolerance is rapidly lost. Indeed some patients may become more sensitive to barbiturates than they had been earlier.

(4) **Characteristics** of **alcohol** type - Overt pathology in tissues; a similar development with barbiturates [or morphine] has not been demonstrated.

iii. Cocaine type

Characteristics:

(1) Psychic dependence - Strong

(2) Physical dependence - None; therefore absence of a characteristic abstinence syndrome when the drug is withdrawn.

(3) Tolerance - Absent; rather, there is sensitization to the drug’s effects in some instances.

(4) A strong tendency to continuation of administration, as in coca-leaf chewing, or rapid repetition of the dose, as in the current practice of IV administration.

Quantitatively, the effects are strikingly different, according to the mode of abuse. [please also see **ch 41**].

iv. Cannabis (Marihuana) type

Characteristics:

(1) Psychic dependence - Moderate to strong because of desired subjective effects.

(2) Physical dependence - None; so that there is no characteristic abstinence syndrome when the drug is discontinued.

(3) Tolerance - None. Little tendency to - the dose.

v. Amphetamine type

Characteristics:

(1) Psychic dependence - variable.

(2) Physical dependence - None; consequently, no characteristic abstinence syndrome, though withdrawal will be followed by a state of mental and physical depression as the individual escapes from the persistent stimulation.

(3) Tolerance - Slow development of a considerable degree of tolerance to many effects, but not participated in equally by all components of the cerebral system, so that nervousness and sleeplessness persist and psychotoxic effects such as hallucinations and delusions may occur.

vi. Khat type

Characteristics:

(1) Psychic dependence - Moderate but often persistent as long as its maintenance is practicable.

(2) Physical dependence - None.

(3) Tolerance - None

vii. Hallucinogen (LSD) type

Characteristics:

(1) Psychic dependence - varies greatly, but it usually is not intense. Users may wish to repeat them, but if such agents are not readily available, they will either do without them or substitute another substance. A minority of users may develop very strong psychic dependence.

(2) Physical dependence - None.
(3) Withdrawal symptoms - None.
(4) Tolerance - A high degree of tolerance to (i) LSD and (ii) psilocybin develops rapidly and disappears rapidly. Tolerance to (iii) mescaline develops more slowly. Persons who are tolerant to any of these three drugs are cross-tolerant to the other two.

5. Drug abuse

The term "**drug abuse**" is not mentioned in DSM-IV-TR at all. However it loosely refers to the self administration of any drug in a manner that deviates from the approved medical or social pattern within a given culture. It is a tradition within some cultures to use psychoactive drugs [eg hallucinogenic mushrooms, Catha edulis, Bhang on Holi and Kasoomba [**ch 40**] on cultural festivities in India]. Such use would not be called drug abuse.

Salient features:

Drug abuse may not necessarily involve any dependency [physiological or psychological]. For example occasional experimental use of LSD by a college student perhaps on his colleague's insistence is drug abuse, but not drug dependence.

6. Physical dependence (physiological dependence)

Alteration in neural systems which is manifested by tolerance and appearance of withdrawal phenomena, when a chronically administered drug is discontinued or displaced from its receptor.

7. Substance abuse

Substance abuse refers to a maladaptive pattern of substance use that results in recurrent and significant adverse consequences related to the repeated use of substances [**DSM-IV-TR**]. While substance abuse is an official term used in **DSM-IV-TR**, in day-to-day communication, the term ***drug abuse*** is used synonymously.

Salient features:

(1) **Characteristics** - There are (i) repeated failure to fulfill major role obligations [i.e. role of a good parent, son etc], (ii) repeated use in situations in which it is physically hazardous [e.g. during car driving], (iii) multiple legal

problems, and (iv) recurrent social and interpersonal problems.

(2) **Difference from substance dependence** - Unlike the criteria for substance dependence, the criteria for substance abuse do not include tolerance, withdrawal, or craving. Instead it includes only the harmful consequences of repeated use.

(3) **Stage** - Substance abuse is the initial stage in an individual who has only recently started taking the substance. Substance dependence is the later stage.

(4) **Difference between drug abuse and substance abuse** – These are synonymous terms. Till 1960s and 70s WHO favored the former term. Substance abuse is an official term mentioned in DSM-IV-TR. This term now appears in several WHO publications also.

(5) **Causes of substance (drug) abuse** – (i) User starts taking drug for **legitimate medical** use (e.g. morphine for pain), and then starts abusing drug. This group is more easily cured than the second (ii) User takes drug for **non-legitimate use** from the very beginning. Drug is taken for (a) relaxation and recreation (b) better performance in sports (c) relieving tension, frustration arising from loss and failures in life (d) spiritual purposes – for getting in touch with God closely. Such drugs are called **entheogens** [Gk en, in; theo, God; gen, generates] e.g. Iboga, Peyote (e) Aphrodisiac (f) Curiosity.

8. Substance dependence

Substance dependence refers to repeated self-administration that results in (1) tolerance (2) withdrawal symptoms when use is reduced or stopped and (3) compulsive use of drugs (***craving***) despite significant substance-related problems [**DSM-IV-TR**]. Substance Dependence does not occur with caffeine. Both Substance abuse and substance dependence are grouped under "**Substance Use Disorders**" by **DSM-IV-TR**.

9. Substance intoxication

Clinically significant maladaptive behavioral or psychological changes associated with excessive intake of substance (e.g. belligerence, mood lability, cognitive impairment, impaired judgment, impaired social or occupational functioning). Substance Intoxication is often associated with substance abuse or dependence. Does not occur with nicotine. Evidence for recent intake of the substance can be obtained from the history, physical examination (e.g., smell of

alcohol on the breath), or toxicological analysis of body fluids (e.g., urine or blood).

10. Tolerance

Need for greatly -ed amounts of the substance to achieve intoxication (or the desired effect) or a markedly diminished effect with continued use of the same amount of the substance.

Salient features:

(1) **Mechanisms** - (i) Adaptation of the cells of the CNS and (ii) -ed peripheral metabolism of the drug (iii) Immune-like mechanism.

(2) **Loss of tolerance** – (i) During hospitalization or imprisonment, when the addict is weaned off from drug, there may be loss of tolerance (ii) When he is released, he goes back to his old environment"takes same amount of drug as before"Tolerance has been lost"death.

11. Cross-tolerance

Decrease in response to one drug due to exposure to another drug [e.g. exposure to amphetamine results in tolerance to methamphetamine; cigarette smokers show tolerance to caffeine; exposure to psilocybin results in tolerance to Mescaline and DMT].

B. Epidemiology

(1) Worldwide 5 % of the population (about 200 million people) consume illicit drugs annually.

(2) **Most commonly abused substances** -

(i) Legal - In order of frequency are (a) Caffeine (b) Nicotine (c) Alcohol. Adverse effects with caffeine and nicotine are not significant enough to generally include them in the category of substance abuse.

(ii) Illegal – (a) Cannabis. (b) cocaine.

C. Symptoms [of Drug Dependence]

(1) Symptoms of drug dependence would depend upon the drug being abused. Some general symptoms **while the person is using the drug** are: Anxiety Blurring of vision Clumsy movements Depersonalization and emotional detachment Depression Impaired memory and concentration Lethargy and

passivity Loss of appetite and weight Loss of interest in surroundings Mood changes Preference for solitude Reddening and puffiness of eyes Sleeplessness Slurred speech Sweating Temper tantrums Tremors and Unsteady gait. Drug paraphernalia are discovered at home. The addict commits theft and other crimes to procure money to buy drugs.

(2) If the drug is suddenly withdrawn, the person develops **withdrawal symptoms**.

(3) **Combination of drugs** – each drug may potentiate each other. The total effect of a combination may be more than the algebraic sum of effects of each drug separately [eg alcohol+benzodiazepines].

D. Withdrawal Symptoms

Withdrawal symptoms (Abstinence syndrome) refer to symptoms with physiological and cognitive components, that occur when blood or tissue concentrations of a substance decline in an individual who had earlier maintained high levels following prolonged heavy use of the substance.

Salient features:

(1) **Repeated administration becomes necessary** - After developing unpleasant withdrawal symptoms, the person is likely to take the substance to relieve or to avoid those symptoms, typically using the substance throughout the day beginning soon after awakening.

(2) **Variation** - Withdrawal symptoms, which are generally the opposite of the acute effects of the substance, vary greatly across the classes of substances.

(3) **Substances showing marked withdrawal symptoms** - (i) alcohol (ii) anxiolytics (iii) hypnotics (iv) opioids and (v) sedatives

(4) **Substances showing weak withdrawal symptoms** - (i) amphetamines (ii) cannabis (iii) cocaine (iv) nicotine.

(5) **Substances showing no withdrawal symptoms** - (i) Hallucinogens (ii) phencyclidine (however withdrawal to phencyclidine has been demonstrated in animals).

(6) **Typical withdrawal symptoms** [of opiates] – Time period - Begin within 6-8 h of stoppage of drug. May last up to a week. Intensity of symptoms depend upon dose and type of drug that was being used, duration of addiction and suddenness of the withdrawal of drug. Stages - 1st Stage [early; up to 12 h] - Intense fear Chilliness Cold sensation Craving -ed sensitivity to pain

Lacrimation Muscle aches Rhinorrhea Sweating Tachycardia Uneasiness, restlessness, irritability and Yawning 2nd Stage [intermediate; 12-24 h] - -ed Bronchial secretions"predispose to chest infections Dilated pupils Piloerection [“flesh creeping“ or “Goose skin“] Respirations labored and rapid Spontaneous orgasms Tremors. Symptoms of 1st and 2nd stages together are referred to as “**cold turkey”** Such skin is called “**turkey skin**” since it resembles the plucked bird. 3rd Stage [late; 24 h – 10 days] – sleep [lasts 8-16 h] Upon awakening all previous symptoms become intense. In addition there are Cramps and Pain in abdomen and legs Fever Hypertension Perspiration Tachypnea and Vomiting and diarrhea. Sudden withdrawal in opioid dependent pregnant females may be life threatening to the fetus. **All symptoms are maximal in 72 h**. Anorexia is present in all stages. Symptoms pass off in 7-10 d. Newborns of addicted mothers - Also show withdrawal symptoms, which appear within 1 h of birth and require treatment Symptoms are convulsions hyperactivity and twitchings.

E. Complications [of Drug Dependence]

Mainly due to IV use. Same as that of heroin. [Please see **ch 40** – CNS depressants].

F. Management [of Drug Dependence]

1. Treatment

(1) Admission to an institution
(2) Constant supervision – so that he can not secretly get drugs
(3) Detoxification – Gradual reduction of drug
(4) Administration of sedatives.
(5) **Ibogaine** – An indole alkaloid obtained from a West African plant Tabernanthe iboga. Used in the management of addiction to alcohol, cocaine, heroin, methadone and methamphetamine.
(6) Occupational therapy – Engaging in meaningful occupation
(7) Psychotherapy – family, group or individual therapy
(8) General health improvement
(9) Symptomatic.

2. Drug rehabilitation

Drug rehabilitation [***syn*** - drug rehab, rehab) is the process of medical ***and*** psychotherapeutic treatment for drug dependency enabling the patient to cease substance abuse.

Salient features:

(1) **Aims** -Social rehabilitation and training for gainful employment.

(2) Uses psychosocial approaches to rehabilitate drug abusers.

(3) Requires strong family and social support

(4) Persons with history of childhood abuse are more likely to leave drug rehabilitation program midway.

II. PSYCHOACTIVE DRUGS

[A] ***Psychoactive drugs, psychotropic drugs*** or ***psychopharmaceuticals*** are drugs that act primarily upon the CNS, resulting in changes in perception, mood, consciousness, cognition, and behavior.

Salient features:

(1) **Legal status** - Many of these drugs are used recreationally, and are outlawed.

(1) They are the common drugs of abuse or addiction.

(3) They are commonly referred to as street drugs

(4) **Additives** – These are substances added to psychoactive drugs. Three main additives are present— contaminants, diluents, and adulterants. [**ch 40**""“Additives”]. Common adulterants for drugs of addiction (mainly heroin) are (i) Baking soda (ii) Lactose(iii) Magnesium silicate (iv) Mannitol (v) Quinine (vi) Sucrose (vii) Talcum powder. Since heroin is white in color, an adulterant must be white. Charcoal cannot be used as an adulterant of heroin.

(5) **Entheogen** (Gk en, in; theo, God; gen generates"A drug which generates God within) is a psychoactive substance used for religious or spiritual purposes. Mostly derived from plant sources (ex. kykeon, ambrosia, iboga, peyote, ayahuasca).

[B] **Classification of psychoactive drugs [drugs of abuse, drugs of addiction]**

(A) CNS Depressants (.CNS activity) – (1) Alcohol [Ethanol] (2) GHB (gamma-hydroxybutyrate) (3) Hydrocarbons (4) Inhalants (Solvents, volatile

substances) – e.g. Gasoline, Glue (5) Ketamine (6) Opioids [morphine, codeine, pentazocine (Talwin), oxycodone (Oxycontin), hydromorphone (Dilaudid), heroin, fentanyl, methadone (7) Sedatives/Hypnotics/tranquillizers/Anti-anxiety medications (i) Benzodiazepines [diazepam (Valium), Lorazepam (Ativan), oxazepam (Serax), clonazepam (Rivotril), alprazolam (Xanax"), temazepam (Restoril")] (ii) Barbiturates [phenobarbital, butalbital] (8) Snake [cobra] venom [**ch 38**]. **(B) CNS Stimulants** (-CNS activity)– (1) Amphetamines [methylphenidate (Ritalin), dextroamphetamine (Dexedrine), mixed amphetamines (Adderall), methamphetamine (crystal meth) (2) Caffeine (3) Cocaine (Crack) (4) Decongestants (ephedrine, pseudoephedrine, norephedrine or phenylpropanolamine) (5) Ecstasy [syn. 3, 4 - **M**ethylene**d**ioxy **m**eth**a**mphetamine or MDMA] It is a designer drug [please see below]. It also has hallucinogenic actions. (6) Ephedra (including Guarna, Ma huang) (7) Khat (Catha edulis) (8) MDA – 3, 4- **m**ethylene**d**ioxy**a**mphetamine (9) Nicotine. **(C) Psychedelics** or **Hallucinogens** (distort CNS activity) – (1) Alkyl nitrites (2) Anabolic steroids (3) Cannabis (Marijuana, Pot, Hash)[also has CNS depressant activity] (4) Datura (5) DOM [STP] (6) Hallucinogenic Mushrooms (Psilocybin) (7) LSD [d-lysergic acid diethylamide] (8) Mescaline (9) Nutmeg [Myristica fragrans] (10) Phencyclidine (PCP) (11) Salvia divinorum [Diviner's Sage, Seer's sage]. **(D) Other Psychotherapeutic Agents (Medications) -** (1) Agents for treating Alcohol Dependence [disulfiram (Antabuse), Naltrexone (Revia), Acamprosate [Campral] (2) Antidepressants (3) Antipsychotics (4) Anxiolytics [Buspirone (Buspar), Benzodiazepines (5) Bronchodilators (6) Mood Stabilizers (7) Muscle relaxants [Carisoprodol (Carisoma, Sanoma, Soma)] (8) Opioid Substitution Therapy [methadone, buprenorphine/naloxone]. Anticonvulsants and antiparkinsonian drugs are not used as drugs of addiction, although they are psychotherapeutic agents]. **(E) Designer drugs** - Drugs created to get around existing drug laws, usually by modifying the molecular structures of existing legal drugs to varying degrees or less commonly by synthesis of a new drug with an entirely different chemical structure.' **Ex** – MDMA, Cathinones. **(F) Combination drugs** - Psychoactive drugs used in combination. **Ex** – speedball [Please see ch 40 – CNS depressants, under the entry "Heroin"], Tuinal [combination of amobarbital and secobarbital]. Both designer and combination drugs are not additional classes of drugs, but combinations or new forms of first four classes.

A. CNS Depressants

1. Inhalant abuse

Inhalant abuse [***glue sniffing***, ***solvent abuse, volatile substance abuse***, ***volatile substance misuse*** (***VSM***)] refers to inhalation of volatile substances (***inhalants***) which produce euphoriant effects.

Salient features:

(1) **Commonly abused inhalants**– [A]Hydrocarbons usually available as household products - (i) acetone (ii) alkyl nitrite and other nitrites (iii) benzene [in glues, paints, and fuels] (iv) butane (v) butyl nitrite (vi) carbon tetrachloride (vii) fluorocarbons (viii) gasoline or petrol (ix) isopropane (x) kerosene [**ch 40**] (xi) methylene and ethylene chloride (xii) propane (xiii) **toluene** [most studied and most commonly misused volatile solvent globally. Present in paint thinners, varnishes, inks, fuels, and adhesives] (xiv) TCE or 1,1,1-trichloroethane [in fabric protectors and cleaners] (xv) trichloroethylene and (xvi) xylene (in permanent markers and laboratory reagents)]. [B]Anesthetic gases – (i) Chloroform (ii) ether (iii) halothane (iv) nitrous oxide

(2) **Unique risk groups** – (i) Air conditioning repairers (ii) anesthesiologists (iii) dentists (iv) dry-cleaning workers (v) hair stylists (vi) nurses (vii) painters and (viii) shoemakers because of access to these chemicals or anesthetics in the workplace.

(3) **Inhalation methods** – Mainly **5** methods (i) Bagging **–** Volatiles are poured in a plastic bag, which is then closely applied to nose and mouth and deep inhalations taken [Both oral and nasal inhalation] (ii) Dusting - Spraying an inhalant directly in mouth or nose (iii) Glading - Inhaling air freshener aerosols by first spraying them in the room and then inspiring deeply (iv) Huffing – Cloth is saturated with the volatile and kept over mouth (oral inhalation) (v) Sniffing [syn, snorting]– inhalation directly from the neck of a bottle or container (nasal inhalation). Jerry cans or petro-fillers may be used. Occasionally, the chemical product is heated [eg glue] to - volatilization, bubbled through water to remove irritants [eg aluminum chlorohydrate in antiperspirants], or combined with ethanol prior to inhalation.

a. Epidemiology

Most abusers are male teenagers.

b. Signs and symptoms

i. Acute poisoning

Symptoms start within seconds, with the "intoxicating" effects lasting from 15 to 60 min. Effects are dose-dependent.

(1) **Drunkenness** – The effects vary from a state resembling alcoholic intoxication, and distortion of perception to actual hallucinations. Symptoms begin with disinhibition and progress to euphoria, excitement, a sensation of "floating", dizziness, slurred speech, ataxia and a feeling of heightened power.

(2) **Detachment from reality** – Heightened sensations, powerful dreams

(3) **Behavior** –

(i) Complete irrational behavior

(ii) Commission of antisocial acts

(iii) Complete amnesia for behaviors

(iv) Injuring self or even commission of suicide

(4) **Physical withdrawal syndrome** – not seen.

ii. Chronic poisoning

(1) **Solvent syndrome** –

(i) Depression, fatigue, headache, insomnia, intolerance to alcohol, irritability, memory lapses, restlessness, vague and confused thoughts, violent temper.

(ii) When seen in painters (due to chronic exposure to paint thinners etc), also known as **painter's syndrome**.

(iii) Inhalant abuse during pregnancy may cause maldevelopment of fetal brain [**fetal solvent syndrome**, **inhalant embryopathy**], which is characterized by mild brain atrophy and behavioral, cognitive, and emotional abnormalities.

(2) **Degreaser's flush** - chronic exposure to trichloroethylene [for at least 3 wks] followed by intake of alcohol can cause vivid red blotches on face. Called degreaser's flush, because industrial workers who work with trichloroethylene as degreasers [they remove grease used as a protective coating on machinery and steel cables] suffer from it when they drink beer after work. So unsightly

has been this skin redness that the workmen shunned taverns to avoid derision. Occurs due to alcohol apparently inhibiting the normal metabolism of TCE, transiently producing an above normal blood TCE concentration, which in turn causes vasodilation of superficial skin vessels.

(3) **Solvent psychosis** - Dementia like conditions. May form a part of solvent syndrome.

c. Cause of death

i. Acute intoxication

(1) **Sudden cardiac arrest** –

(i) Major COD

(ii) Commonly follows arrhythmias [eg ventricular fibrillation], which in turn is precipitated by any sudden "fight, flight or fright" stimulus

(iii) Such stimulus can precipitate arrhythmic attacks, even considerable time after sniffing has ceased.

(2) **Anoxia, hypoxia and hypercapnia** –

(i) From persistent rebreathing solvents, to the exclusion of oxygen.

(ii) Accidental asphyxia - (a) plastic bag asphyxia] [a type of environmental suffocation; **ch 19**] (b) accidental smothering.

(3) **Aspiration** – fluids, vomit etc

(4) **Loss of tolerance** [please see under "tolerance" above].

(5) **Accidents** –

(i) Drowning

(ii) Falls - from balcony, windows etc

(iii) Burns and explosions - lighting a cigarette after inhaling a volatile substance in a closed space like a car.

(6) **Violence** - during street fights.

(7) **Other** - (i) allergic reactions (ii) CNS depression (iii) severe injury to the lungs.

ii. Chronic intoxication

(1) Liver tumors and liver failure

(2) Renal failure.

d. PM appearances

(1) **Nose and mouth** – surrounding skin may be reddened or excoriated from the irritant action of the solvent.

(2) **Bone marrow, CNS, Kidneys, Liver** – cellular damage.

e. Preservation of samples

(1) **Organs, viscera** – (i) Blood (ii) Brain (iii) Fat (iv) lungs (v) nasal swabs [**ch 5**"details of techniques, especially of blood and lungs].

(2) **Clothings** – may reveal inhalants, if accidentally spilled over them.

2. Sedatives/Hypnotics/Tranquillizers

a. Benzodiazepines

Benzodiazepines (BZD) are psychoactive drugs whose core chemical structure is the fusion of a benzene ring and a diazepine ring.

Salient features:

(1) **History** - The first benzodiazepine, **chlordiazepoxide** (Librium), was discovered accidentally by Leo Sternbach in **1955**, and made available in **1960** by Hoffmann–La Roche. It also marketed diazepam (Valium) since **1963**.

(2) **Uses** - (i) Antianxiety drugs, (ii) muscle relaxants. (iii) sedatives.

(3) **Common preparations** are (i) Alprazolam [Xanax] (ii) Chlordiazepoxide [Librium] (iii) Diazepam [Valium] (iv) Flurazepam [Dalmadorm] (v) Lorazepam [Ativan] (vi) Nitrazepam [Mogadon] (vii) Oxazepam [Seresta] (viii) Quazepam [Doral] (ix) Triazolam [Halcion]

(4) Potentially addictive drugs if used for long time. Excretion in urine may continue for several days.

i. Mechanism of action

Enhances the inhibitory actions of the neurotransmitter GABA located in the brain.

ii. Signs and symptoms

(1) **Acute poisoning** – Symptoms appear within 1 h. (i) Ataxia (ii) Breathing is shallow (iii) Diplopia (iv) Dysarthria (v) Nystagmus (vi) Sedation (vii) Slurred speech (viii) Somnolence (ix) Staggering walk and (x) Vertigo. (xi) Coma

occurs in final stages.

(2) **Chronic poisoning** – Long term administration with high doses [eg 30-40 mg diazepam daily] produces chronic poisoning. Withdrawal symptoms occur on sudden withdrawal. Withdrawal symptoms are (i) ***CNS*** – (a) Anxiety (b) Convulsions [rare] (c) Headache (d) Insomnia (e) Muscle spasms (f) Psychiatric disturbances (g) Tremors (ii) ***GIT*** – (a) anorexia (b) vomiting (iii) ***Resp Sys*** - respiratory depression [rarely].

iii. Management

(1) **Decontamination** -

(i) Gastric lavage. Use cuffed endotracheal tube if comatosed

(ii) Emesis – not recommended because of potential for CNS depression

(iii) Activated charcoal.

(2) **Airway and breathing** - (i) Establish clear airway (ii) Assisted ventilation (iii) oxygen.

(3) **IV Fluids**.

(4) **Antidote** – Flumazenil

(i) Dose - (a) 0.2 mg/min, slow IV to a total dose 3 mg. **1.5 mg** of Flumazenil leads to 55% receptor occupancy; 10 times [15 mg] is required to 100% blockade [as revealed by PET]. (b) Extravasation should be avoided because of the risk of local irritation. (c) Resedation may occur at 20–120 minutes, and readministration of flumazenil may be necessary [repeated doses are required because duration of action of flumazenil is shorter than that of most benzodiazepines. Vigilance is also required].

(ii) Also useful in Zolpidem and in select cases of ethyl alcohol [**ch 40**]

(iii) Mechanism of action - Competitive benzodiazepine receptor antagonism on the GABA-A receptor complex. Blocks CNS actions of BZDs. But does not reliably reverse respiratory depression induced by IV BZDs.

(iv) Ideal for patients who are new to benzodiazepines and who overdose solely on a benzodiazepine.

(v) Induces benzodiazepine withdrawal symptoms [eg seizures in benzodiazepine dependent patients].

(5) Correction of hypotension [**ch 32**].

(6) Forced diuresis and hemodialysis are ineffective.

iv. Fatal dose

100-300 mg/kg [20g for a 70 kg man]. 4000 tablets of 5 mg. Thus death is extremely rare if taken alone. May cause death if taken with alcohol or other potentiating drugs.

v. MLI

Mortality rates – Among benzodiazepine users are 2 fold -er. Reasons are same as those of -er mortality rates among drug abusers [please see below under MLI of drug abuse].

B. CNS Stimulants

1. Amphetamine

Amphetamine is a psychostimulant drug that produces increased wakefulness in association with decreased fatigue and appetite.

a. Derivatives

The title "amphetamines" includes amphetamine itself and its derivatives (1) Dextroamphetamine, (2) Fenfluramine, (3) MDA. (4) MDEA - 3, 4-methylene-dioxyethylamphetamine (5) MDMA (Ecstasy) (6) Mephentermine, (7) Methamphetamine [methylamp-hetamine] (8) Methylphenidate, (9) Phentermine, (10) Synthetic amphetamines.

b. Mechanism of action

Amphetamine -s the synaptic conc of dopamine, serotonin, and norepinephrine in the brain. Dopamine is involved in various **reward pathways of the brain** causing euphoria.

c. Signs and symptoms

i. Acute poisoning

(1) **GIT** - Nausea and vomiting

(2) **CVS** - (i) Aortic dissection (ii) Dysrhythmias (iii) Hypertension (iv) Myocardial ischemia (v) Tachycardia (vi) Vasospasm

(3) **CNS** - (i) Agitation and restlessness (ii) Anorexia (iii) Bruxism (iv)

Choreoathetoid movements (v) Confusion (vi) Euphoria (vii) Headache (viii) Hyperactivity (ix) Hyperreflexia (x) Hyperthermia (xi) insomnia (xii) Intracerebral hemorrhage (xiii) Paranoid psychosis (xiv) Seizures " Rhabdomyolysis (xv) talkativeness

(4) **Other sympathetic symptoms** - (i) Diaphoresis (ii) Mydriasis (iii) Tachypnea (iv) sweating (v) Tremor

(5) **Other organ systems** - (i) Acute lung injury (ii) Ischemic colitis (iii) Muscle rigidity.

ii. Chronic poisoning

(1) **Amphetamine psychosis** - Characterized by (i) Delusions [usually of persecution] (ii) Hallucinations [usually visual, sometimes tactile] (iii) Paranoid personality (iv) Stereotyped compulsive behavior

(2) **Aortic and mitral regurgitation**

(3) Cardiomyopathy

(4) Permanent damage to dopaminergic and serotonergic neurons

(5) Pulmonary hypertension

(6) Vasculitis.

(7) **Skin** - Grey, dry [due to methamphetamines]. Pruritus, tactile hallucinations similar to cocaine bugs [**meth mites**].

(8) **Meth mouth**–darkly stained and crumbling teeth, akin to baby bottle tooth decay, extreme cavities, rampant caries, xerostomia and lockjaw.

d. Dangers of misuse

(1) **-d risk of accidents** - because of both the excitation produced by these agents and the excessive fatigue which may break through and manifest itself at an inopportune time.

(2) -d risk of suicide

(3) intraventricular hemorrhages [**ch 17**]

(4) Overactivity and aggressive behavior

(5) Paranoid psychosis

(6) **Serious antisocial behaviour** - especially in abuse by IV administration, with its concomitant bizarre mental effects.

(7) Shock and collapse

(8) Toxic Epidermal Necrolysis.

e. Diagnosis

(1) **History** - rarely reliable as patients often do know the exact drug they have used. However the prevalence of amphetamine abuse in the local geographic region should heighten the suspicion of amphetamine toxicity in patients presenting with typical signs and symptoms.

(2) **Laboratory Findings** – Non-specific (i) -CPK (ii) -liver enzymes (iii) Hyperglycemia (iv) hyperkalemia (v) Hyponatremia (vi) Leukocytosis (vii) Myoglobinuria

(3) **Qualitative urine immunoassay testing** [QUIT]- Available, but several pitfalls exist

(4) **ECG** – Findings of ischemia, hyperkalemia.

f. Management

(1) Gastric lavage

(2) Activated charcoal

(3) Continuous cardiac monitoring

(4) Acidification of urine

(5) Chlorpromazine [for amphetamine psychosis]

(6) **Immunotherapy** - Vaccines that would generate antibodies to sequester the drug and prevent its access to the brain. Currently under development for 4 major drugs of abuse – cocaine, methamphetamine, morphine/heroin and nicotine.

(7) **Symptomatic** – mainly treatment of agitation, delirium, hypertension, hyperthermia and seizures [**ch 32**].

g. Fatal dose

Oral Amphetamine - 150-2000 mg.

h. Tests

(1) **Mandelin test** - Mandelin reagent [1% sol^{n} of ammonium vanadate (NH_4VO_3) in conc H_2SO_4] is added to sample. Amphetamine gives a blue-green colour.

(2) **Marquis' test** - please see **ch 40**.

(3) **Simon's test** [modified sodium nitroprusside test] -

(i) **Reagents** (a) Reagent 1 - 1 g of sodium nitroprusside in 100 mL water + 2 mL of acetaldehyde. Mix thoroughly. (b) Reagent 2 - Freshly prepared 2% sodium carbonate in distilled water.

(ii) **Procedure** - Suspect sample + 1 drop of reagent 1 + 2 drops of reagent (2)

(iii) **Interpretation** – (a) Primary amines [e.g. amphetamine, methylenedioxyamphetamine (MDA)]"Pink to cherry–red colour. (b) Secondary amines [e.g. methamphetamine, ephedrine, 3,4–methylenedioxymetamfetamine (MDMA)]"A dark blue colour.

i. PM appearances

(1) Appearances similar to those of asphyxia.

(2) Meth mouth.

(3) Amphetamine can be detected in blood, body fluids and viscera.

j. ML importance

(1) **Drug Facilitated Sexual Assault** – MDMA is one of the drugs used in DFSA [**ch 25**].

(2) **Liquid gold** - Kidney excretes 40% of amphetamine unchanged. Some users ingest amphetamine up to 2000 mg per day. About 800 mg amphetamine would be excreted unchanged in these users. They bottle this urine in vials and sell this yellow fluid in the underworld market as **liquid gold** [reasons for calling it “liquid gold” (i) liquid state (ii) golden yellow (iii) sold at very high cost]. Liquid gold refers to urine containing any psychoactive drug. Classically Eastern Siberians who ingested hallucinogenic mushrooms [fly agaric] would collect their urine; this would be drunk by others while still warm.

2. Ecstasy [MDMA]

Ecstasy [MDMA (3,4-***M***ethylene***D***ioxy***M***eth***A***mphetamine)] is an entactogen of the phenethylamine and amphetamine families, having a tendency to induce euphoria, a sense of intimacy with others, and diminished anxiety and depression. An **Empathogen** or **Entactogen** [Gk en, within); Latin tactus, touch and Gk gen, produce; literally “producing a touching within”] is a drug that produces feelings of empathy, love, and emotional closeness to others.

a. Clinical uses

Post-traumatic stress disorder (PTSD) Anxiety associated with terminal cancer.

b. Signs and symptoms

100 mg orally produces effects within 30 minutes. General signs and symptoms of stimulants are seen hyperactivity [e.g. an uncontrollable urge to dance] Increased alertness, awareness, and wakefulness and Feelings of empathy, compassion, and forgiveness towards others bruxism [grinding to teeth] trismus [spasm of muscles of mastication].

c. Cause of death

Death may be due to Disseminated intravascular coagulation [DIC] – dural sinus thrombosis [DST], particularly in the superior sagittal sinus is an imp PM finding [**ch 5**]. hyperthermia, rhabdomyolysis and renal failure.

d. Tests

Simon's test - Please see under amphetamines.

e. ML importance

(1) **Candyflapping** – Intentional combination of Ecstasy with LSD.

(2) **Stacking** - Combination of Ecstasy with another drug [most typically a depressant, e.g. alcohol]. This - the danger of overdose, and both Ecstasy [stimulant] and alcohol [depressant] mask each other's effect

(3) **Sexstacy** - Combination of Ecstasy with sildenafil [Viagra] to - sexual pleasure.

C. Psychedelics (Hallucinogens)

A ***psychedelic substance*** is a psychoactive drug whose primary action is to ***alter the cognition and perception of the mind***.

1. Agents

a. Alkyl nitrites (Poppers)

(1) **Alkyl nitrites** inhaled for recreational purposes are (i) **amyl nitrite** [also used for treatment of CN poisoning] (ii) **butyl nitrite** (iii) **isopropyl nitrite**

and (iv) **isobutyl nitrite**.

(2) **Uses** - used in common household products eg air freshener, finger nail polish remover and video head cleaners.

(3) **Abuse** -

(i) As club drugs. To get a "high". Cause a rush of warm sensations and dizziness when inhaled (due to sudden vasodilation).

(ii) Heterosexual males - use them as aphrodisiacs, sexual enhancer, for prolonging sexual orgasm

(iii) Homosexual males - to relax anal sphincter.

(4) **Adverse effects** -

(i) Methemoglobinemia

(ii) Neurological damage [heavy long-term use]

(iii) Swallowing or aspirating the liquid rather than inhaling can prove fatal. They have a low risk of harm to society and the individual compared to other recreational drugs.

b. Anabolic steroids

(1) Taken orally or by injection.

(2) Acneiform skin lesions.

(3) May produce sudden mood swings, paranoia, depression (-suicidal tendencies) and aggressive behavior ["**roid rage**"].

c. DOM [STP]

2, 5-Dimethoxy-4-methylamphetamine [DOM; known on the street as STP, standing for "Serenity, Tranquility, and Peace"]. **Signs and symptoms** - perceptual changes eg blurred vision, multiple images, vibration of objects, visual hallucinations, distorted shapes, enhancement of details, slowed passage of time, -ed sexual drive and pleasure, -ed contrasts, pupillary dilation, -ed BP.

d. Hallucinogenic mushrooms

Also known as psychoactive mushrooms [please see **ch 39**].

e. Lysergic acid diethylamide (LSD)

LSD is a colorless, odorless and mildly bitter semisynthetic compound, the lysergic acid portion of which is a natural product of the ergot fungus Claviceps

purpurea.

i. Mechanism of action

(1) **Serotonin receptor agonist** - LSD binds to most serotonin receptor subtypes except for 5-HT_3 and 5-HT_4 [There are 7 subtypes in all – from 5-HT_1 to 5-HT_7].

(2) **Psychedelic effects of LSD** - (i) Attributed to its strong partial **agonist** effects at **5-HT2A** receptors.

ii. Toxicokinetics

(1) Absorbed from GIT. Data about the binding of LSD to human plasma proteins is not available.

(2) **Distribution** – Rapidly distributed to body tissues. Highest conc appears in lungs, liver, bile, kidneys and brain.

(3) Half-life in humans - 175 min.

iii. Mode of administration

(1) Oral – Most common.

(i) Taken after placing on - (a) absorbent blotter paper [“blotter acid”, “postage stamps”]. Most common mode (b) gelatin squares [“window panes”] (c) sugar cube

(ii) in blue pills

(iii) in capsule (iv) as tiny tablets [“microdots”].

(2) IM and IV - In liquid form.

(3) Smoked – very rarely. Heating destroys LSD, so smoking is not much effective.

iv. Dose

(1) Effects appear at: **25 µg**.

(2) Average recreational dose: **100-500 µg** [equal to one-tenth the mass of a grain of sand].

v. Availability

LSD (usually as the tartrate salt) is distributed as tablets, liquid, powder, and in gelatin squares. Recently it has been available as “blotter acid,” which are sheets

of paper soaked with LSD, dried, and perforated into small squares.

vi. Signs and symptoms

(1) Symptoms from LSD [“taking a trip”] are of rapid onset and relatively mild.

(2) **Duration** - A trip usually occurs within half an hour of ingestion, peaks in 2-6 h and fades after 12 h. In the recovery stage there may be apprehension and distraction that is not immediately obvious to onlookers.

(3) **Effects** - depend very much on the individual and the circumstances. Same user may have a good or a bad trip on different occasions and even within the same trip.

(4) **Sequence** - Somatic symptoms appear first, then mood and perceptual changes and finally psychological changes, although effects overlap.

(5) **Symptoms** –

(i) Pupils – Dilated. There may be intense photophobia.

(ii) Muscular - -ed deep tendon motor reflexes and muscle tension, mild motor incoordination, and ataxia.

(iii) -HR, -RR, and -BP General - Nausea, salivation (sometimes dry mouth), sweating, intestinal cramping, .ed appetite.

(iv) Psychological – (a) illusions (b) hallucinations. (c) Perceptual changes occurring in a clear consciousness. These are (I) intensification of perceptions (II) depersonalization [Individuals feel separate from their own personal physicality [A similar state occurs in Locked-in Syndrome (LiS) – ch 8]. They sense
their body sensations, feelings, emotions and behaviors as not belonging to themselves. Life “feels like a movie”. Things seem unreal or hazy] (III) derealization [external world seems strange or unreal]. Victim may become violent or panic stricken. May attack others or hurt himself from disregard of reality (IV) synesthesias [stimulation of one sense leads to experiences in another sense, eg colors are heard, sounds are felt] (V) Feeling of being able to fly – may jump out of windows.

(v) Bad trip – LSD may trigger panic attacks or feelings of extreme anxiety [good trip is when symptoms are pleasurable].

(vi) Return trip – Reappearance of symptoms long after the last dose of LSD was taken.

(vii) The symptoms produced by LSD resemble those produced by mescaline,

psilocybin, and some of the amphetamine analogs. The major difference between LSD, psilocybin, and mescaline is potency. A 1.5 µg/kg dose of LSD = 225 µg/kg of psilocybin = 5000 µg/kg of mescaline. With mescaline onset is slower.

vii. Adverse effects

After effects may persist for days or weeks. These are (1) Chronic dread (2) Depression (3) Mood swings (4) Panic attacks (5) Paranoid states (6) psychosis (7) Schizophrenic episodes. Repeated intake of

LSD causes permanent psychosis. LSD does not cause chromosomal breakage and is not teratogenic.

viii. Tolerance

(1) Tolerance is associated with frequent use of any of the hallucinogens.

(2) **Tolerance to LSD** – develops in 2-3 days with daily intake

(3) **Loss of tolerance** – occurs in 4-6 days if LSD is not taken.

(4) **Cross-tolerance** - between mescaline, psilocybin, and LSD occurs, but not between amphetamine and LSD, despite the chemical similarity of amphetamine and mescaline.

ix. Withdrawal syndrome

(1) **Withdrawal syndrome** - does not occur with LSD.

(2) **Flashback phenomenon** - (i) Spontaneous recurrence of the LSD use experience in a drug free state (ii) Occurs weeks to months after the last experience. (iii) Induced by – alcohol intake, fatigue, marijuana intoxication, severe physical illness and stress. Can sometimes also be self-induced by will for positive reexperiences [**free trips**]. (iv) Occurs with abuse of most psychotomimetics eg (a) LSD (b) Mescaline (c) psilocybin (d) STP (e) Tryptamines (e) May lead to eccentric behavior – Homicide, suicide.

x. Fatal dose

200 µg/kg. Thus **14,000 µg** [or 14mg] for a 70 kg human.

xi. Tests

Van Urk's Test – A Spot Test for LSD, psilocin and psilocybin. **Ehrlich's [or**

Van Urk's] Reagent [1% p-dimethylaminobenzaldehyde in 10% HCl] gives following colors in the presence of following psychedelics. (i) **LSD** – purple (ii) **Psilocin** - blue-grey (iii) **Psilocybin** - red-brown.

xii. Cause of death

Paralysis and respiratory failure.

xiii. Management

Treatment is required if a "bad trip" occurs.

(1) **Anxiolytic agents** - eg diazepam (Valium) 10-20 mg or other benzodiazepines.

(2) **Removal of stimuli** – eg bright lights and loud noises are helpful [same as in strychnine].

(3) **Prolonged talking** – known as "Talking the person down". Continues for 12-18 h.

(4) Psychotherapy.

xiv. MLI

Please see MLI of psychedelics below.

f. Phencyclidine (PCP)

Phencyclidine [PCP, angel dust], is a recreational hallucinogenic drug, formerly used as an anesthetic agent.

Salient features:

(1) First synthesized in **1926**, it was patented in 1952 by the Parke-Davis pharmaceutical company and marketed under the brand name **Sernyl**.

(2) **MOA** - It acts as an NMDA receptor antagonist [blocks the activity of NMDA receptor]. Other NMDA receptor antagonists are dextromethorphan, ketamine, nitrous oxide and tiletamine. Primary psychoactive effects last for a few hours, but total elimination takes more than a week.

(3) **Signs and symptoms** – see box.

Memory Aid 1: Signs and symptoms of PCP poisoning

RED DANES had **BAD COLD: R**age, **E**rythema, **D**ilated pupils, **D**elusions, **A**mnesia, **N**ystagmus, **E**xcitation, **S**kin dryness. **B**izarre behavior, **B**P-, **A**cute psychosis [delusions and hallucinations], **A**gitation, **A**taxia, **D**ysarthria, **C**atatonia, **C**oma, **L**ethargy, violent ten**D**encies +Tachycardia, tremors

(4) **Fatal dose** - (i) ingestion – 100 mg (ii) blood levels – 0.1mg%.

(5) **Management** - (i) Gastric lavage (ii) AC (iii) Immuno-therapy - PCP-specific antigen binding fragments [Fab] therapy has been tried in animals. Shows promise in humans also. [please also see **ch 36**"Arsenic].

2. MLI of psychedelics (Hallucinogens)

(1) The chief dangers to the individual arise from the psychological effects. Impairment of judgment could lead to dangerous decisions or accidents. Occasional persons may become depressed, so that suicide is a possibility in users of these drugs.

(2) Hallucinogens are mainly used for self-exploration, to experience varied hallucinations and to get out of day-to-day boredom.

(3) LSD Has been used in treatment of alcoholism [**ch 40**].

D. Other Psychotherapeutic Agents (Medications)

1. Antidepressants

Antidepressants are used to alleviate mood disorders, e.g. major depression and dysthymia (chronic mood disorder). Major antidepressants are

(1) Monoamine oxidase inhibitors (MAOIs),

(2) Tricyclic antidepressants (TCAs),

(3) Selective serotonin reuptake inhibitors (SSRIs) and

(4) Serotonin-norepinephrine reuptake inhibitors (SNRIs). Amphetamine was used earlier.

a. MAO inhibitors

i. Classification

Following MAOI are important.

(1) **Nonselective MAO-A/MAO-B Inhibitors** -

(i) Hydrazines - (a) Benmoxin [Nerusil] (b) Iproniazid [Iprozid] (c) Isocarboxazid [Marplan] (d) Nialamide [Niamid] (e) Octamoxin [Nimaol] (f) Phenelzine [Nardil] (g) Pheniprazine [Catron] (h) Safrazine [Safra]

(ii) Non-Hydrazines - (a) Caroxazone [Surodil] (b) Tranylcypromine [Parnate]

(2) **Selective MAO-A Inhibitors**-(i) Brofaromine (Consonar)

(3) **Selective MAO-B Inhibitors**-(i) Lazabemide [Pakio].

ii. Mechanism of action

Inhibiting the activity of monoamine oxidase" breakdown of monoamine neurotransmitters prevented "-Monoamines [serotonin, noradrenaline and dopamine].

iii. Signs and symptoms

Mortality occurs from acute ingestions of 200 mg tranylcypromine (typical daily dose 20-30 mg) and 400 mg phenelzine (typical daily dose 60-90 mg). Effects appear within 24 hours.

(1) **Symptoms** - Sympathetic hyperactivity, anxiety, agitation, delirium, diaphoresis, flushing, hallucinations, headache, hyperthermia, hypertonia, Irritability, seizures, trismus

(2) **Clinical findings** – Dysrhythmias, DIC, multiorgan failure, mydriasis, neuromuscular irritability, nystagmus, opsoclonus [alternating “ping-pong” gaze], tachycardia, tachypnea, Severe hypertension followed by hypotension and cardiovascular collapse.

iv. Fatal dose

2-5mg/kg.

v. Treatment

Gastric lavage, symptomatic.

2. Antipsychotics [Neuroleptics]

Antipsychotics were earlier called **neuroleptics**.

a. Classification

(1) **Phenothiazines** -

(i) Aliphatic side chain – Chlorpromazine, Triflupromazine

(ii) Piperidine side chain – Mesoridazine, Thioridazine

(iii) Piperazine side chain – Fluphenazine, Perphenazine, Prochlorperazine, Trifluoperazine.

(2) **Butyrophenones** -

(i) Haloperidol
(ii) Penfluridol [Exceptionally long acting]
(iii) Trifluperidol.
(3) **Thioxanthenes** -
(i) Chlorprothixene (Cloxan, Taractan, Truxal).
(ii) Clopenthixol (Sordinol).
(iii) Flupenthixol or Flupentixol (Depixol, Fluanxol) – "Cis" form is effective, but not "trans" form, because that does not block DA
(iv) Thiothixene (Navane)
(v) Zuclopenthixol (Cisordinol, Clopixol, Acuphase).
(4) **Other heterocyclics** -
(i) Loxapine
(ii) Pimozide.
(5) **Atypical neuroleptics** (second generation antipsychotics)-
(i) Benzepines - (a) Clozapine [Clozaril] (b) Loxapine (c) Olanzapine [Zyprexa] (d) Quetiapine [Seroquel]
(ii) Indoles - (a) Risperidone [Risperdal] (b) Sertindole (c) Ziprasidone (Geodon, Zeldox).
(iii) Quinolinones - (a) Aripiprazole [Abilify]: Partial agonism at the D-2 receptor.

b. Mechanism of action

All antipsychotics (except atypical) have potent dopamine D-2 receptor blocking action; antipsychotic potency has shown good correlation with their capacity to bind to D2 receptor. Blockade of dopaminergic projections to the temporal and prefrontal areas constituting the 'limbic system' and in mesocortical areas is responsible for the antipsychotic action.

c. Signs and symptoms [of toxicity]

(1) **General**-.ed sweating, hypothermia or hyperthermia.
(2) **Eyes** - corneal opacities, miosis.
(3) **CNS** - sedation, seizures.
(4) **CVS** - Myocardial depression, orthostatic hypotension, ventricular tachycardia.
(5) **RS** - laryngospasm.

(6) **GIT** - .ed intestinal motility and secretions, .ed salivation, cholestatic jaundice.
(7) **Blood** - agranulocytosis, hemolytic anemia.
(8) **Skin** - dermatitis, gray-blue pigmentation, photosensitivity, urticaria.
(9) **Male specific** - gynecomastia, priapism.
(10) **Female specific** - amenorrhea.
(11) **Movement disorders** - Occur within 60 days of initiating therapy. (i) Acute dystonia - oculogyric crisis, spasms of jaw, tongue, lip and throat, neck twisting, facial grimacing, Rabbit syndrome – Peri-oral tremor, abdominal wall spasms, opisthotonos. Symptoms rapidly resolve with parenteral antihistamines, anticholinergics or benzodiazepines. (ii) Akathisia - Restlessness and inability to sit, feeling of discomfort, apparent agitation [compelling desire to move about, but without anxiety]. May be mistaken for exacerbation of psychosis. Mechanism not known. No antidote. Central anticholinergics only reduce intensity in some cases. (iii) Parkinsonism - Bradykinesia [slowness of movement], fine movements, mask like expression, muscle weakness, pill rolling, resting tremor, rigidity and shuffling gait.

d. Fatal dose

2-5 g.

e. Management

(1) Emesis and gastric lavage
(2) Multiple Dose Activated Charcoal (MDAC)
(3) Catharsis
(4) Symptomatic.

III. BODY PACKER AND BODY STUFFER SYNDROMES

The ***Body packer syndrome*** is accidental and unintentional poisoning with illicit drugs in persons, who attempt to transport drugs illegally across national borders by packing them in plastic packets, ingesting them or inserting them in body orifices, and then subsequently retrieving them in a foreign country, thus safely bypassing custom authorities.

Salient features:

(1) **Smuggling procedure** – (i) Methods of transportation - (a) Ingestion - Typically the persons who smuggle drugs (variously known as couriers, internal carriers, mules and swallowers) pack drugs in small plastic packets and ingest them. Usually about 100 packets are ingested, each packet containing about 10 g of drug. The total smuggled drug is 1 kg. Occasionally persons carrying more than 200 packets have been described. (b) Body cavities and orifices - Occasionally the drug is inserted in ears [**ear packing**], anus, vagina, under the foreskin of uncircumcised males or strapped under the breasts. (c) Unusual methods - Rarely A drug packet may be tied to a thread and swallowed, the other end tied to a tooth for easy retrieval at destination. (ii) Composition of packets - Instead of plastic packets the drug may be packed in balloons, capsules, condoms, fingers of rubber gloves, heat sealed plastic films or multiple layers of latex. (iii) Measures to reduce gut motility – Antidiarrhoeals, antipropulsives [eg diphenoxylate, loperamide] and antiemetics during drug transportation. (iv) Evacuation – When the body packer reaches the country of destination, he takes a laxative, defecates, retrieves the packets from feces and delivers them to the drug dealer ("pusher"). Enema and suppositories may also be used.

(2) **How the smuggler comes to notice** – (i) Bursting or leaking of packets - If one or more packets begin to leak or rupture while the smuggler is still at airport or in the flight, he suffers poisoning symptoms and comes to the notice of law. Sometimes death also occurs. (ii) Osmotic seepage – Even if packets do not rupture, osmotic seepage occurs across latex wrappings, causing traces of drug to appear in circulation and urine. Suspicious persons may be retained for urine examination and X-ray.

(3) **Terminology**: (i) Body packers - are those, who smuggle illicit drugs across nations by either ingesting them or inserting them in body cavities. (ii) Body stuffers - do not smuggle illicit drugs. They peddle drugs locally. When police comes to arrest them, they quickly ingest drugs available with them to eliminate potential incriminating evidence. Practice is also used to hide drugs by prisoners from prison officials during sudden checking and concealing drug from other addicts.

A. Investigation

1. Physical examination

Gentle rectal and vaginal examination may disclose packets.

2. Stomach lavage

to recover packets [**ch 32**].

3. Radiography

a. X-ray

(1) Unusual number of rounded cigar-shaped or oblong masses with a complete gas halo

(2) **Radiodensities of different drugs of abuse** -

(i) Hashish is denser than stool;

(ii) Cocaine appears similar to stool; and

(iii) Heroin has a gaseous transparency.

(3) **Tic Tac sign** – [Tic tac is a brand of small, hard oblong shaped mints]. Multiple oblong, uniformly shaped packages (of the shape of tic tac) are seen in the abdomen.

(4) **Double-condom sign** - Well-defined, homogeneous oval or oblong densities surrounded by a translucent rim (which represents air trapped between the individual layers of packaging material – most often two condoms). If these densities are multiple, and found in the proximal ascending colon or upper GIT, the diagnosis is almost assured.

(5) **Rosette sign** - Densities associated with a translucent rosette at one end. Indicates air trapped in the knot where the condom is tied.

(6) **Parallelism** - Relatively rigid packages align parallel to each other in the bowel lumen.

b. CT scan

c. Barium contrast studies

d. Ultrasonography and MR imaging

Do not have an important role.

4. Lab tests

Blood and urine tests for illicit drugs.

B. Management

(1) **Nil orally** – till packets are within GIT.

(2) **Removal of packets** -

(i) Whole bowel irrigation – to remove packets [**ch 32**]. Some recommend polyethylene glycol as an irrigation solution, but others do not approve of it because it can dissolve packages causing their rupture. Following alternatives must also be kept in mind

(ii) Metoclopramide – 10 mg, 8 hourly, to promote gastric emptying

(iii) Rule out bowel obstruction; wait and allow packets to pass in colon"high volume saline enemas, or low volume phosphosoda enemas

(iv) Bisacodyl suppository – to empty rectum

(v) Endoscopy or surgery – If all measures fail, or if there is bowel obstruction.

(3) **Symptomatic** - Appropriate to the drug which has leaked and caused poisoning.

C. PM Findings

(1) Presence of drug packets in stomach and intestines, some of which may be ruptured

(2) Congestion of gastric and intestinal mucosa

(3) Congestion of lung, meninges and abdominal organs Edema of lung and brain.

D. Medicolegal Aspects

Body packing increased after 9/11, because of the increased border security, which made conventional smuggling more difficult.

IV. INVESTIGATION OF DRUG ABUSE DEATHS

A. History

Bizarre acts, irrational behavior or unexpected coma followed by death,

especially in younger people returning from night long parties, points to drug related death.

B. Clothes

Drugs may be hidden in belt, eye glass case, jewellery, matchboxes, money purse, pens, pockets, seams, shoes, socks etc. These must be thoroughly searched.

C. Scene of Death

(1) Follow all protocols of a scene of death investigation [**ch 1**]. Following specific points are important.

(2) **Psychedelic posters** – indicate drug addicts.

(3) Drug may be hidden anywhere in the room – either in obvious places eg cupboards, or cleverly hidden eg between pages of a book, tourniquet etc. Any sample of drug recovered should be preserved and sent for analysis.

(4) **Equipment** [syn, works]– may be found at the scene (i) intravenous apparatus [**Fig 46.1**] used by drug addicts consists of paper for mixing drugs (1), drug (2), needles (3), homemade eyedropper syringe (4), a teaspoon cooker (5), cotton to remove impurities (6), shoelace tourniquet (7) and a source of heat (8). Method of injection [**ch 40**"Heroin]. Half filled syringes or even empty syringes should be preserved and sent for chemical analysis. (ii) Aerosol cans, bags, balloons, plastic tubes etc – indicate death due to inhalant abuse.

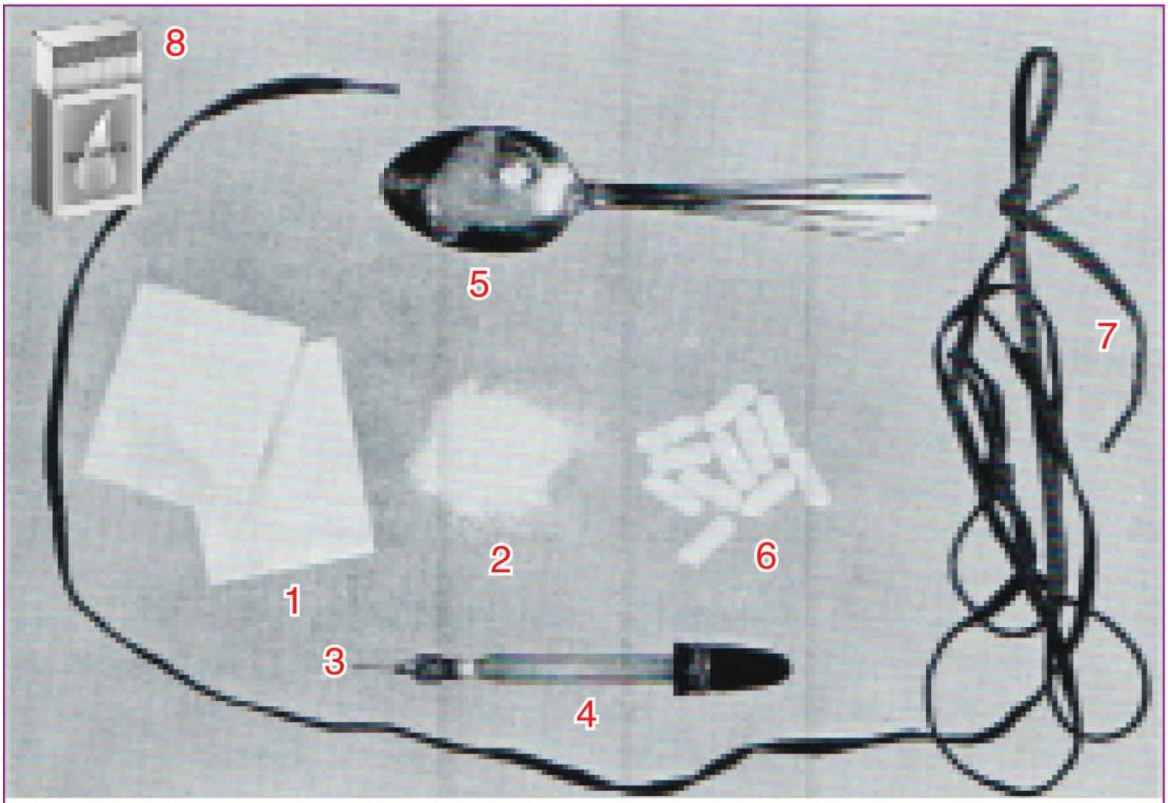


Fig. 46.1: Equipment used by drug addicts, which may be found at the scene of crime. Please correlate nos (given in red). with text.

D. Autopsy

PM findings are same as in heroin [**ch 40**], cocaine [**ch 41**], amphetamines or inhalants [please see above for both] if any of these drugs were used. Because any other illicit drug may be involved, there may be additional, specific findings related to that drug.

1. External

Body – Stains – on tips of fingers. Color and smell indicates possible type of capsule or pill handled.

2. Internal

GIT – (i) may contain pills or capsules (ii) Stomach – (a) dye from capsules – may color mucosa with various colors (b) microscopic examination under polarized light may show particles of optically active filler material [cellulose,

starch, talc] adherent to the gastric mucosa.

E. Toxicology

Drugs of abuse may be detected by modern analytical methods from various body fluids.

V. TOXICOLOGIC RADIOLOGY

Radiology in Drug Abuse

Body packers – drug packets are revealed by x-rays [please see above].

VI. GLOSSARY OF DRUG ABUSE

(1) Acid head – heavy user of LSD.
(2) Bad, bum or freak out trip – LSD experience in which the drug effects are unpleasant and frightening. Lasts 8-12 h.
(3) Bang - To inject drugs.
(4) Bean - Capsules for drugs.
(5) Bombed out - Very much intoxicated by drugs.
(6) Burned out - poor psychological functioning in someone who has taken drugs for too long.
(7) Busted – to be arrested.
(8) Buzz, flash, rush - euphoric reaction to a drug.
(9) Connect - To purchase drugs.
(10) Crash - the end of a drug experience.
(11) Cutting – adulterating a drug with additives.
(12) Flip – to go psychotic.
(13) Flying - Under the influence of drugs.
(14) Guide – A person who “babysits” for the drug user during a drug session.
(15) Hard drug, soft drug, gateway drug – These are arbitrary terms, with no clear criteria or scientific basis. **Hard drugs** are drugs that lead to severe physical addiction. They are generally injectable [heroin, methamphetamine]. **Soft drugs** do not cause physical addiction [cannabis, mescaline, psilocybin and LSD]. **Gateway drug** is a “midway drug” that opens the door to the use of other, harder drugs. Cannabis is sometimes considered a gateway drug.

(16) Hook up, connect, score - make a drug purchase.
(17) Jones; jonesing - experiencing drug withdrawal.
(18) Junkie - heroin addict.
(19) k-hole - under the influence of ketamine, unable to move.
(20) Line - a measured amount of cocaine.
(21) Needle freak - A person who prefers to take drugs with a needle.
(22) Pot – Marijuana.
(23) Roached - under the influence of Rohypnol (roofies).
(24) Rolling - under the influence of MDMA (Ecstasy).
(25) Shooting – IV injection.
(26) Speed freak - Person who repeatedly takes Amphetamines, usually intravenously.
(27) Spaced out - Under the influence of drugs.
(28) Spunion - someone under the influence of MDMA or other club drugs.
(29) Stoned – intoxicated.
(30) Taste - A small sample of drugs.
(31) Tripping - hallucinating, usually associated with LSD or psilocybin.
(32) Tweeking - prolonged drug use, usually meth.
(33) Works, rig - equipment for injecting drugs.
(34) Zombi - Heavy user of drugs.
(35) Zonked - Extremely high on drugs.

VII. ML ASPECTS OF DRUG ABUSE [GENERAL]

For MLI of individual drugs, please see under relevant sections.

(1) **Contaminated currency** - Paper money in almost all parts of the world shows drug contamination

(i) Causes - (a) handling during drug deals (b) use of rolled up notes for snorting drugs (c) Cross-infection - After the initial contamination, the drug is "infected to" other notes in close contact, often stacked together. Money-counting machines are infected which further infect clean notes.

(ii) Smaller notes are more likely to be contaminated because of more frequent use

(iii) May even serve as vectors of disease if associated infective agents are

present.

(2) **Death** – is more often the result of combinations rather than the abuse and overdose of a single agent.

(3) **Driving under influence of drugs** - Drowsiness and accidents may be caused by amphetamine, antihistamines, benzodiazepines, cannabis products, cocaine, hypotensive drugs, opiates and opioids [morphine and congeners], sedating antidepressants and tranquillizers. Sedating antidepressants [eg amitriptyline, imipramine, doxepin, and mianserin] impair driving equally as blood alcohol conc of ≥80mg%. Driving under influence of drugs is prohibited by **S.185(b)** of Motor Vehicles Act, 1988 [MVA, 1988]. Punishment same as that for alcohol [**ch 40**].

(4) Children of mothers who used drugs of abuse during pregnancy have - risk of SIDS [**ch 27**].

(5) Can cause several complications, eg needle embolism [please see **ch 40** for details].

(6) **Mortality rates** –

(i) Among drug abusers are substantially -er than in normal populations. Unnatural causes far exceed those natural causes.

(ii) Reasons - (a) Diseases (I) cirrhosis (II) HIV (b) Homicide [Attempts to procure drug, Disinhibition, Paranoia] (c) Overdose [particularly among opioid users] (d) Suicide - - levels of suicide risk factors [childhood abuse, effects of acute intoxication, psychopathology, social isolation, unemployment] (e) Trauma [falls, vehicular accidents].

(7) **EDH**, **SDH** etc can be preserved for chemical analysis in late deaths, when drug has disappeared from peripheral blood [ch 40"MLI of ethyl alcohol].

47. Miscellaneous Poisons

I. FORMALDEHYDE

Formaldehyde (HCHO) is a gas at room temperature. Aqueous solutions of formaldehyde are referred to as ***formalin***. Saturated solution of formaldehyde

(100% formalin) contains 40% by volume or 37% by mass of formaldehyde (i.e. if 1000 g of commercial solution of formalin is taken, 370 g of it would be formaldehyde. It is customary to add a small amount of stabilizer, usually **10% to 12% methanol**; it helps prevent oxidization and polymerization of the formaldehyde, either of which could result in fire or explosion). (i) For histopathology work **10% formalin** is prepared by mixing 1 part formaldehyde (100% formalin) with 9 parts water. Since it contains 4 parts by volume of formaldehyde it is also known as **4% formaldehyde**. Thus **4% formaldehyde = 10% formalin**.

A. Signs and Symptoms

1. Acute exposure

(a) **Dermal Contact**: (1) skin hardening (2) cracking (3) bleeding (4) Contact dermatitis (b) **Inhalation**: (1) Burning of eyes and lacrimation (2) violent coughing (3) constriction in chest and palpitation4.asthma like symptoms (c) **Ingestion**: (1) Similar to strong corrosive acid. (2) Since methanol is added to formalin solution, concomitant **methanol toxicity** is also seen. (3) **Extreme tachycardia** – is a prominent feature (4) .body temperature.

2. Chronic exposure

(1) Chronic obstructive pulmonary disease
(2) Reduction in ventilatory capacity
(3) Optic neuritis.

B. Fatal Dose

60-90 ml[4]

C. Fatal Period

1-2 days.

D. Management

(1) **Stomach wash** – with milk, water, 1% ammonium carbonate or 0.1% solution of ammonia. The latter reacts with formalin to form harmless

Hexamine. (2) Activated charcoal (3) Mild saline catharsis (4) Keep the patient warm (5) Watch for GIT hemorrhage.

E. PM Appearances

(1) **Smell** – of formaldehyde on opening the body
(2) **Mucosa of stomach** – Hard and tough like leather. Red, inflamed and eroded
(3) **Internal organs** – congested
(4) **Liver** – Fatty degeneration.

F. ML Importance

(1) **Accidental poisoning** - occurs mostly. But suicidal and homicidal poisoning are also known
(2) **Occupational hazards** - Formaldehyde is used by embalmers and laboratory workers. It is a potential occupational carcinogen. Embalmers and funeral directors exhibit (i) a higher incidence of leukemia and cancers of brain and colon. (ii) "**Embalmer's eczema**", a persistent skin condition. Embalmers must use masks and wear gloves during embalming work.
(3) **Toxicology** - Formalin should never be used as a preservative [**ch 5**].
(4) **Used illegally as a food preservative** – Formaldehyde is not allowed as a food preservative, but it continues to be used so. In 2005 in Indonesia and in 2007 in Vietnam, it was found to be used for **preservation of noodles** and other food products. Causing a major scare.

II. NITRITES AND NITRATES

Nitrites (NO_2^-) and ***nitrates*** (NO_3^-) are naturally occurring inorganic ions that can cause toxicity.

Salient features:

(1) **Microbial action** - in soil or water decomposes wastes containing organic nitrogen into ammonia, which is then oxidized to nitrite and nitrate.
(2) **Production** - Nitrite is easily oxidized to nitrate"Nitrate is thus predominantly found in groundwater and surface waters.
(3) **Contamination** - with nitrogen containing fertilizers (e.g. potassium nitrate and ammonium nitrate), or animal or human organic wastes, can raise the

concentration of nitrate in water.

(4) **Migration** - Nitrate-containing compounds in the soil are generally soluble and readily migrate with groundwater

(5) **Major salts:**

(i) Inorganic nitrates - (a) Nitrates may be converted to nitrites in a number of ways and can cause poisoning [eg by intestinal bacteria, by Bacillus subtilis spores in dried milk powder] (b) Poisoning with nitrates is easy because it tastes like and may be mistaken for common salt. (c) **Ex** - w Bismuth subnitrate - present in contaminated well water w Potassium nitrate w Silver nitrate w Sodium nitrate.

(ii) Organic nitrates - (a) ethyl nitrate (b) isosorbide dinitrate (c) mannitol hexanitrate (d) nitroglycerin

(iii) Inorganic nitrites - Sodium nitrite

(iv) Organic nitrites - Alkyl nitrites

(6) **Uses:**

(i) Amyl nitrite - used as medicines.

(ii) Alkyl nitrites - drugs of abuse [**ch 46**]

(iii) Sodium nitrate - as a mordant by weavers.

A. Mechanism of Action

(1) Relaxation of smooth muscles, especially of small blood vessels.

(2) Converts Hb to methHb by oxidizing iron from Fe^{2+} to Fe^{3+}.

Memory Aid 1: Understanding oxidation at atomic level

LEO - **L**oss of **E**lectrons is **O**xidation

B. Signs and Symptoms

(1) **General** - cyanosis, syncope, especially when attempting to stand upright

(2) **GIT** - nausea, vomiting, bloody, colicky diarrhea

(3) **CNS:** (i) disorientation, (ii) -Intracranial pressure, (iii) paralysis (iv) throbbing headache (v) vertigo (vi) coma followed by clonic convulsions

(4) **Blood** - methemoglobinemia

(5) **RS** - dyspnea

(6) **CVS** - .BP, palpitations, .pulse

(7) **Eye** - -Intraocular pressure.

C. Cause of Death

Hypotension or methemoglobinemia. Earlier a non-specific term "circulatory collapse" was used.

D. Fatal Dose

(1) Barium Nitrate – 15 g.
(2) Nitroglycerine – 200 mg
(3) Potassium Nitrate - 30 g.
(4) Silver Nitrate – 2-10 g
(5) Sodium nitrate – 1-2 g.

E. Fatal Period

Few hours to few days.

F. Management

(1) Emetics
(2) Activated charcoal
(3) Gastric lavage with intubation
(4) Cathartics [**ch 32**]
(5) For **methemoglobinemia** - Levels >25% may be dangerous.
(i) Supplemental oxygen
(ii) Methylene blue [**MB**] - converts MethHb back to Hb. (a) Routes - (I) IV -1% sol^{n} [10 mg/ml] 1-2 mg/kg administered IV slowly over 5 min followed by IV flush with normal saline. (II) 50mg/kg orally. (b) Mechanism of action - Normal blood level of MethHb is <1%. Its half-life is **1-3 h**. When blood levels >1%, methemoglobinemia results. Methylene blue is an electron acceptor. Thus it is an oxidizing agent.

Memory Aid 2: Methylene Blue

MBA - **M**ethylene **B**lue is **A**cceptor

In **low doses** [1-2 mg/kg] it accepts electrons from NADPH and passes them to MethHb^{3+}, reducing it back to Hb. Interestingly it behaves in diametrically opposite manners when in higher concentrations. At ≥ **5 mg/kg**, it starts accepting electrons from Hb itself producing methemoglobinemia.

(iii) **Exchange transfusion** - Indications (a) infants (b) patients who do not respond within ½-1 h (c) MethHb ≥ 70%.

Appendix 1: Medicolegally Important Sections and Acts

Note

1. **[Q] indicates a potential question [theory, viva, MCQ]. They have also been rendered in bold type. Other sections may be remembered for extra credit.**
2. **This appendix is meant to help the student quickly revise important sections. For details, chapter number mentioned must be referred to.**
3. **Abbreviations**

SI- Simple Imprisonment [no work allotted to prisoner]

RI - Rigorous Imprisonment [work allotted to prisoner]

4. **Chapter numbers in blue** refer to **chapters of IPC, CrPC and IEA [Also mentioned as a whole word "Chapter"]**; **chapter numbers in black** refer to **chapters within this book [mentioned as an abbreviated word "ch"]**. *Exact page within the chapter can be seen from the index.*

I. IPC

Chapter II: General Explanations [S 6-52A]

1. S.31. – Definition of a will - ch 28
2. S.34. - Acts done by several persons in furtherance of common intention — each person is liable equally - ch 28
3. S.40 - Definition of offence – ch 1.
4. **S.44[Q]. Definition of Injury - ch 11**.
5. S.46. Definition of Death – ch 8.
6. **S.51.** Oath - The word "oath" includes a solemn affirmation substituted by law for an oath, and any declaration required or authorized by law to be made before a public servant or to be used for the purpose of proof, whether in a Court of Justice or not - **ch 1**.
7. S.52. Good faith — Nothing is said to be done or believed in "good faith"

which is done or believed without due care and attention – ch 2.

Chapter IV: General Exceptions [S 76-106]

8. **S.80**. Accident in doing a lawful act — Nothing is an offence which is done by accident or misfortune, and without any criminal intention or knowledge in the doing of a lawful act in the lawful manner by a lawful means and with proper care and caution – **ch 1**.
9. **S.81**. Act likely to cause harm, but done without criminal intent, and to prevent other harm — Nothing is an offence merely by reason of its being done with the knowledge that it is likely to cause harm, if it be done without any criminal intention to cause harm, and good faith for the purpose of preventing or avoiding other harm to person or property – **ch 1**.
10. **S.82**[Q]. **Age of criminal responsibility** in India is **7 y - ch 3**.
11. **S.83**[Q]. **Act of child between 7-12 y** of **immature understanding** — not an offence - **ch 3.**
12. **S.84**[Q]. **Criminal responsibility of mentally ill**. Indian equivalent of British McNaugthen rule - **ch 28**.
13. **S.85**[Q]. **Person intoxicated against his will** - not responsible for his criminal actions - **ch 31**.
14. **S.86**. Person intoxicated voluntarily - Knowledge of act is presumed; intention to do the act is to be proven by prosecution - **ch 31**.
15. **S.87**. To participate in a risky activity, the consenting person must be >18 y. Not meant for doctors - **ch 2**.
16. **S.88**[Q]. **Act done is in good faith for the benefit of the person** [as in case of doctor treating patient] the consenting person must be >12 y. Meant for doctors - **ch 2**.
17. **S.89**[Q] . **Consent on behalf of minors - ch 2**.
18. **S.90**[Q]. **Conditions of a valid consent - ch 2**.
19. **S.92**[Q]. **Consent not required to save the life of person - ch 2**.

Chapter X - Of Contempts Of The Lawful Authority Of Public Servants [s 172-190]

20. **S.172**. Absconding to avoid service of summons or other proceeding — **ch 1**.
21. **S.174**. Non-attendance in obedience to an order from public servant -

Applies also to **non-attendance in court after service of summons — ch 1**. Not to be confused with **S.174 CrPC**.

22. **S.176**. Omission to give notice or information to public servant by person legally bound to give it — **ch 31.**
23. **S.178**. Refusing oath or affirmation when duly required by public servant to make it — **ch 1**.

Chapter XI: Of False Evidence And Offences Against Public Justice [S.191-229A]

24. **S.191^{Q}. Definition of perjury** [Giving false evidence] — **ch 1**.
25. **S.193^{Q}. Punishment for perjury — ch 1**.
26. **S.197**. Issuing false certificate — **ch 1**.
27. **S.198**. Using false certificate — **ch 1**.
28. **S.201^{Q}. Causing disappearance of evidence of offence — ch 31**.
29. **S.202.** Intentional omission to give information of offence by person bound to inform — **ch 31**.
30. **S.228A.** Identity of victim of rape cannot be disclosed **- ch 25**.

Chapter XIV: Of Offences Affecting The Public Health, Safety, Convenience, Decency And Morals [S268-294A]

31. **S.268.** Public nuisance **-** Exhibitionism, transvestism, fetishism, masturbation in public **- ch 25**.
32. **S.269**. Negligent act likely to spread infection of disease dangerous to life, eg HIV **- ch 2, 25**. [**Mnemonic - 69** is a sexual position]
33. **S.270.** Malignant act likely to spread infection of disease dangerous to life **- ch 2, 25**.

[**Both 269 and 270 are applicable in ML aspects of HIV and AIDS. Also sale of adulterated food**]

34. **S.272.** Adulteration of food or drink — **ch 31**.
35. **S.273.** Sale of noxious food or drink — **ch 31**.
36. **S.274.** Adulteration of drugs — **ch 31**.
37. **S.275.** Sale of adulterated drugs — **ch 31**.
38. **S.276.** Sale of drug as a different drug or preparation — **ch 31**.
39. **S.279**. Rash driving or riding on a public way so as to endanger human life "6 months or 1000 Rs fine or both.

40. **S.284**[Q]. **Negligent conduct with respect to poisonous substance** — **ch 31**.
41. **S.285**. Negligent conduct with respect to fire or combustible matter — **ch 14**.
42. **S.287**. Negligent conduct with respect to machinery — **ch 11**.
43. **S.290**. Punishment for public nuisance in cases not otherwise provided for, eg transvestism — **ch 25**.
44. **S.294**. Obscene acts and songs, eg Exhibitionism, masturbation in public — **ch 25**.

Chapter XV: Of Offences Relating to Religion [S.295-298]

45. **S.297**. Trespassing on burial places, etc, eg necrophilia — **ch 25**.

Chapter XVI: Of Offences Affecting The Human Body [S.299-377]

46. **S.299**[Q]. **Definition of culpable homicide - ch 11**.
47. **S.300**[Q]. **Definition of murder - ch 11**.
48. **S.301**. Culpable homicide by causing death of person other than person whose death was intended - **ch 11**.
49. **S.302**[Q]. **Punishment for murder - ch 11**.
50. **S.303 -** Punishment for murder by life-convict - **ch 11**.
51. **S.304**[Q]. **Punishment for culpable homicide - ch 11**.
52. **S.304A**[Q]. **Causing death by rash and negligent act** [including death by **medical negligence**] - **ch 11**.
53. **S.304B**[Q]. **Dowry death - ch 11**.
54. **S.305**. Abetment of suicide of child or insane person - **ch 11**.
55. **S.306**. Abetment of suicide - **ch 11**.
56. **S.307.** Attempt to murder - **ch 11**.
57. **S.308.** Attempt to commit culpable homicide - **ch 11**.
58. **S.309**[Q]**. Attempt to commit suicide - ch 11**.
59. **S.312.** Causing miscarriage - **ch 26**.
60. **S.313.** Causing miscarriage without woman's consent - **ch 26**.
61. **S.314.** Death caused by act done with intent to cause massacring - **ch 26**.
62. **S.315.** Act done with intent to prevent child being born alive or to cause it to die after birth - **ch 26**.
63. **S.316.** Causing death of quick unborn child by act amounting to culpable homicide - **ch 26**.

64. **S.317.** Exposure and abandonment of child <12 y, by parent or person having care of it - **ch 3**.
65. **S.318.** Concealment of birth by secret disposal of dead body - **ch 27**.
66. **S.319[Q]. Definition of Hurt** - **ch 11**.
67. **S.320[Q]. Definition of Grievous Hurt** - **ch 11**.
68. **S.321.** Voluntarily causing hurt - **ch 11**.
69. **S.322.** Voluntarily causing grievous hurt - **ch 11**.
70. **S.323[Q]. Punishment for voluntarily causing hurt** - **ch 11**.
71. **S.324[Q]. Punishment for voluntarily causing hurt by dangerous weapons or means** - **ch 11**.
72. **S.325[Q]. Punishment for voluntarily causing grievous hurt** - **ch 11**.
73. **S.326[Q]. Punishment for voluntarily causing grievous hurt by dangerous weapons or means** - **ch 11**.
74. **S.326A[Q]. Voluntarily causing grievous hurt by use of acid – Definition and punishment** - **ch 11**.
75. **S.326B[Q]. Voluntarily throwing or attempting to throw acid** – **Definition and punishment** - **ch 11**.
76. **S.328.** Causing hurt by means of poison, with intent to commit an offence**,** eg Drug Facilitated Sexual Assault - **ch 31**.
77. **S.336.** Punishment for negligent act when no injury to the patient occurs - **ch 2**.
78. **S.337.** Punishment for negligent act when hurt is caused to patient - **ch 2**.
79. **S.338.** Punishment for negligent act when grievous hurt is caused to patient - **ch 2**.
80. **S.339.** Wrongful restraint - **ch 11**.
81. **S.340.** Wrongful confinement - **ch 11**.
82. **S.341.** Punishment for wrongful restraint - **ch 11**.
83. **S.342.** Punishment for wrongful confinement - **ch 11**.
84. **S.350.** Intentionally using force on any person - **ch 2**.
85. **S.351[Q]. Assault** - **ch 2**.
86. **S.352.** Punishment for Assault or Criminal force - **ch 11**.
87. **S.354[Q]. Assault or criminal force to woman with intent to outrage her modesty** - 1-5y +fine

88. **S.354A.** Sexual harassment - **ch 25**.
89. S.**354B**. Assault or criminal force to woman with intent to disrobe or compelling her to be naked- **ch 11**.
90. S.**354C**. Voyeurism – **ch 25**
91. S.**354D**. Stalking – **ch 25**
92. S.**361.** Kidnapping from lawful guardianship - **ch 3.**
93. S.**362.** Abduction - **ch 3.**
94. S.**363**^{Q}. **Punishment for kidnapping - ch 3.**
95. S.**363A.** Kidnapping or maiming a minor for purposes of begging - **ch 3.**
96. **S.364**. Kidnapping or abducting for murder - **ch 3.**
97. S.**364A.** Kidnapping or abducting for ransom - **ch 3.**
98. S.**365.** Kidnapping or abducting to secretly and wrongfully confine - **ch 3**
99. S.**366.** Kidnapping or abducting woman to compel marriage or illicit intercourse - **ch 3**
100. **S.366A.** Procuring of girl <18 y for illicit intercourse - **ch 3**
101. **S.366B.** Importation of girl <21 y from J&K or from foreign country for illicit intercourse - **ch 3**
102. **S.367.** Kidnapping or abducting for causing grievous hurt, slavery or unnatural sexual act - **ch 3**
103. **S.368.** Wrongfully concealing or keeping in confinement, kidnapped or abducted person - **ch 3**
104. **S 369**. Kidnapping or abducting child <10 y with intent to steal from its person - **ch 3**

Memory Aid 1: S.363, 366 and 369: Most imp sections related to kidnapping

363, **366** and **369** are in arithmetic progression with a difference of **3**. Furthermore 363 has **2 sub-laws**, 366 has **3** and 369 has **1**. [First write 1"2"3. Then transpose 1 to the end to get 2"3"1]

105. **S.372**. Selling minor for purposes of prostitution, etc. - **ch 3**
106. **S.373**. Buying minor for purposes of prostitution, etc. - **ch 3**
107. **S.375**^{Q}. **Definition of Rape - ch 25**
108. **S.376**^{Q}. **Punishment for Rape - ch 25**
109. **S.377**^{Q}. **Unnatural offences - ch 25**

Chapter XX: Of Offences Relating To Marriage [S493-8]

110. **S. 494**. Marrying again during lifetime of husband or wife - **ch 23**

111. **S. 495.** Same offence with concealment of former marriage from person with whom subsequent marriage is contracted **- ch 23**

Memory Aid 2: S.497, IPC

Sum of all digits in **497** is **20** [a round figure]. Also comes immediately before **498A**, another vital section in relation to women]

Chapter XXA: Of Cruelty By Husband Or Relatives Of Husband [S.498A – only one section]

112. **S. 498A**[Q]. **Punishment to husband or relative of husband of a woman subjecting her to cruelty - ch 23.**

Chapter XXI - Of Defamation [S.499-502]

113. **S. 499**[Q]. **Defamation.** Breaking of professional secrecy **- ch 2.**

114. **S. 500**[Q]. **Punishment for defamation - ch 2.**

Chapter XXII: Of Criminal Intimidation, Insult And Annoyance [S.503-510] - CIIA

115. **S. 509**. Word, gesture or act intended to insult the modesty of a woman - Similar to **s354 - ch 25.**

116. **S. 510**. Misconduct in public by a drunken person **- ch 40**.

Chapter XXIII: Of Attempts To Commit Offences [S.511 – only one section]

117. **S. 511**. Punishment for attempting to commit offences punishable with imprisonment for life or other imprisonment **- ch 26**.

II. CRPC

Chapter I: Preliminary [S.1-5]

1. **S.2(c)** – Definition of cognizable offence – **ch 1**.
2. **S.2(l)** – Definition of non-cognizable offence – **ch 1**.
3. **S.2(u)** Public prosecutor [PP]– **ch 1**.

Chapter II: Constitution of criminal courts and offences [S.6-25A]

4. **S.8.** Definition of metropolitan areas – **ch 1**.
5. **S.9.** Court of Session – **ch 1**.

Chapter III: Powers of courts [S.26-35]

6. **S.26a.** Rape cases to be tried by a Court presided over by a woman **– ch 25**.
7. **S.28.** Sentences which High Courts and Sessions Judges may pass – **ch 1**.
8. **S.29.** Sentences which Magistrates may pass – **ch 1**.

Chapter IV-B: Aids to the Magistrates and the Police [S.37-40]

9. **S.39^{Q}. Public to give information of certain offences – ch 31**.

Chapter V: Arrest of Persons [S.41-60]

10. **S.53^{Q}**. Examination of accused by medical practitioner at the request of police officer – **ch 11**.
11. **S.53A^{Q}**. Examination of person **accused of rape** by medical practitioner – **ch 25**.
12. **S.54^{Q}. Examination of arrested person by medical officer – ch 2**.

Chapter VI: Process to compel appearance [S.61-90]

Chapter VII: Process to compel the production of things [S.91-105]

13. **S.91**. Summons to produce document or other thing (Subpoena duces tecum) **– ch 1**.

Chapter IX: Order For Maintenance Of Wives, Children And Parents [S125-128]

14. **S.125**. Order for maintenance of wives, children and parents. This section is important for paternity disputes, illegitimacy, affiliation cases etc – **ch 24**.

Chapter XII: Information To The Police And Their Powers To Investigate [S.154-176]

15. **S.154.** Information in Cognizable cases – **ch 1**.
16. **S.155.** Information in non-cognizable cases – **ch 1**.
17. **S.157(1)(b)** - Investigation in relation to an offence of rape – **ch 25**.
18. **S.164(5A)** - Judicial magistrate to record the statement of victim of rape and some other sexual offences – **ch 25**.
19. **S.164A^{Q}. Medical Examination of the victim of rape ch 25**.

Memory Aid 3: S.164A, CrPC

Corresponding section for accused is (***S.53***) Add ***111 164A***

20. **S.173(1A).** Investigation in relation to rape of a child – **ch 25**.
21. **S.174[Q]. Police inquest - ch 1**.
22. **S.175.** Power of police to summon persons - **ch 1**.
23. **S.176[Q]. Magistrate inquest- ch 1**.

Chapter XIV: Conditions requisite for initiation of proceedings [S.190-199]

24. **S.198(6)**. Cognizance of marital rape not to be taken by court if wife is <18 y and >1 y has elapsed from the date of commission of offence – **ch 25**.

Chapter XXIII: Evidence in Inquiries and Trials [S.272-299]

25. **S.273** - Victim of rape or other sexual offences if <18 y, not to be confronted with the accused – **ch 25**
26. **S.291** - Evidence of a doctor recorded in court can be used in other trials – **ch 1**.
27. **S.292** - Written reports of mint officers etc accepted as such – **ch 1**.
28. **S.293** - Reports of certain Government scientific experts admissible without oral evidence – **ch 1**.

Chapter XXIV: General provisions as to enquiries and trials [S.300-327]

29. **S.300**. Person once convicted or acquitted not to be tried for same offence [Res judicata] – **ch 2**.
30. **S.309(1).** In some cases of rape, inquiry or trial shall be completed within 2 months – **ch 25**.
31. **S.327(2).** The enquiry into and trial of rape shall be conducted in camera – **ch 25**.
32. **S.327(3).** Proceedings of trial of rape not to be published without previous permission of court – **ch 25**.

Chapter XXVII - The Judgment [S.353-365]

33. **S.357B** - Additional compensation by state to be given over and above the fine paid by convicted person in cases of **gang rape** and **vitriolage** – **ch 25**.
34. **S.357C** - All hospitals, whether Govt or private to provide immediate first-aid or medical treatment free of cost to any victim of **rape** and **vitriolage** – **ch**

25.

Chapter XXVIII: Submission of Death Sentences for confirmation [S.366-371]

35. **S.366(1) CrPC**. Sentence of death passed by Sessions court must be submitted to High Court for confirmation – **ch 1**.

Chapter XXXII: Execution, Suspension, Remission And Commutation Of Sentences [Memory Aid Ea Sy Ra Ce] [S.413-435]

36. **S.416.** Commutation of capital sentence on pregnant woman – **ch 24**.
37. **S.433(b) . Power to commute sentence** — Appropriate Govt may **commute** life imprisonment to **14 y**. Ch 1. Pl also see S.55, IPC.

III. IEA [INDIAN EVIDENCE ACT]

Chapter II – Of the relevancy of facts [s5-55]

1. **S.6.** ***Res gestae*** – **ch 1**.
2. **S.32(1)^{Q}. Dying declaration** – **ch 1**.
3. **S.35** – Public records made in performance of official duties are acceptable in court of law – **ch 1**.
4. **S.45^{Q}. Expert witness** – **ch 1**.

Memory Aid 4: Two important sections are consecutive numbers

S.44, IPC - Injury
S.45, IEA - Expert Witness

Chapter IV – Of oral evidence [S.59-60]

5. **S.60**. Author of a book not to be called in a court as witness – **ch 1**.

Chapter V: Of Documentary Evidence [S.61-90A]

6. **S.62.** Primary evidence – **ch 1**.
7. **S.63.** Secondary evidence – **ch 1**.
8. **S.74**. Definition of public documents – These are the documents forming the acts or records of the acts (i) of the sovereign authority (ii) of official bodies and tribunals, and (iii) of public officers, legislative, judicial and executive of any part of India or of the Commonwealth, or of a foreign country **- ch 1**.
9. **S.76.** Certified copies of public documents – Must be given by public officers

having custody of such documents – **ch 1**.

10. **S.78.** How certain public documents can be proved in a court of law.

Chapter VII - Burden of proof [S.101-114A]

11. **S.105** - Burden of proving that case of accused comes within general exceptions of Ch IV of IPC – **ch 28**.
12. **S.107^{Q}. Presumption of life** – **ch 9**.
13. **S.108^{Q}. Presumption of death** – **ch 9**.
14. S**.112.** Period of Legitimacy of child – **ch 24**.
15. **S.113A.** Presumption as to abetment of suicide by a married woman – **ch 11**.
16. **S.113B.** Presumption as to dowry death – **ch 11**.
17. **S.114A^{Q}. Presumption of absence of consent in certain cases of rape** – **ch 25**.

Chapter IX – Of witnesses [S.118-134]

18. **S.118. Who may testify** – **ch 1**.
19. **S.119. Dumb witnesses** – **ch 1**.

Chapter X: Of The Examination Of Witnesses [S135-166]

20. **S.137**. Examination in chief – **ch 1**.
21. **S.138**. Order of examinations – **ch 1**.
22. **S.141**. Leading questions – **ch 1**.
23. **S.142**. When LQs can not be asked – **ch 1**.
24. **S.143**. When LQs may be asked – **ch 1**.
25. **S.154.** Leading questions by party to his own witness – **ch 1**.
26. **S.157**. **Victim survives** after making a dying declaration – witness must give oral evidence. D/dec used only or corroboration – **ch 1**.
27. **S.159**. Refreshing memory by witness during examination is allowed – **ch 1**.
28. **S.160**. Testimony from documents prepared by witness – ch 1.
29. **S.161**. Right of the adverse party as to writing used by witness to refresh memory – **ch 1**.

IV. MEDICOLEGALLY IMPORTANT ACTS AND RULES

1. Biomedical waste (Management and Handling) Rules 1998.
2. Consumer Protection Act 1986 – **Ch 2**.
3. Cantonments Act, 2006– **Ch 2**.
4. **Children Act 1960** – Provides for the care, protection, maintenance, welfare, training, education and rehabilitation of neglected or delinquent children and for the trial of delinquent children in the Union territories.
5. Clinical Establishments Act 2010 - **ch 2**
6. Drugs and Cosmetics Act 1940 – **ch 31**
7. Dentists Act 1948 - **ch 2**
8. Dock Workers (safety, health and welfare) Act 1986
9. Drugs and Magic Remedies (Objectionable advertisement) Act 1954
10. Drug Control Act 1950
11. Epidemic Diseases Act 1897
12. Employees' State Insurance Act 1948
13. Environment Protection Act 1986
14. Factories Act 1948
15. Fatal Accidents Act 1855
16. Homeopathy Central Council Act 1973 - **ch 2**
17. Information Technology Act 2000 – ch 25 [Shakti Mills Gang rape case]
18. Insecticides Act 1968
19. Indian Majority Act 1875
20. Indian Medicine Central Council Act, 1970 - **ch 2**
21. Indian Medical Council Act, 1956 - **ch 2**
22. Indian Medical Degrees Act 1916 - **ch 2**
23. Leprosy Act 1898
24. Indian Medical Degrees Act 1916
25. Indian Medical Council Act 1956

Indian Nursing Council Act - **ch 2**

26. Maternity Benefit Act 1961
27. Mental Health Act 1987- **ch 28**
28. Minimum Wages Act 1948
29. Medical Council of India Regulations - **ch 2**
30. Medical Termination of Pregnancy Act 1971
31. Mines Act 1952

32. Motor Vehicles Act 1988
33. Personal Injuries (Emergency Provisions) Act 1962
34. Personal Injuries (Compensation, Insurance) Act 1963
35. Pharmacy Act 1948 - **ch 2**
36. Poisons Act 1919
37. Preconception and prenatal Diagnostic Techniques (Regulations and Prevention of misuse) Act 1994
38. Protection of Human Rights Act 1993
39. Registration of Births and Death Act 1969
40. Transplantation of Human Organs Act 1994
41. Vaccination Act 1880
42. Workmen's Compensation Act 1923

Appendix 2: Important Values Regarding Fatal Doses etc

Q in superscript denotes a possible theory or viva question.

I. COMMON NAMES AND ACTIVE PRINCIPLES OF POISONOUS PLANTS

S.No.	Poisonous plant (Chapter No.)	Common name[1]	Main active principles[2]
1.	*Abrus precatorius* (**ch 37**)	*Gunchi, Ratti*Q	Abrin[Q], Abrine
2.	*Aconitum napellus* (**ch 43**)	*Meetha Bish, Meetha zeher*Q	Aconitine[Q], Mesaconitine
3.	*Atropa belladonna* (**ch 41**)	Deadly nightshadeQ	Atropine[Q], hyoscyamine, scopolamine
4.	*Calotropis gigantea* (**ch 37**)	*Akdo, Madar*Q, *Rakta Arka*	Calactin, uscharin
5.	*Calotropis procera* (**ch 37**)	Apple of Sodom, *Shveta Arka*	Calactin, uscharin
6.	*Cannabis sativa* (**ch 41**)	Indian hemp[Q], pot	Δ^9- tetrahydrocannabinol[Q], cannabidiol
7.	*Capsicum annum* (**ch 37**)	chili, *Lal mirch*	Capsaicin, capsicin
8.	*Cerbera odollam* (**ch 43**)	Suicide tree, Ordeal tree, *Pilikirbir*	Cerapain, Odolotoxin
9.	*Cerbera thevetia (syn, Thevetia peruviana)* (**ch 43**)	*Pila kaner*, yellow oleander	Cerberin, Neriifolin
10.	*Conium maculatum* (**ch 42**)	Poison hemlock, *Vish jeera*	Coniine
11.	*Croton tiglium* (**ch 37**)	Jaipala, Jamalgota	Crotin, crotonoside
12.	*Strychnos curare* (**ch 42**)	Dog button, *Kuchila*	Strychnine, brucine
13.	*Datura alba* (**ch 41**)	Safed Datura	Atropine[Q], hyoscyamine, scopolamine
14.	*Datura fastuosa* (**ch 41**)	Thorn apple	Atropine[Q], hyoscyamine, scopolamine
15.	*Datura niger* (**ch 41**)	Kala Datura	Atropine[Q], hyoscyamine, scopolamine
16.	*Datura stramonium* (**ch 41**)	Jimson weed, Thorn apple	Atropine[Q], hyoscyamine, scopolamine
17.	*Hyoscyamus niger* (**ch 41**)	Henbane, *Khorasani ajwayan*	Atropine[Q], hyoscyamine, scopolamine
18.	*Nerium oleander* (**ch 43**)	White *Kaner*, pink *Kaner*	Nerin[Q], Oleandrin
19.	*Nicotiana tabacum* (**ch 43**)	tobacco	Nicotine[Q], Nicotianine
20.	*Papaver somniferum* (**ch 40**)	Afim, Pasto	Morphine[Q], codeine[Q]

1 Only the most well known common names are given here. For an exhaustive list of ALL common names, please refer to the concerned chapter.

2 Only the most common active principles are given here. For an exhaustive list of ALL active principles, please refer to the concerned chapter.

II. FATAL DOSE, FATAL PERIODS AND ANTIDOTES IN A NUTSHELL

S.No.	Poison (ch no)	Fatal dose	Fatal period	Antidote
1.	Acid, hydrochloric (**ch 33**)	15-20 mL	12-24 h	-
2.	Acid, nitric (**ch 33**)	10-15 mL	12-24 h	-
3.	Acid, sulfuric (**ch 33**)	10-15 mL	12-24 h	-
4.	Aconite (**ch 43**)	Root 1-2 g Aconitine 2 mg	2-6 h	Atropine

S.No.	Poison (ch no)	Fatal dose	Fatal period	Antidote
5.	Aluminum phosphide^{Q} (**ch 35**)	1-3 tab	24 h	-
6.	Arsenic trioxide^{Q} (**ch 36**)	200-300 mg	1-2 d	BAL
7.	Aspirin [acetylsalicylic acid] (**ch 45**)	15-20 g	Few min-several h	-
8.	Atropine (**ch 41**)	120 mg	24 h	Physostigmine
9.	Barbiturates [long acting]^{Q} (**ch 40**)	3-5 g	1-2 d	-
10.	Barbiturates [short acting]^{Q} (**ch 40**)	1-2 g	1-2 d	-
11.	Cannabis (**ch 41**)	**THC** – 30mg/kg; **Charas** - 2g/kg; **Ganja** - 8g/kg; **Bhang** - 10g/kg	Several days	-
12.	Cantharides (**ch 38**)	10-80 mg	24-36 h	-
13.	Carbon dioxide (**ch 44**)	90,000 ppm	5 min	O_2
14.	Carbon monoxide (**ch 44**)	50,000 ppm	few seconds	O_2
15.	Castor seeds [crushed]^{Q} (**ch 37**)	5 well-chewed seeds	2 days	-
16.	Cocaine (**ch 41**)	1 g IV	Few minutes to few hrs.	-
17.	Copper sulphate^{Q} (**ch 36**)	0.15-0.3 g/kg	1-3 days	d-Penicillamine, BAL

18.	Croton^{Q} (**ch 37**)	4 seeds; 20 drops of croton oil	6 h-3 d	-
19.	Curare (**ch 42**)	60 mg	1-2 h	
20.	Cyanide^{Q} (**ch 34**)	(1) HCN - 50 to 60 mg (2) NaCN, KCN – 200 to 300 mg	(1) HCN - 2-10 minutes (2) KCN or NaCN - 30 min	Amyl nitrite, sodium nitrite, sodium thiosulphate
21.	Datura (**ch 41**)	(1) Atropine or hyoscyamine – 120 mg (2) Scopolamine – 30 mg (3) Datura seeds – 50-100	24 hrs	Physostigmine
22.	DDT (**ch 35**)	15-30 g	One to several hrs	-
23.	Diamond powder (**ch 34**)	Not fatal	Not fatal	-
24.	Diazepam (**ch 46**)	100-300 mg/kg [20g for a 70 kg man].	1 day	Flumazenil
25.	Diazinon (**ch 35**)	1 g	24 hrs	Atropine
26.	Digitalis (**ch 43**)	(1) Digitalin – 15-30 mg (2) Digoxin [present in D. lanata] – 10 mg (3) Digitoxin – 4mg; (4) Leaves 1-2 (2g)	24 hrs	Digoxin-Specific Antibody Fragments or Fab
27.	Ethyl alcohol (**ch 40**)	(1) Adults - 5-8 g/kg absolute ethanol. (2) Children - 3 g/kg (3) Blood conc → >500 mg%	12-24 h	-
28.	Ethylene glycol (**ch 40**)	100-200 mL	3 days	Ethanol, fomepizole
29.	Formaldehyde (**ch 47**)	60-90 ml	1-2 days	1% ammonium carbonate; 0.1% sol^{n} of ammonia

S.No.	Poison (ch no)	Fatal dose	Fatal period	Antidote
30.	Heroin (**ch 40**)	50 mg	6-12 hrs	Naloxone
31.	Hydrogen sulphide (**ch 44**)	(1) 400-700ppm - dangerous after an exposure of half to 1 h. (2) >1000 ppm - instantly fatal.	Few min to few hrs depending of dose	Same as that of CN. Also O_2.
32.	Iron (**ch 36**)	60 mg/kg	1-2 days	Desferrioxamine
33.	Isopropanol (**ch 40**)	(1) 250-300 mL; (2) Blood levels >300 mg%	Variable depending on dose	-
34.	Lead acetate (**ch 36**)	20 g	1-2 days	EDTA, BAL
35.	Lead carbonate (**ch 36**)	40 g	1-2 days	EDTA, BAL
36.	Malathion^{Q} (**ch 35**)	1 g	24 hrs	Atropine
37.	Marking nut^{Q} (**ch 37**)	5-10g	12-24 h	-
38.	Mercuric chloride^{Q} (**ch 36**)	1-4g	3-5 days	BAL, DMSA, Cuprimine
39.	Methyl alcohol^{Q} (**ch 40**)	100-200 ml; Blood level - 200 mg%	24-36 hour	4-methyl pyrazole; 10% ethanol in a dose of 10mL/kg
40.	Morphine^{Q} (**ch 40**)	200 mg	6-12 hrs	Naloxone
41.	Mushrooms (**ch 39**)	One mushroom	24 hrs	Anti-phalloidin antibodies
42.	Nicotine (**ch 43**)	50-100 mg	5-15 min	Atropine
43.	Oleander (**ch 43**)	(1) Root: 15-20 g (2) Leaves: 5-15	24-36 hrs	Digoxin-Specific Antibody Fragments or Fab
44.	Opium [raw]^{Q} (**ch 40**)	2 g	6-12 hrs	Naloxone
45.	Oxalic acid (**ch 33**)	600 mg/kg	1-2 hrs	**Calcium gluconate IV**
46.	Paracetamol (**ch 45**)	20-25 g [40-50 tab]	2-4d	N-Acetylcysteine
47.	Parathion^{Q} (**ch 35**)	175 mg orally; 80 mg IM	24 hrs	Atropine
48.	Phosphorus [red] (**ch 34**)	Not poisonous	Not poisonous	-
49.	Phosphorus [white]^{Q} (**ch 34**)	1 mg/kg	½ day to 8 days	Vitamin K
50.	Snake [Common cobra] (**ch 34**)	15 mg	8 hrs	Polyvalent Antisnake Venom [PASV]
51.	Snake [Common krait] (**ch 34**)	6 mg	18 hrs	PASV
52.	Snake [Russell's viper] (**ch 34**)	20 mg	3 days	PASV
53.	Snake [Saw scaled viper] (**ch 34**)	8 mg	5 days	PASV
54.	Strychnine^{Q} (**ch 42**)	30mg/kg; 1 crushed seed on *Nux vomica*	1-2 hrs	Barbiturates
55.	Tetraethyl lead (**ch 36**)	100 mg/kg	1-2 days	EDTA, BAL
56.	Thallium (**ch 36**)	6-40 mg/kg	24-36 hrs	1% KI; Prussian blue

Appendix 3: Weights and Measurements of Organs

Table 1: Normal weights and measurements of organs in adults [M-Male; F-Female; NB – Newborn; P – Puberty; BW – Body Weight]

S.No.	Organ	Weight/measurement
(1) **CNS**		
1.	Brain	(1) **Wt** - (i) **NB** - 380 g; (ii) **P** - 1150 g; (iii) **F** - 1275 g; (iv) **M** - 1400 g (v) **NB** - 1.4% of Body Weight in adults (2) **Dimensions** - (i) **Vertical diameter** = **F** :15-16 cm; **M** :16-17 cm (ii) **Coronal [transverse] diameter** = **F** :11-12 cm; **M** :12-13 cm (iii) **Child's brain** – attains adults dimensions by about 5 y
2.	Cerebellum	150 g
3.	Spinal cord	(1) **Wt:** 27-28 g; (2) **length** - 45 cm (3) **AP diameter** (i) **Cervical** -13.5 mm (ii) Thoracic – 10 mm (iii) Lumbar – 12 mm (4) **transverse diameter** (i) **Cervical** -9 mm (ii) **Thoracic** – 8 mm (iii) **Lumbar** – 9 mm
4.	Pituitary	(1) **size:** 2.1x1.4x0.5 cm (2) **wt:** 500-600 mg; [up to 1g in pregnant women]
5.	Pineal gland	(1) Dimensions: 10x5x5 mm (2) wt: 100—180 mg
(2) **CVS**		
6.	Heart	(1) 23 g [NB]; 150 g [P]; 250 g [F]; 300 g [M]; (2) 0.4-0.45% of BW in adults; (3) Length – 12.5 cm; (4) Width [at AV groove]-9-10 cm (5) LV+septum: RV ratio = 2.3–3.3 : 1 (6) Left ventricular hypertrophy >3.6:1 or LV+S >225 g (7) Right ventricular hypertrophy <2.1 or RV > 80 g (8) Circumference – 25 cm
7.	Pericardial fluid	5-50 ml
8.	Atria	Thickness of walls : 1-2 mm
9.	Right Ventricle	Thickness : 3-5 mm
10.	Left ventricle	Thickness : 10-15 mm [at 1 cm below AV valve]
11.	Circumferences of valves	(1) **T**ricuspid – 12 cm (2) **M**itral – 10 cm (3) **P**ulmonary – 8.5 cm (4) **A**ortic - 7.5 cm **Memory Aid 1–** **T**ricuspid **T**ops in circumference with **T**welve cm. **Memory Aid 2–** (i) TaMPA. (ii) Differences in circumferences are in arithmetic progressions [2, 1.5, 1 cm] as we go down the list. **The figures are similar to intraocular pressures after death [ch 9]**
12.	Aorta	(1) **Length:** 42-50 cm (2) **Diameter** - (i) **Ascending** = 2.5 cm (ii) **Descending** = 1.5-2.0 cm (iii) **Abdominal** = 1.2-1.5 cm (3) **Thickness of wall** = 1.5-2 mm
(3) **Respiratory**		
13.	Trachea	12 cm
14.	Bronchi	(1) Right - 2.5 cm (2)Left - 3.5-5 cm
15.	Lungs	(1) Right – 360-570 g [mean 450 g] (2) Left – 325-480 g [mean 375 g] (3) Both – 1% of BW

S.No.	Organ	Weight/measurement
(4) **Immune / lymphatic system**		
16.	Spleen	(1) **Wt:** 150-200 g; 0.2% of BW (2) **Size:** 10-12 x 7-8 x 2.5-4 cm
17.	Thymus	(1) birth : about 15 g (2) puberty : about 35 g (3) 25 y : 25 g (4) 60 y : < 15 grams (5) 70 y : 5 g
18.	Thoracic duct	40-45 cm
(5) **GIT, Digestive system**		
19.	Pharynx	12 cm
20.	Salivary glands	(1) Parotid: 25 g; (2) sublingual: 3-4 g (about almond size); (3) submandibular - (i) **Wt:** 6-7 g (about walnut size) (ii) size: 3.5 x 3 x 1.8 cm
21.	Esophagus	(1) **Length** = 25-30 cm (2) **Diameter** = 3 cm (3) **Thickness of wall** = 6-8 mm
22.	Stomach	(1) **Length:** 25-30 cm; (2) **Thickness of wall** – 6 mm (3) **Wt** - 300 g; (4) **Capacity** - 1200 mL
23.	Duodenum	20-30 cm
24.	Jejunum	Upper 2/5th of small intestine
25.	Ileum	Lower 3/5th of small intestine
26.	Small intestine	(1) **Length:** 550-650 cms; (2) wt: 800-900 g
27.	Cecum	6-7 cm
28.	Ascending colon	20 cm
29.	Transverse colon	50 cm
30.	Descending colon	12-15 cm
31.	Pelvic colon	40-45 cm
32.	Large intestine	(1) **Length:** 150-170 cms; (2) **wt:** 600 g
33.	Rectum	12-15 cm
34.	Anal canal	2.5-4 cm
35.	Liver	(1) **Wt** = 1400-1500 g; (2) **Relative Wt** = 1.8% of Body Weight; (3) **Dimensions** = 30x18x9 cm; (4) **Diameter of lobules** = 1-2 mm
36.	Gall bladder	(1) **Length** =8 cm; (2) **Diameter** =4 cm (3) **Capacity** - 50 mL [when fully distended]
37.	Bile ducts	7.5 cm
38.	Pancreas	(1) **Wt** - 60 to 100 gm; 0.1% of BW (2) **Dimensions** - 12.5-15 x 4 x 3 cm
39.	Appendix	(1) **Length :** 7.5-10 cms (2) **Wt** : 4-5 g
(6) **Genitourinary system**		
40.	Kidney	(1) 120-150 g [F]; (2) 130-160 g [M] (3) In both sexes, left is **heavier** than the right by about **10 g**. (4) **Size** = 12x6x4 cm (5) **Thickness of cortex** = 6-8 mm (6) **Cortico-medullary ratio** = 1:3 (7) **Volume of pelvis** = 10-15 mL
41.	Ureter	25 cm
42.	Urinary bladder	(1) **Capacity** – (i) **Undistended** - 250 mL (ii) **Normal distension** – 500 mL (2) **Thickness of wall** : 2-3 mm; (3) **Wt [empty]** = 30-60 g
43.	Seminal vesicles	**Size – 4-4.5 x 1.5-1.7 x 0.8-1.0 cm**
44.	Prostatic urethra	2.5-4.0 cm

S.No.	Organ	Weight/measurement
45.	Membranous urethra	2 cm
46.	Penile urethra	15-20 cm
47.	Male urethra [total]	20-25 cm
48.	Female urethra	2.5-4.0 cm
49.	Inguinal canal	3.5 cm
50.	Vas deferens	40-45 cm
51.	Testes	(1) **Size - 5 x 2 x 3 cm [L,B,H]** (2) **Vol** - 12 cc-30 cc [average - 18 cc] (3) **Wt - 10.5-14 gm** [each]; Total 21-28 g; 0.05% of BW [combined]
52.	Prostate	(1) **Wt:** 15-20 g; (2) **Size** : 3.6 x 2.8 x 1.9 cm
53.	Ovary **[ch 24]**	(1) **Each ovary wt** = 2-3.5 gm.; (2) **size** = 3 cm x 1.5 cm x 1 cm
54.	Fallopian tubes	10-12 cm
55.	Uterus **[ch 24]**	(1) **Virgin** – **Wt :** 40-60 g; **Size:** 3″x2″x1″ (2) **Parous** – **Wt:** 80-120 g; **Size:** 4″x2.5″x1″
56.	Placenta **[ch 26]**	(1) **Volume** -500 ml; (2) **wt**- 500 g; (3) **Diameter**- 18.5 cm ; (4) **thickness**- 2.5 cm
57.	Vagina	(1) Anterior wall -7.5 cm (2) Posterior wall – 10 cm
(7) **Endocrine system**		
58.	Thyroid	(1) **Wt:** 20-40 g; (2) **Size :** 5-7cm x 3-4 cm x 1.5-2.5 cm
59.	Parathyroids	(1) **Size** - 6 x 4 x 2mm each. (2) **Wt** - 50 mg each [total 200 mg]
60.	Adrenals [Suprarenals]	(1) **Length** = 4.5 cm (2) **Breadth** = 3 cm (3) **Thickness** = 0.5 cm (4) **Wt** = 4-7 g
(8) **Musculoskeletal system**		
61.	Bones **[ch 3]**	M=4.5 kg; F=3.0 kg [dry weight]; in moist state they are about 12% of body weight
62.	Skeletal Muscles	28-30% of body weight
(9) **Miscellaneous**		
63.	Fats	Males : 20-25% of BW; Females :25-30% of BW
64.	Specific gravity **[ch 19]**	(1) body= **1.08**. (2) **Fat**-0.92 (3) **Brain**-1.04 (4) **Soft organs**-1.05 (5) **Muscle**-1.08 (6) **Bone**-2.01.

Appendix 4: Common Mistakes in Some Books

Enumerated below are **common mistakes** given in **some books.** For proper explanation of why the information is incorrect, pl refer to chapter number mentioned.

S.No.	Chapter	Incorrect information	Correct information
1.	1	Inquest of "deaths in police firing" is done by magistrate	Inquest of "deaths in police firing" is done by police.
2.	1	**1st Class Judicial Magistrate** can award a fine up to **5,000 Rs**.	He can award fine up to **10,000 Rs** [since **23.6.2006**]. Previous to this, he could indeed fine only up to Rs. **5,000**.
3.	1	**2nd Class Judicial Magistrate** can award a fine up to **1,000 Rs**.	He can award fine up to **5,000 Rs** [since **23.6.2006**]. Previous to this, he could indeed fine only up to Rs. **1,000**.
4.	2	*Res indicata* [given as a legal term, and even defined along with *res judicata*]	There is no such legal term as *res indicata*.
5.	3	Incidence of Davidson's body in females is about **6%** of cells.	The incidence is **3%**, i.e. out of 100 neutrophils in females, 3 would show Davidson's body.
6.	3	Negroids have fragmented medulla.	All human races have fragmented medulla. Fragmentation of medullas helps us to differentiate a human from an animal hair – not different races of man.
7.	3	Sciatic notch index is 4-5 in males and 5-6 in females	It is **145** in males and **166** in females. Figures 4-5 and 5-6 are for male and female fetuses respectively.
8.	3	Age of criminal responsibility is **5 years** according to *Railways Act [RA] 1989*	No such age is prescribed in *Railways Act*. A child of any age can be convicted
9.	3	In fingerprinting, **16 points** of comparison are accepted as proof of identity	This figure varies from country to country, and within India from state to state
10.	3	In dental charting systems, **quadrant grids** are shown in several notations	**Quadrant grids** were used only in **Zsigmondy** and **Palmer** systems. Since they were cumbersome, they were never used after Palmer system in 1870.
11.	4	Mass disaster is death of >12 victims in a single event	Mass disaster is not related to number of victims. It is any event that produces more fatalities than can be handled using local resources.
12.	5	Period for declaring a body unclaimed is 72 hours	Period for declaring a body unclaimed is **48 hours**
13.	8	Bishop's tripod of life/Bishop's criteria	Bichat's tripod of life/Bichat's criteria
14.	8	Somatic death is the complete and irreversible stoppage of circulation, respiration and brain functions	This is according to old concept. According to current concept, somatic death is death of brain stem.
15.	8	Surest sign of death is putrefaction	Signs of death today are determined according to Minnesota criteria. Putrefaction was considered surest sign of death only till 19th century.
16.	8	Negative autopsy includes autopsies which have been improperly carried out by the forensic pathologist	Negative autopsies are only those autopsies which have been carried out with utmost care and yet no cause of death is found. Autopsies which are improperly done are "**defective autopsies**".

S.No.	Chapter	Incorrect information	Correct information
17.	9	Postmortem staining can be seen *before death* in some cases like cholera	Any staining *occurring before death* cannot be called "postmortem staining".
18.	9	PM staining is black in opiates	It is deep blue. There is no reason why it should be black
19.	9	Rigor mortis [and adipocere] is not seen in fetuses < 7 months	They are seen in all fetuses who have muscle and fat
20.	9	Slight expulsion of seminal fluid at tip of penis suggests sexual activity prior to death	It is due to contraction of muscles of seminal vesicles by rigor mortis and is seen in all types of deaths
21.	11	Justifiable and excusable homicide are genuine legal terms, which are even defined in many books	Indian law does not define these two terms anywhere
22.	11	S309 IPC (attempt to commit suicide) has been decriminalized	It is merely a proposal at this stage.
23.	11	Simple injury and Grievous injury are genuine legal terms, which are even defined in many books	Indian law does not define these two terms anywhere. The correct terms are "Hurt" and "Grievous Hurt"
24.	11	Punishment for dowry death [u/s 304B IPC] is 7 y + 20,000 Rs. fine. Different books give different amounts of fine	No fine is prescribed for dowry death. Punishment is for a minimum of 7 years, which can go up to life
25.	11	Scar on the face of young unmarried girl or an actress is grievous, while that on the face of an old woman is not, because in first two cases, it would affect their life	Law is same for everyone. Scar on the face of anyone is grievous
26.	12	Juice of *Calotropis gigantea* [madar] produces artificial bruises	It does not produce any bruise – true or artificial
27.	12	Lacerated looking incised wounds are seen in scrotum	Incised wound over scrotum looks exactly like incised
28.	12	Wounds produced by glass are lacerated	Wounds produced by glass are incised. Only if the glass is very thick, they would give the appearance of a cut laceration
29.	13	Gas produced by black powder is **3,000-4,500 cc/gram**; and by smokeless powder **12,000-13,000 cc/gram**	Black powder produces **280 cc/gram**; nitroglycerine **850 cc/gram** and nitrocellulose **950 cc/gram**
30.	14	Antemortem and postmortem blisters can be differentiated on the basis of proteins and chloride	They cannot be differentiated on this basis
31.	14	According to a Home Ministry circular, if the age of woman dying of dowry death with burns (or other reasons) and her age is <30 y, a panel of 2 doctors will conduct the postmortem	There is no such circular
32.	17	Fracture of outer table occurs in pond fracture	There is no **diploë**, and thus no outer table in newborns. Fracture may however be more pronounced on the outer aspect of skull.
33.	19	Commonest cause of death in hanging is combined asphyxia and venous congestion	Commonest cause in *complete hanging* is **asphyxia**; that in *incomplete hanging* is **occlusion of neck bv**.

S.No.	Chapter	Incorrect information	Correct information
34.	19	Slight expulsion of seminal fluid at tip of penis suggests antemortem hanging	It is due to contraction of muscles of seminal vesicles by rigor mortis and is seen in all types of deaths
35.	19	Cause of death in café coronary is asphyxia due to choking	COD in *café coronary* is **reflex vagal inhibition** of the **heart**
36.	23	Impotence and sterility are grounds for divorce	None of them is a ground for divorce according to Indian law. Impotence is a ground for voidable marriage. Sterility is not a ground for even voidable marriage.
37.	23	Questions of impotence and sterility arise in suits of adoption	The *Hindu Adoptions And Maintenance Act 1956* nowhere mentions impotence or sterility as a valid condition for adoption
38.	24	Hanging in a pregnant woman is postponed for 6 months [period varies in different books]	A pregnant woman **cannot be hanged** anytime even after birth of baby.
39.	25	Every act done against the will of a person is done without his consent	This is not so. An act may be done against the will, but with proper legal consent
40.	25	Sexual intercourse with an unconscious woman cannot be said to be against her will	Sexual intercourse with an unconscious woman is both against her will and without her consent
41.	25	A girl under 12 years is incapable of giving consent to sexual act u/s 90 IPC.	S90 IPC is a general section for consent. Specific section for sexual consent is s375 IPC, in which the age defined is **18 years**.
42.	25	**S154(4) IEA** states that in a case of rape, it may be shown by defense that the woman was of immoral character	There is no **s154(4) IEA**. Before 2002 there was **s155(4) IEA**, which indeed stated so, but **it has been removed in 2002**.
43.	25	Consent of victim is necessary for informing case of rape to the police	No such consent is necessary in law. Consent is required only for examination.
44.	25	In India, there is no age limit to bring a charge of rape against an accused. A boy of any age can be charged for rape.	No person <7 y can be charged of any offence [s82 IPC]. Thus age limit for accused to bring a charge of rape against him is 7 y.
45.	26	Under the **MTP Act 1971,** grounds for MTP are **4**	**MTP Act 1971** mentions only 2 grounds. Rest 2 are explanations
46.	27	Neonaticide is killing of an infant within **28 days** of birth.	Neonaticide is killing of an infant within **24 hours** of birth. This is despite the fact that the definition of **neonate** is a newborn up to the age of 28 days
47.	27	Age of viability in India is **210 days**	Age of viability in India is not defined in law, but by common consensus it is taken as **28 weeks**
48.	27	Wredin's test	Wreden's test
49.	31	**Poisons Act 1919** has been repealed in 1960.	It is still prevalent
50.	31	**Schedule E** of "*Drugs and Cosmetic Rules, 1945*" contains list of poisons	*Schedule E* has been omitted since **1982**. Instead there is schedule **E(1)** which gives a **list of poisonous substances** *under the Ayurvedic, Siddha and Unani Systems of Medicine*.
51.	31	**Schedule F** of "*Drugs and Cosmetic Rules, 1945*" contains a list of vaccines and sera.	**Schedule F** is divided in **4 parts** - F(I), F(II), F(III) and FF. **F(1)** is further subdivided into 3 parts. Part 1– **Vaccines**. Part II –**Antisera**. Part III- **Diagnostic antigens** [Bacterial Origin]

S.No.	Chapter	Incorrect information	Correct information
52.	31	**Schedule G** of "*Drugs and Cosmetic Rules, 1945*" contains hormone preparations	It contains drugs which must be taken "**under medical supervision**".
53.	31	**Schedule L** of "*Drugs and Cosmetic Rules, 1945*" contains list of antibiotics, antihistaminics and other recent chemotherapeutic agents	*Schedule L* has been omitted since **1982**.
54.	31	Only homicidal poisoning need to be informed to police. In some books Govt. doctor must inform all cases of poisoning, but private doctor only homicidal cases	All cases of poisoning [suicidal, homicidal, accidental] must be informed to police by all doctors, whether Govt or private
55.	32	Emetics are very good and effective agents in poisoning	Emetics are virtually obsolete now. May come in handy when nothing else is available.
56.	33	The only corrosive in which gastric lavage can be done is carbolic acid	Gastric lavage can be done with due care in some other organic acids also, eg oxalic acid
57.	34	Diwali poisoning is due to phosphorus	There is no such term as "Diwali poisoning". If we take this term to mean "poisoning due to ingestion of Diwali crackers", it can be due to several other elements and compounds
58.	35	OP produces Chromolacryorrhea	The correct term is chromo**d**acryorrhea [pl note "d"]
59.	35	The average normal values of cholinesterase are 77-142 in the red cells and 41-140 in the plasma [*No units are mentioned. It is not clear if these are in gms, mgs or µgs*]	No scientific value has any meaning "**without units**". The normal values for **RBC ChE** in men are **0.76** [in ΔpH/hour/0.02 ml red cells at 25^0C]. For other values and details, pl see **ch 35**.
60.	37	A **toxalbumin** is a toxic protein which resembles a bacterial protein in action, and causes agglutination of red cells with some hemolysis and is antigenic.	A **toxalbumin** is a toxic protein found in certain plants and bacteria, capable of stopping protein synthesis.
61.	37	Antiabrin is given in *Abrus precatorius* poisoning	It is completely ineffective, and is no more recommended
62.	38	Poisonous and non-poisonous snakes	The correct terminology is venomous and non-venomous snakes. A poisonous snake would be harmful when eaten.
63.	39	*Stigmata maides* (Maize) causes pellagra due to lack of nicotinic acid	The correct botanical name of maize is *Stigmata maydis*.
64.	40	Absolute alcohol is **99.5% ethyl alcohol**	Absolute alcohol is ethanol containing not more than 1% by mass of water **[WHO definition]**. Thus any solution of ethanol containing between **99-100%** alcohol is absolute alcohol.
65.	40	Rectified spirit is 90% ethyl alcohol.	Rectified spirit is **95% ethyl alcohol**.